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THE AMERICAN JOURNAL OF PSYCHIATRY

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In this issue

F. Redlich and R. F. Mollica on Ethical Issues in Psychiatry

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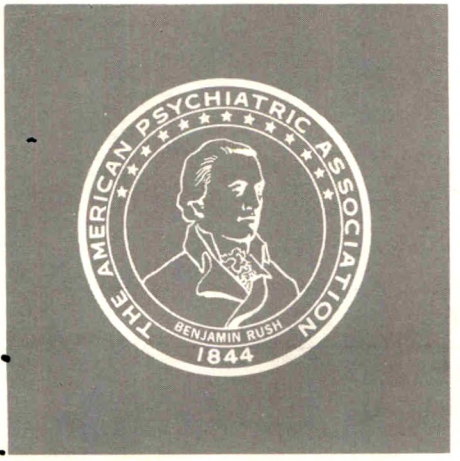
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Released from symptoms of psychotic depression...



Productive again.

By helping to reduce the frequency and intensity of psychotic symptoms, Navane (thiothixene) often permits resumption of more normal, more productive living.

The antipsychotic effectiveness of Navane—with relatively little drowsiness—helps patients remain more active, more alert, better able to meet the ordinary demands of life.

Cardiovascular side effects such as hypotension and nonspecific EKG changes are relatively rare with Navane (thiothixene). Extrapyramidal symptoms have been reported, but are usually controlled by reduction in dosage and/or administration of antiparkinson drugs.

Once you've controlled acute psychosis or psychotic depression, some patients can be maintained on a simple once-a-day dose. This once-a-day regimen can reduce the risk of missed doses with no loss of efficacy.

ROERIG Pfizer
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P, 24,346

Navane®

(thiothixene) (thiothixene hydrochloride)

Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg/Concentrate 5 mg/ml

Once-a-day–to help control symptoms of psychosis

For prescribing information, including adverse reactions and contraindications please see following page of this advertisement.

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg, of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

More detailed professional information available on request.

ROERIG Pfizer
A division of Pfizer Pharmaceuticals

In clinically significant depression



TABLETS, 5 mg and 10 mg

Vivactil®*

(Protriptyline HCl | MSD)

*** When you want an antidepressant that is characteristically nonsedating for the patient under close medical supervision (Symptoms such as anxiety or agitation may be aggravated.)**

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information please see following page.

In clinically significant depression

TABLETS, 5 mg and 10 mg

Vivactil®

(Protriptyline HCl | MSD)

Contraindications: Known hypersensitivity; acute recovery phase following myocardial infarction. Should not be given concomitantly with an MAOI; hyperpyretic crises, severe convulsions, and deaths have occurred in patients receiving tricyclic antidepressant and MAOI drugs simultaneously. When it is desired to substitute protriptyline HCl for an MAOI, a minimum of 14 days should be allowed to elapse after the latter is discontinued. Protriptyline HCl should then be initiated cautiously with gradual increase in dosage until optimum response is achieved.

Warnings: May block the antihypertensive effect of guanethidine or similarly acting compounds. May impair mental and/or physical abilities required for the performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Should be used with caution in patients with a history of seizures and, because of its autonomic activity, in patients with a tendency to urinary retention or increased intraocular tension.

Tachycardia and postural hypotension may occur more frequently than with other antidepressant drugs. Should be used with caution in elderly patients and patients with cardiovascular disorders; such patients should be observed closely because of the tendency of the drug to produce tachycardia, hypotension, arrhythmias, and prolongation of the conduction time. Myocardial infarction and stroke have occurred with drugs of this class. On rare occasions, hyperthyroid patients or those receiving thyroid medication may develop arrhythmias when this drug is given.

Usage in Children: Not recommended for use in children because safety and effectiveness in the pediatric age group have not been established.

Usage in Pregnancy: Safe use in pregnancy and lactation has not been established; therefore, use in pregnant women, nursing mothers, or women who may become pregnant requires that possible benefits be weighed against possible hazards to mother and child.

Precautions: When protriptyline HCl is used to treat the depressive component of schizophrenia, psychotic symptoms may be aggravated; likewise, in manic-depressive psychosis, depressed patients may experience a shift toward the manic phase; paranoid delusions, with or without associated hostility, may be exaggerated. In any of these circumstances, it may be advisable to reduce the dose of protriptyline HCl or to use a major tranquilizing drug concurrently. Symptoms, such as anxiety or agitation, may be aggravated in overactive or agitated patients.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. May enhance response to alcohol and effects of barbiturates and other CNS depressants. Possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent administration with electroshock therapy may increase hazards of therapy; such treatment should be limited to patients for whom it is essential. Discontinue drug several days before elective surgery, if possible. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions which have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when protriptyline HCl is administered. Protriptyline HCl is more likely to aggravate agitation and anxiety and produce cardiovascular reactions such as tachycardia and hypotension.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions, anxiety, restlessness, agitation; insomnia, panic, and nightmares; hypomania; exacerbation of psychosis.

Neurological: numbness, tingling, and paresthesias of extremities; incoordination, ataxia, tremors, peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns, tinnitus.

Anticholinergic: dry mouth and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of the urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (general, or of face and tongue), drug fever.

Hematologic: bone marrow depression; agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of other tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

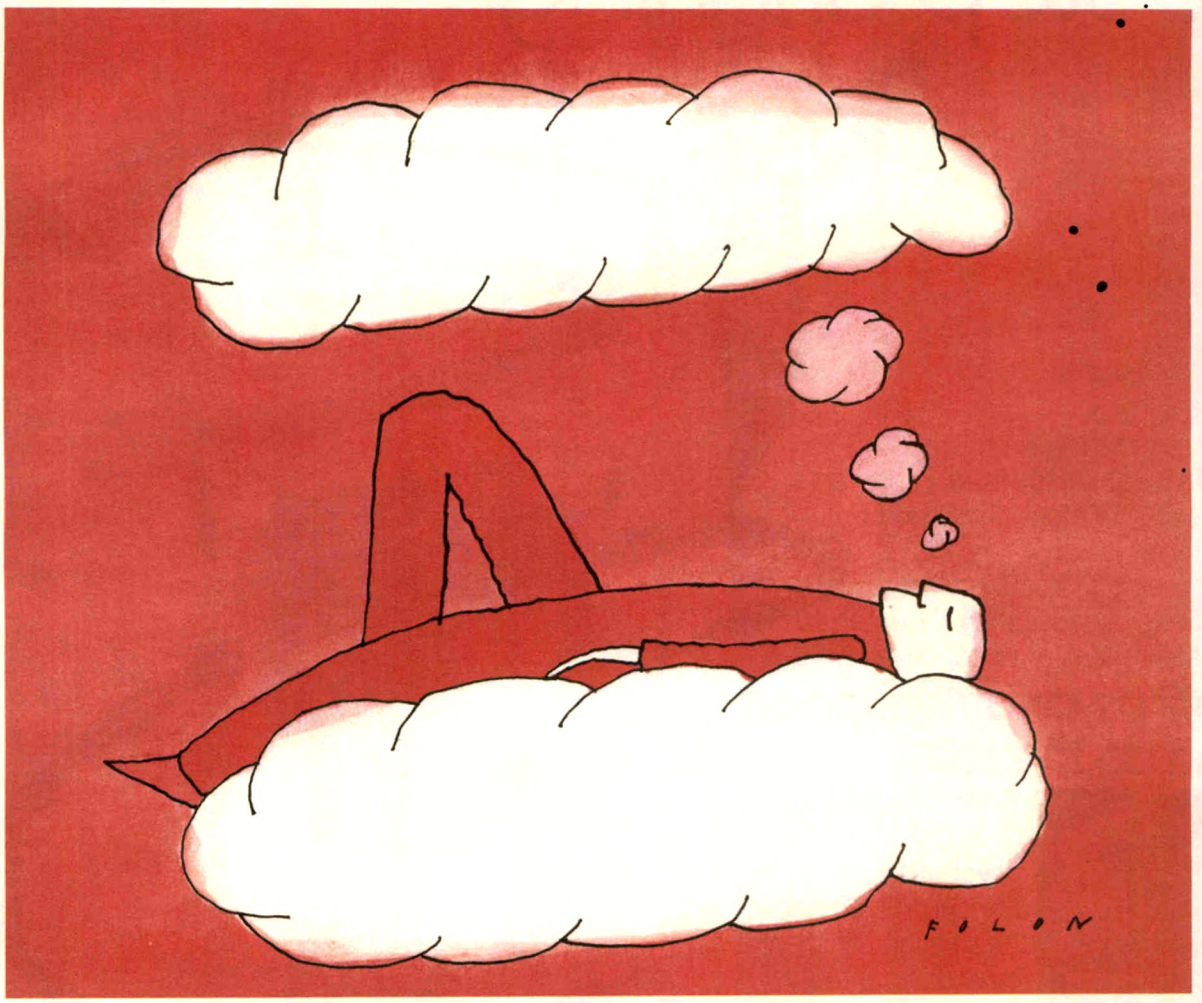
How Supplied: Tablets, containing 5 mg and 10 mg protriptyline HCl each, in single-unit packages of 100 and bottles of 100 and 1000.

For more detailed information, consult your MSD representative or see full prescribing information.

Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

MSD
MERCK SHARP & DOHME

Doctor, I dreamed last night you saved me lots of money!



Prescribe low-priced

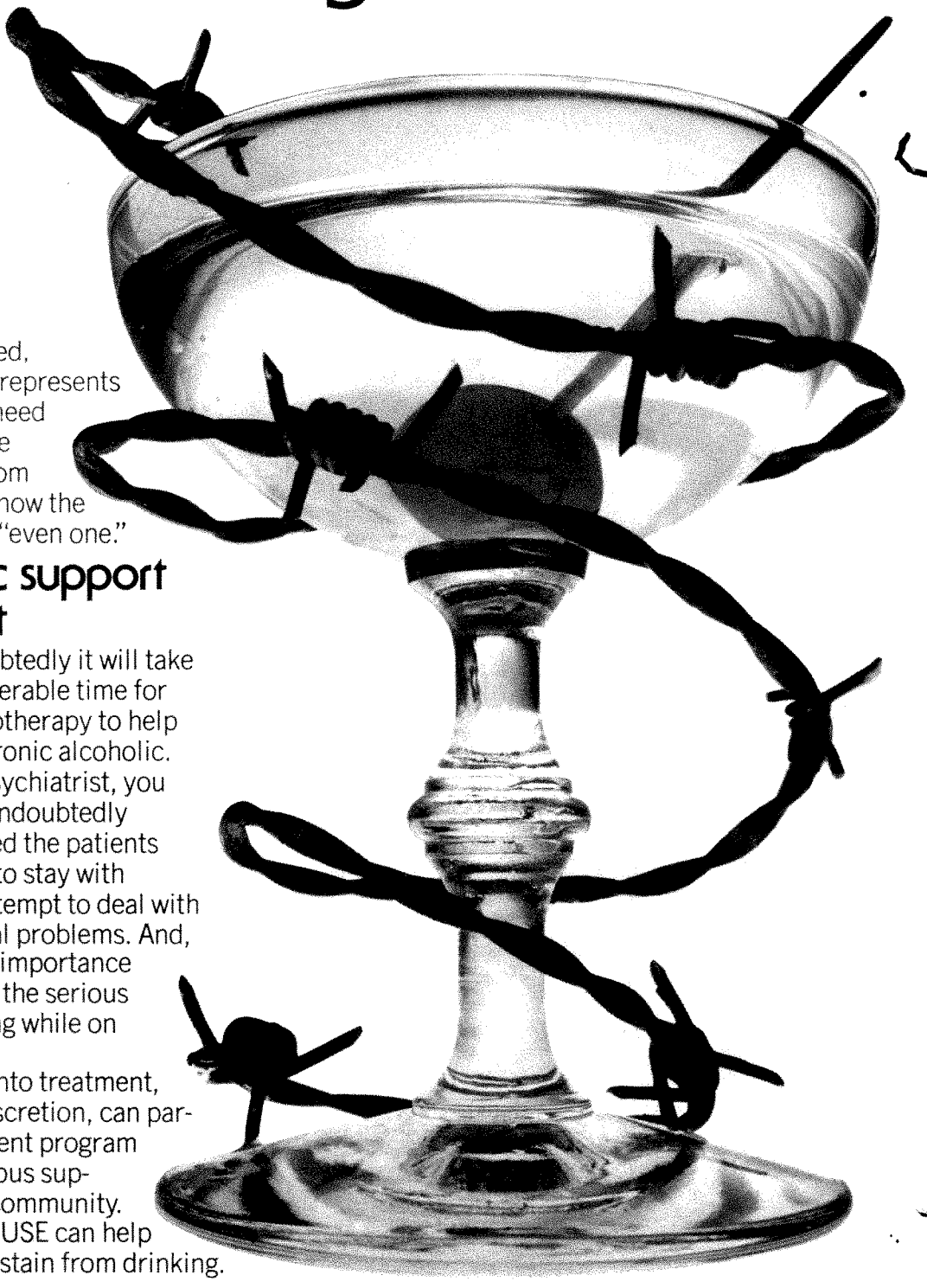
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(imipramine HCl, SK&F)

Tablets 10 mg., 25 mg., 50 mg.

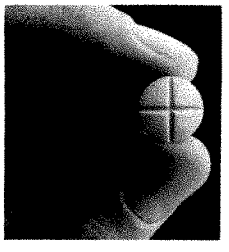
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Philadelphia, Pa.

ANTABUSE: "Social security" for the alcoholic who wants to stop drinking...



The temptation is resisted, ANTABUSE (disulfiram) represents the security alcoholics need in any situation. They are better able to abstain from drinking because they know the consequences of taking "even one."

until psychiatric support can do the rest



Undoubtedly it will take considerable time for psychotherapy to help the chronic alcoholic. As a psychiatrist, you have undoubtedly selected the patients who are best motivated to stay with ANTABUSE while you attempt to deal with the underlying emotional problems. And, of course, you know the importance of reminding patients of the serious consequences of drinking while on ANTABUSE.

Once sober, and well into treatment, the alcoholic, at your discretion, can participate in a total treatment program which makes use of various supportive resources in the community. In the meantime, ANTABUSE can help the chronic alcoholic abstain from drinking.

BRIEF SUMMARY
(For full prescribing information, see package circular.)

ANTABUSE® (disulfiram) in Alcoholism

INDICATION: ANTABUSE is an aid in the management of selected chronic alcoholic patients who want to remain in a state of enforced sobriety so that supportive and psychotherapeutic treatment may be applied to best advantage. (Used alone, without proper motivation and without supportive therapy, ANTABUSE is not a cure for alcoholism, and it is unlikely that it will have more than a brief effect on the drinking pattern of the chronic alcoholic.)

CONTRAINDICATIONS: Patients who are receiving or have recently received metronidazole, paraldehyde, alcohol, or alcohol-containing preparations, *e.g.* cough syrups, tonics, and the like, should not be given ANTABUSE.

ANTABUSE is contraindicated in the presence of severe myocardial disease or coronary occlusion, psychoses, or hypersensitivity.

WARNINGS: ANTABUSE should never be administered to a patient when he is in a state of alcohol intoxication or without his full knowledge.

The physician should instruct relatives accordingly.

The patient must be fully informed of the ANTABUSE-alcohol reaction. He must be strongly cautioned against surreptitious drinking while taking the drug, and he must be fully aware of possible consequences. He should be warned to avoid alcohol in disguised form, *i.e.* in sauces, vinegars, cough mixtures, and even aftershave lotions and back rubs. He should also be warned that reactions may occur with alcohol up to 14 days after ingesting ANTABUSE.

THE ANTABUSE-ALCOHOL REACTION: ANTABUSE plus alcohol, even small amounts, produces flushing, throbbing in head and neck, throbbing headache, respiratory difficulty, nausea, copious vomiting, sweating, thirst, chest pain, palpitation, dyspnea, hyperventilation, tachycardia, hypotension, syncope, marked uneasiness, weakness, vertigo, blurred vision, and confusion. In severe reactions there may be respiratory depression, cardiovascular collapse, arrhythmias, myocardial infarction, acute congestive heart failure, unconsciousness, convulsions, and death.

The intensity of the reaction varies with each individual, but is generally proportional to the amounts of ANTABUSE (disulfiram) and alcohol ingested. Mild reactions may occur in the sensitive individual when the blood alcohol concentration is increased to as little as 5 to 10 mg. per 100 cc. Symptoms are fully developed at 50 mg. per 100 cc., and unconsciousness usually results when the blood alcohol level reaches 125 to 150 mg.

The duration of the reaction varies from 30 to 60 minutes to several hours in the more severe cases, or as long as there is alcohol in the blood.

DRUG INTERACTIONS: Disulfiram appears to decrease the rate at which certain drugs are metabolized and so may increase the blood levels and the possibility of clinical toxicity of drugs given concomitantly.

Disulfiram should be used with caution in those patients receiving diphenylhydantoin and its congeners, since toxic levels of these antiepileptic agents have been reported during concomitant disulfiram therapy.

It may be necessary to adjust the dosage of oral anticoagulants upon beginning or stopping disulfiram, since disulfiram may prolong prothrombin time.

Patients taking isoniazid when disulfiram is given should be observed for the appearance of unsteady gait or marked changes in mental status and the disulfiram discontinued if such signs appear.

CONCOMITANT CONDITIONS: Because of the possibility of an accidental ANTABUSE-alcohol reaction, ANTABUSE (disulfiram) should be used with extreme caution in patients with any of the following conditions: diabetes mellitus, hypothyroidism, epilepsy, cerebral damage, chronic and acute nephritis, hepatic cirrhosis or insufficiency.

USAGE IN PREGNANCY: The safe use of this drug in pregnancy has not been established. Therefore, ANTABUSE (disulfiram) should be used during pregnancy only when, in the judgment of the physician, the probable benefits outweigh the possible risks.

PRECAUTIONS: It is suggested that every patient under treatment carry an Identification Card, stating that he is receiving ANTABUSE and describing the symptoms most likely to occur as a result of the ANTABUSE-alcohol reaction. In addition, this card should indicate the physician or institution to be contacted in emergency. (Cards may be obtained from Ayerst Laboratories upon request.)

Alcoholism may accompany or be followed by dependence on narcotics or sedatives. Barbiturates have been administered concurrently with ANTABUSE (disulfiram) without untoward effects, but the possibility of initiating a new abuse should be considered.

Base line and follow-up transaminase tests (10-14 days) are suggested to detect any hepatic dysfunction that may result with ANTABUSE therapy. In addition, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

ADVERSE REACTIONS: (See Contraindications, Warnings, and Precautions.)

Occasional skin eruptions are, as a rule, readily controlled by concomitant administration of an antihistaminic drug.

In a small number of patients, a transient mild drowsiness, fatigability, impotence, headache, acneform eruptions, allergic dermatitis, or a metallic or garlic-like aftertaste may be experienced during the first two weeks of therapy. These complaints usually disappear spontaneously with the continuation of therapy or with reduced dosage.

Psychotic reactions have been noted, attributable in most cases to high dosage, combined toxicity (with metronidazole or isoniazid), or to the unmasking of underlying psychoses in patients stressed by the withdrawal of alcohol.

There have been reports of polyneuritis and peripheral neuritis, and rare instances of optic neuritis. One case of cholestatic hepatitis has been reported, but its relationship to ANTABUSE has not been unequivocally established.

DOSAGE AND ADMINISTRATION: ANTABUSE (disulfiram) should never be administered until the patient has abstained from alcohol for at least 12 hours.

INITIAL DOSAGE SCHEDULE: In the first phase of treatment, a maximum of 500 mg. daily is given in a single dose for one to two weeks. Although usually taken in the morning, ANTABUSE may be taken on retiring by patients who experience a sedative effect. Alternatively, to minimize, or eliminate, the sedative effect, dosage may be adjusted downward.

MAINTENANCE REGIMEN: The average maintenance dose is 250 mg. daily (range, 125 to 500 mg.); it should not exceed 500 mg. daily.

NOTE: Occasional patients, while seemingly on adequate maintenance doses of ANTABUSE (disulfiram), report that they are able to drink alcoholic beverages with impunity and without any symptomatology. All appearances to the contrary, such patients must be presumed to be disposing of their tablets in some manner without actually taking them. Until such patients have been observed reliably taking their daily ANTABUSE tablets (preferably crushed and well mixed with liquid), it cannot be concluded that ANTABUSE is ineffective.

DURATION OF THERAPY: The daily, uninterrupted administration of ANTABUSE must be continued until the patient is fully recovered socially and a basis for permanent self-control is established. Depending on the individual patient, maintenance therapy may be required for months or even years.

TRIAL WITH ALCOHOL: During early experience with ANTABUSE, it was thought advisable for each patient to have at least one supervised alcohol-drug reaction. More recently, the test reaction has been largely abandoned. Furthermore, such a test reaction should never be administered to a patient over 50 years of age. A clear, detailed, and convincing description of the reaction is felt to be sufficient in most cases.

However, where a test reaction is deemed necessary, the suggested procedure is as follows:

After the first one to two weeks' therapy with 500 mg. daily, a drink of 15 cc. (½ oz.) of 100 proof whiskey or equivalent is taken slowly. This test dose of alcoholic beverage may be repeated once only so that the total dose does not exceed 30 cc. (1 oz.) of whiskey. Once a reaction develops, no more alcohol should be consumed. Such tests should be carried out only when the patient is hospitalized, or comparable supervision and facilities, including oxygen, are available.

MANAGEMENT OF ANTABUSE (DISULFIRAM)-ALCOHOL REACTION: In severe reactions, whether caused by an excessive test dose or by the patient's unsupervised ingestion of alcohol, supportive measures to restore blood pressure and treat shock should be instituted. Other recommendations include: oxygen, carbogen (95 per cent oxygen and 5 per cent carbon dioxide), vitamin C intravenously in massive doses (1 Gm.), and ephedrine sulfate. Antihistamines have also been used intravenously. Potassium levels should be monitored particularly in patients on digitalis since hypokalemia has been reported.

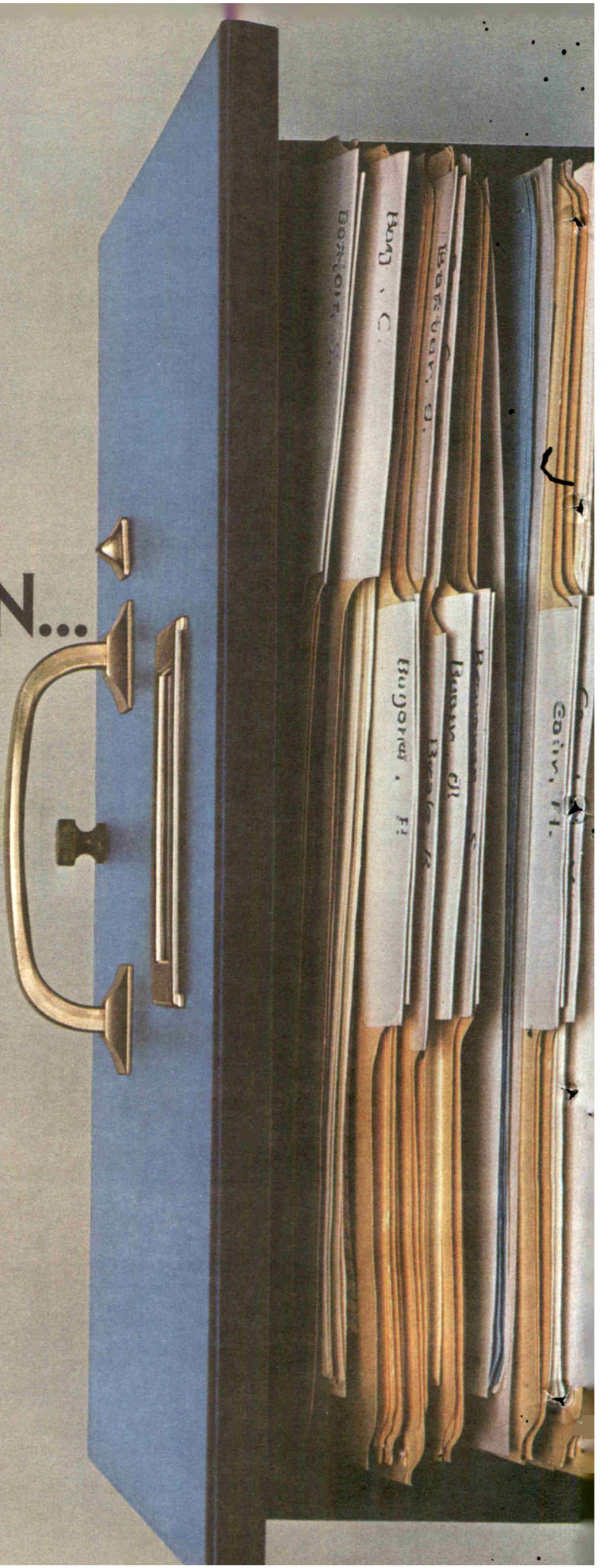
HOW SUPPLIED: No. 809—Each tablet (scored) contains 250 mg. disulfiram, in bottles of 100. No. 810—Each tablet (scored) contains 500 mg. disulfiram, in bottles of 50 and 1,000.

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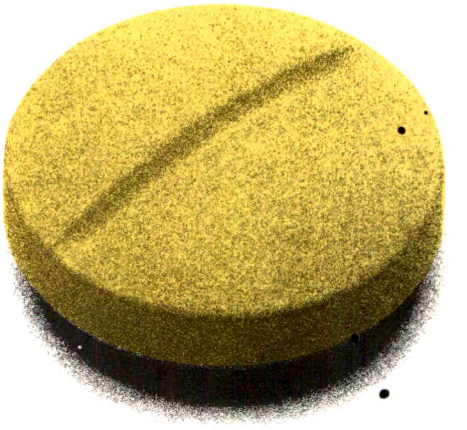
Endep™ 75 mg

amitriptyline HCl / Roche

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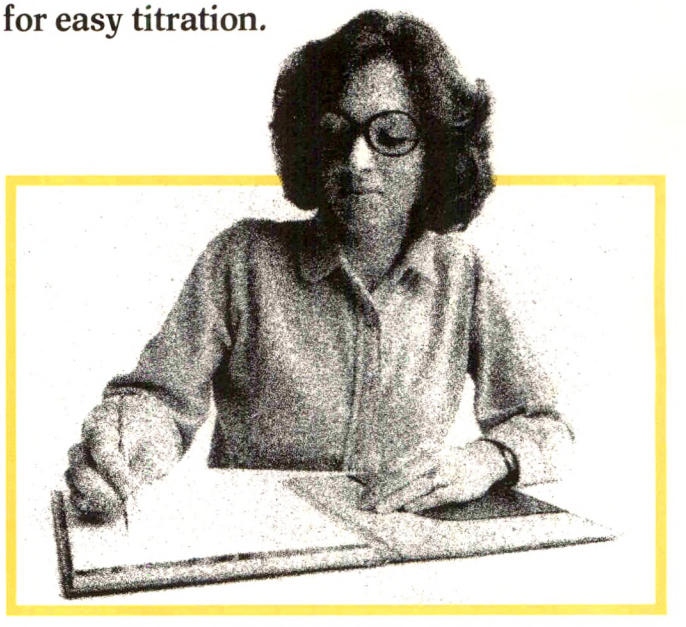
Patients are more likely to remember the single bedtime dosage of Endep (amitriptyline HCl).



You'll remember 1 or 2 tablets *h.s.*, because fewer forgotten doses produce a more consistent clinical effect, and because it is the only tricyclic antidepressant scored for easy titration.



Patients will also remember family/interpersonal relationships that they have forgotten or neglected.



In three or four weeks patients will usually remember how well they used to feel.

Endep™ 75 mg

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day

Before prescribing, please consult complete product information, a summary of which appears on the following page.

Introducing Endep™

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day



(orange)	(orange)	(orange)	(yellow)
10-mg scored tablets	**25-mg** scored tablets	**50-mg** scored tablets	**75-mg** scored tablets

■ Endep (amitriptyline HCl), in responsive patients, lifts mood and relieves symptoms such as anorexia, loss of interest, inability to concentrate, decreased libido or GI disturbances, often within three or four weeks after starting therapy.

■ Endep usually relieves accompanying sleep disturbances, particularly early in the treatment regimen.

■ All four dosage strengths are scored for greater flexibility and convenience in titration.

■ The 75-mg *h.s.* dose minimizes skipped doses, maximizes patient acceptance and compliance.

■ Specifying Endep/Roche assures quality plus potential economy.

Important Considerations

Should not be given concomitantly with an MAO inhibitor.

Not recommended for use during the acute recovery phase following myocardial infarction.

Contraindicated in patients with hypersensitivity to amitriptyline.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh potential benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia. purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels; increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier. *Initial Adult Dosage:* Outpatients – 25 mg t.i.d.; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate – 50 to 100 mg h.s., gradually increasing h.s. dose up to 150 mg/day. Hospitalized Patients – Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day. *Adolescent and Elderly Patients:* In general, 10 mg t.i.d. with 20 mg h.s. may be satisfactory for those who do not tolerate higher doses. *Maintenance Dosage:* With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg b.i.d. to q.i.d., or 10 mg q.i.d. Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg and 75-mg scored tablets – bottles of 100 and 500; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg), available singly and in trays of 10.

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Behavior Therapy in Psychiatry

This comprehensive report reviews the historical development of behavior therapy, its efficacy for the treatment of psychiatric problems, its current forms and uses as well as potential abuses, and its relationship to dynamic psychiatry. Approved for publication by the APA Council on Research and Development, the Report was prepared by a Task Force comprising Lee Birk, M.D., John Paul Brady, M.D., Alan J. Rosenthal, M.D., W. Douglas Skelton, M.D., Joseph B. Stevens, M.D., and Consultants Stephanie B. Stolz, Ph.D., Joseph V. Brady, Ph.D., Arnold A. Lazarus, Ph.D., James J. Lynch, Ph.D., and Edwin J. Thomas, Ph.D.

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This report marks the first formal statement the Association has ever issued delineating the vital role of its private practitioner members in the delivery of mental health services in the U.S. Contains sections on patterns of private practice, the economics of it, the impact of third party payments on it, its relation to the public sector, and an assessment of the overall contribution of the private sector to the treatment of mental illness. Stresses the theme that any national system for the delivery of mental health services must be a "balanced mix" of both the private and public sectors, each reinforcing the other. Approved for publication by the Council on Mental Health Services, the Report was prepared by a Task Force comprising Drs. Ewald Busse, Rogers J. Smith, Reed S. Andrus, Winston Cochran, Albert A. Lorenz, Robert L. Leopold, Louis W. Nie, and Consultants Alan I. Levenson, Zigmond M. Lebensohn, Walter E. Barton, and Mr. Robert L. Robinson.

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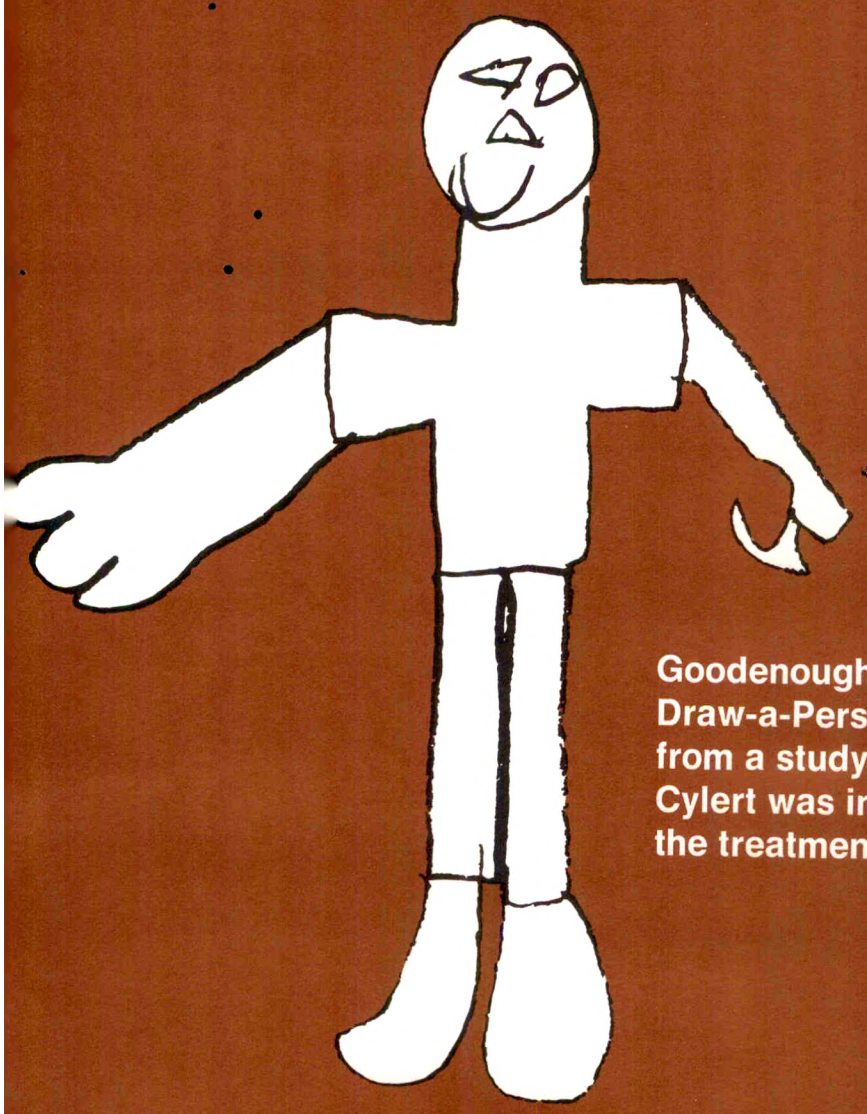
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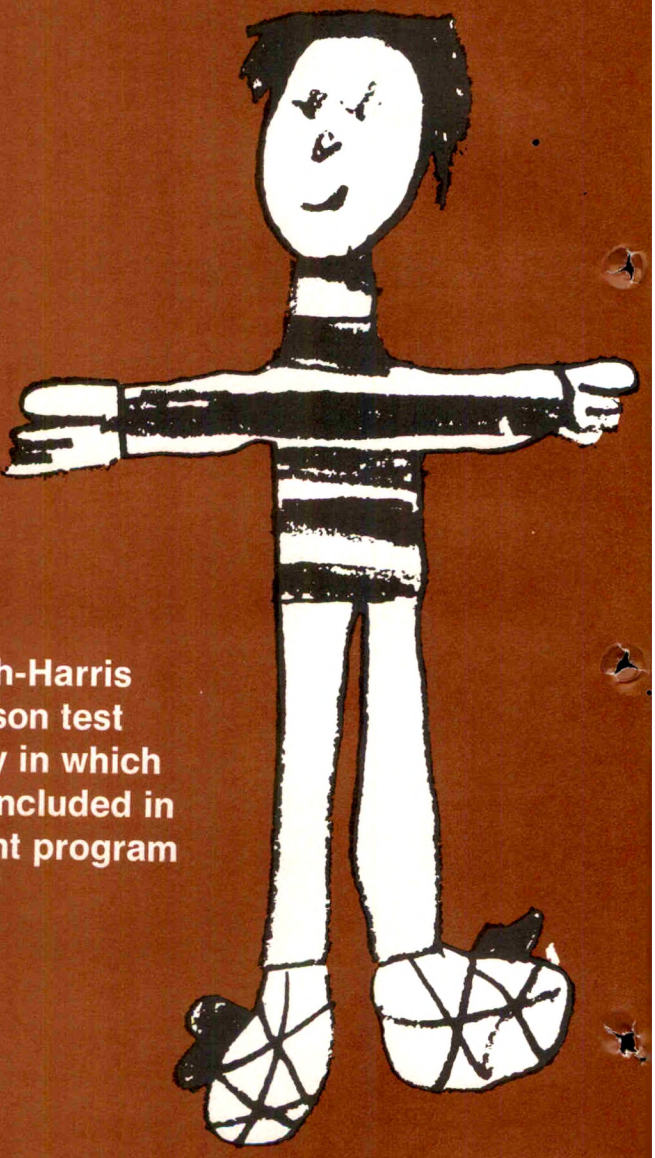
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Drawing made prior to treatment

Goodenough-Harris Draw-a-Person test from a study in which Cylert was included in the treatment program



Drawing made at week 8 during treatment

Cylert (pemoline) will not in itself "enhance learning" or resolve difficult behavioral problems. But it can increase attention span in the hyperkinetic child and reduce the impulsivity that often interferes with the learning process.

Cylert®
(pemoline)

offers these benefits in a treatment program for MBD

- **Single daily dose administration**
- **Minimal cardiovascular effects**
- **Mean dosage in long-term studies remained remarkably constant**

EFFICACY

Multi-clinic study[1,2]
21 investigators from 10 states and two provinces in Canada took part in the clinical studies.

Double-blind, placebo control
413 patients were randomly assigned to Cylert or placebo groups. 238 patients met all criteria for evaluation of efficacy.

Psychological test results
Children on Cylert had significantly higher scores statistically than those on placebo on these psychological tests:

- The Wechsler Intelligence Scale for Children (WISC) and its performance IQ Sub-Component
- The Wide Range Achievement Test (WRAT) (reading and arithmetic)
- The Lincoln-Oseretsky Motor Performance Test Factor II

Overall results
Approximately two out of three patients were significantly improved by treatment with Cylert as reflected by global ratings.

1. Conners, C. K., ed., *Clinical Use of Stimulant Drugs in Children*, Excerpta Medica, 1974, p. 98.
2. Page, J. G., et al., J. Learning Disabilities, 7:498, Oct., 1974.

SAFETY

Multi-clinic study (9 weeks); safety data analyzed on 407 patients
There was no significant difference between Cylert and placebo groups in:

- Blood pressure
- Laboratory tests
- Pulse
- Neurological status

Insomnia and anorexia were the most frequently seen side effects and often improved with continuation of treatment or reduction of dosage.

Mean weight loss of 1.1 lbs. was demonstrated in the Cylert group during early weeks of treatment; long-term studies have shown that by 3-6 months, most children return to the normal rate of weight gain for their age group.

Long-term study on Cylert; up to 3 years and continuing

Mean dosage . . . remained remarkably constant.
Blood pressure . . no significant changes attributed to Cylert.
Pulse rate . . . . . no significant changes attributed to Cylert.

Laboratory examination—mild to moderate increase in transaminase (SGOT and SGPT) levels in 1-2% of patients (no clinical symptoms); levels returned to normal on withdrawal of medication.

No clinically significant abnormalities in the other tests.

Please see last page of this advertisement for Prescribing Information.

Importance of single daily dosage to the child, the parents and the teacher

For the child		For the adults
No drug in child's possession while at school	⟷	Control of medication remains with parents
Avoids situation in which child is repeatedly singled out as being "different"	⟷	Obviates need for nurse or teacher to supervise taking of mid-day doses
Helps prevent possible variations in effect caused by missed, forgotten or delayed doses	⟷	Helps assure that the prescribed dosage is being given each day



Cylert (pemoline), alone among CNS stimulants used to treat MBD, is inherently long-acting, permitting once-daily dosage.

Cylert can be taken with meals

You can prescribe Cylert a.c., p.c., or with meals. Although the speed of absorption is slightly slowed by food, the total absorption is not affected.

Dosage and administration

Cylert is given as a single oral dose each morning.

The recommended starting dose is 37.5 mg. per day. This daily dosage should be gradually increased at one-week intervals using increments of 18.75 mg. until the desired clinical response is obtained.

The mean daily effective dose ranges from 56.25 to 75 mg. per day. The maximum recommended daily dose of Cylert is 112.5 mg.

Using the recommended schedule of dose titration, significant benefits may not be seen until the third or fourth week of drug therapy. Side effects may be seen prior to optimum clinical results.

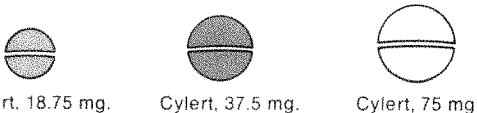
When not to use Cylert

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school-age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

The three dosage strengths of Cylert (pemoline)



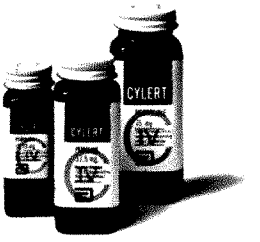
Cylert, 18.75 mg. (yellow-colored, grooved)

Cylert, 37.5 mg. (orange-colored, grooved)

Cylert, 75 mg. (tan-colored, grooved)

Tablets are actual size.

Cylert (PEMOLINE) CIV



Prescribing Information

Description: Cylert (pemoline) is a white, tasteless, odorless powder which is relatively insoluble (less than 1 mg/ml) in water, chloroform, ether, acetone, and benzene. In 95% ethyl alcohol, the solubility of pemoline is 2.2 mg/ml.

Actions: Cylert (pemoline) is a central nervous system stimulant. The pharmacologic activity of pemoline is similar to that of other known stimulants but with minimal sympathomimetic effects. Pemoline is structurally dissimilar from the amphetamines and methylphenidate. Although the exact mode of pharmacodynamic action is undetermined in man, pemoline has been reported to increase the rate of synthesis of dopamine in rat brain.

In human subjects, Cylert produces peak blood levels within 2-4 hours. The serum half-life is approximately 12 hours. Multiple dose studies in adults at several dose levels indicate that serum levels plateau in approximately three days. Cylert and its metabolites are primarily excreted by the kidneys with approximately 75% of an oral dose appearing in the urine within a 24-hour period. Approximately 43% of pemoline is excreted unchanged. Metabolites include pemoline dione, conjugated pemoline and mandelic acid.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: MINIMAL BRAIN DYSFUNCTION IN CHILDREN—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations:

Specific etiology of minimal brain dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis includes the use not only of medical but of psychological, educational, and social resources.

Characteristics commonly reported include: A chronic history of moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindication: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See PRECAUTIONS)

Warnings: Cylert is not recommended for children under six years of age since safety and efficacy in this age group have not yet been established.

Since Cylert (pemoline) and its metabolites are excreted primarily by the kidneys, caution should be observed in administering the drug to children with significantly impaired renal function.

Sufficient data on safety and efficacy of Cylert administration for periods beyond two years duration in children with minimal brain dysfunction are not yet available. Although a definite causal relationship has not been established, some temporary suppression of predicted growth pattern(i.e., weight and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.

Drug Interactions: Interactions between Cylert and other drugs have not been studied in humans. As with most other drugs, concurrent administration with other agents, especially drugs with central nervous system activity, should be carefully monitored.

Usage in Pregnancy: Safety for use in pregnancy has not been established. Standard studies of fertility, teratology and reproduction were conducted in rats and rabbits. Daily oral doses of pemoline of 18.75 and 37.5 mg/kg beginning at conception produced no abnormalities in the fetuses and did not affect viability at birth. Further studies using similar dose levels with drug administration beginning 14 days before conception demonstrated an increased incidence of stillbirths in these animals.

Drug Dependence: Studies of the drug abuse potential of Cylert (pemoline) in primates have not demonstrated a potential for self-administration. However, the pharmacologic similarities between Cylert and other CNS stimulants with known abuse liability suggest that drug dependence of the stimulant type might occur. There have been isolated reports of transient psychotic symptoms in adults following long-term misuse of pemoline taken orally in excessive quantities. Therefore, caution should be observed in emotionally unstable patients considered to have a psychological potential for drug dependence.

Precautions: Delayed hypersensitivity reactions involving the liver have been reported in 1-2% of the patients receiving Cylert usually after several months of therapy. No clinical symptomatology has been observed, but mild to moderate increases in transaminase (SGOT and SGPT) levels have occurred in these cases. These effects appear to be completely reversible when drug treatment is discontinued. Transaminase levels should be determined periodically during therapy with Cylert to detect any such reactions.

Adverse Reactions: The most frequently reported adverse reaction with Cylert is insomnia. Insomnia has been observed prior to optimum therapeutic response and in the majority of cases was transient in nature or responded to dosage reduction. Anorexia with weight loss during the first few weeks of therapy has also been reported. With continuing therapy, a return to a normal weight curve usually occurred within three to six months. Other adverse reactions reported include stomachache, skin rash, irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations. Mild adverse reactions appearing early in treatment often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage reduction or discontinuation should be considered.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning. The recommended starting dose is 37.5 mg per day. This daily dosage should be gradually increased at one week intervals using increments of 18.75 mg until the desired clinical response is obtained. The mean daily effective dose ranges from 56.25 to 75 mg per day. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally to determine if behavioral symptoms sufficient to require continuing therapy recur.

Overdosage: Cylert overdosage has been reported to produce symptoms of tachycardia, hallucinations, agitation, or restlessness. The treatment of acute massive overdosage with pemoline is essentially the same as that for overdosage with any drug having CNS stimulatory effects. Management is largely symptomatic and may include induction of emesis, gastric lavage or other measures as appropriate.

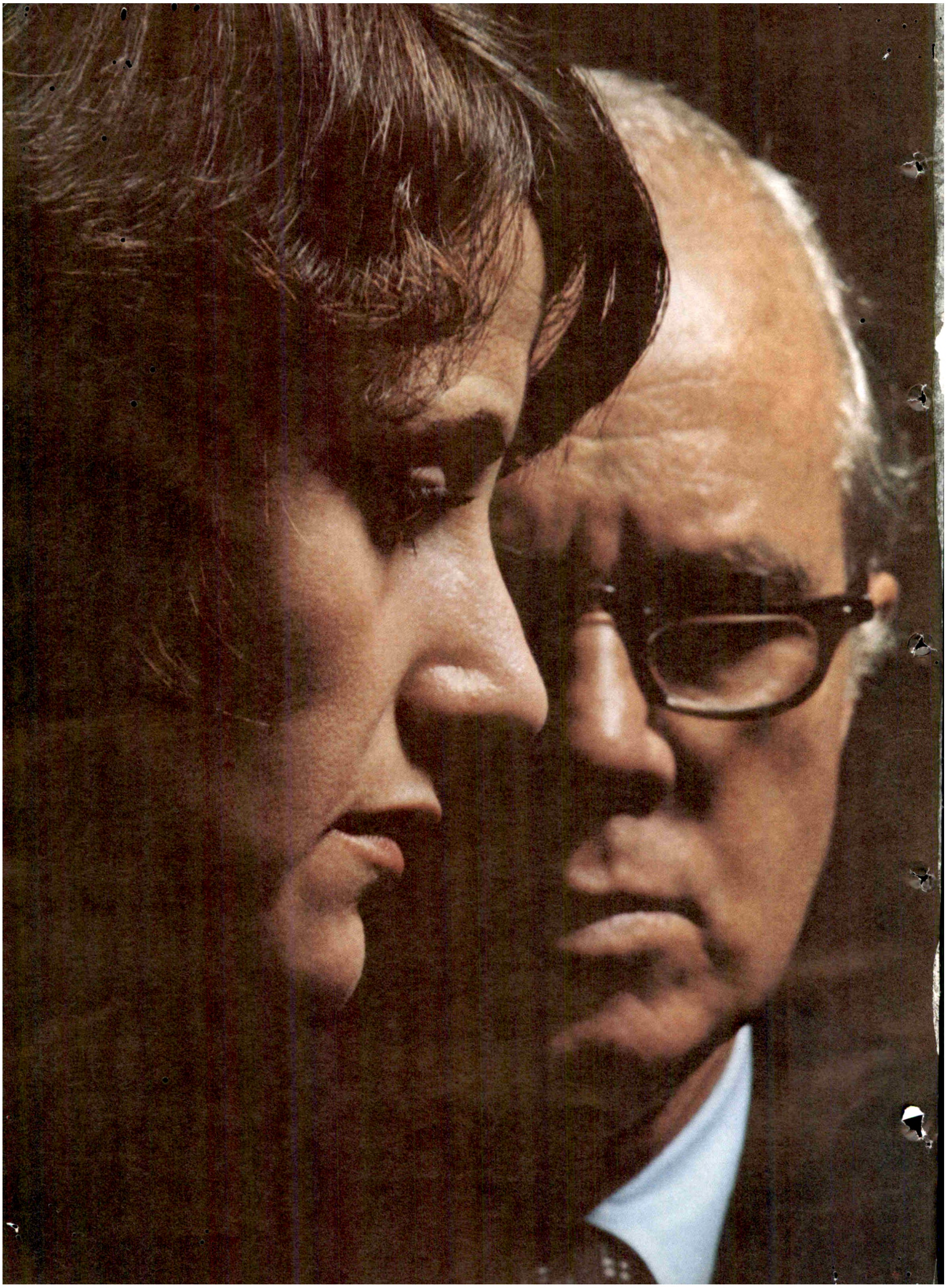
How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (**NDC** 0074-6025-13)

37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)

75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13)

ABBOTT LABORATORIES
North Chicago, IL60064 6013128



The TRIAVIL® Potential in the management of moderate to severe anxiety with depression

When time and talk are not enough...
The therapist is the primary catalyst for change in the psychotherapeutic relationship. However, when patients suffer from moderate to severe anxiety with depression, there are situations when TRIAVIL can often be a useful adjunct.

...TRIAVIL may help
There are three important benefits you may expect when TRIAVIL is part of the treatment program: (1) When symptoms of moderate to severe anxiety or agitation with depression are relieved, the patient may become more accessible and cooperative. (2) As somatic manifestations are controlled, attention may be focused on underlying causative factors. (3) Symptomatic relief may enable the patient to function more effectively in his daily life while your work with the patient progresses.

Tablets TRIAVIL are available in four different combinations affording flexibility and individualized dosage adjustment. Since it is simpler to remember to take one tablet rather than several (particularly in multiple daily doses), your patients on TRIAVIL will be more likely to take proper doses of the medication.

TRIAVIL is contraindicated in CNS depression from drugs; in the presence of evidence of bone marrow depression; and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please turn to the following page.

when patients exhibit moderate to marked anxiety or agitation with symptoms of depression

TRIAVIL® **containing perphenazine and amitriptyline HCl**

a tranquilizer-antidepressant

for highly effective relief of depression with moderate anxiety

TRIAVIL®

containing perphenazine and amitriptyline HCl
a tranquilizer-antidepressant

Available:

TRIAVIL® 2-25: Each tablet contains
2 mg perphenazine and 25 mg amitriptyline HCl

TRIAVIL® 2-10: Each tablet contains
2 mg perphenazine and 10 mg amitriptyline HCl

TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl

TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl

INITIAL THERAPY FOR MANY PATIENTS
TRIAVIL® 2-25 (or TRIAVIL® 4-25) t.i.d. or q.i.d.

FOR FLEXIBILITY IN ADJUSTING MAINTENANCE THERAPY
TRIAVIL® 2-10 (or TRIAVIL® 4-10)

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Do not give concomitantly with MAOI drugs because hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. Allow minimum of 14 days between therapies, then initiate therapy with TRIAVIL cautiously, with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given with guanethidine or similarly acting compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, particularly in high doses, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. Caution patients performing hazardous tasks, such as operating machinery or driving motor vehicles, that drug may impair mental and/or physical abilities. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Side effects may be any of those reported with phenothiazine drugs: extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and that the full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; hypnotic effects; pigmentary retinopathy; corneal and lenticular pigmentation; occasional lassitude, muscle weakness, mild insomnia. Other adverse reactions reported with various phenothiazine compounds include blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); liver damage (jaundice, biliary stasis); grand mal convulsions; cerebral edema; polyphagia; photophobia; skin pigmentation; and failure of ejaculation.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness; weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; jaundice; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

For more detailed information, consult your MSD Representative or see full Prescribing Information
Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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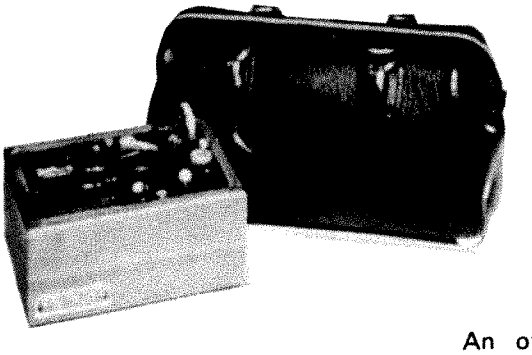
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An officially approved, extremely durable instrument which has earned world-wide professional acceptance, the MOL-AC II is economically priced with a fine, genuine leather physician's bag included.

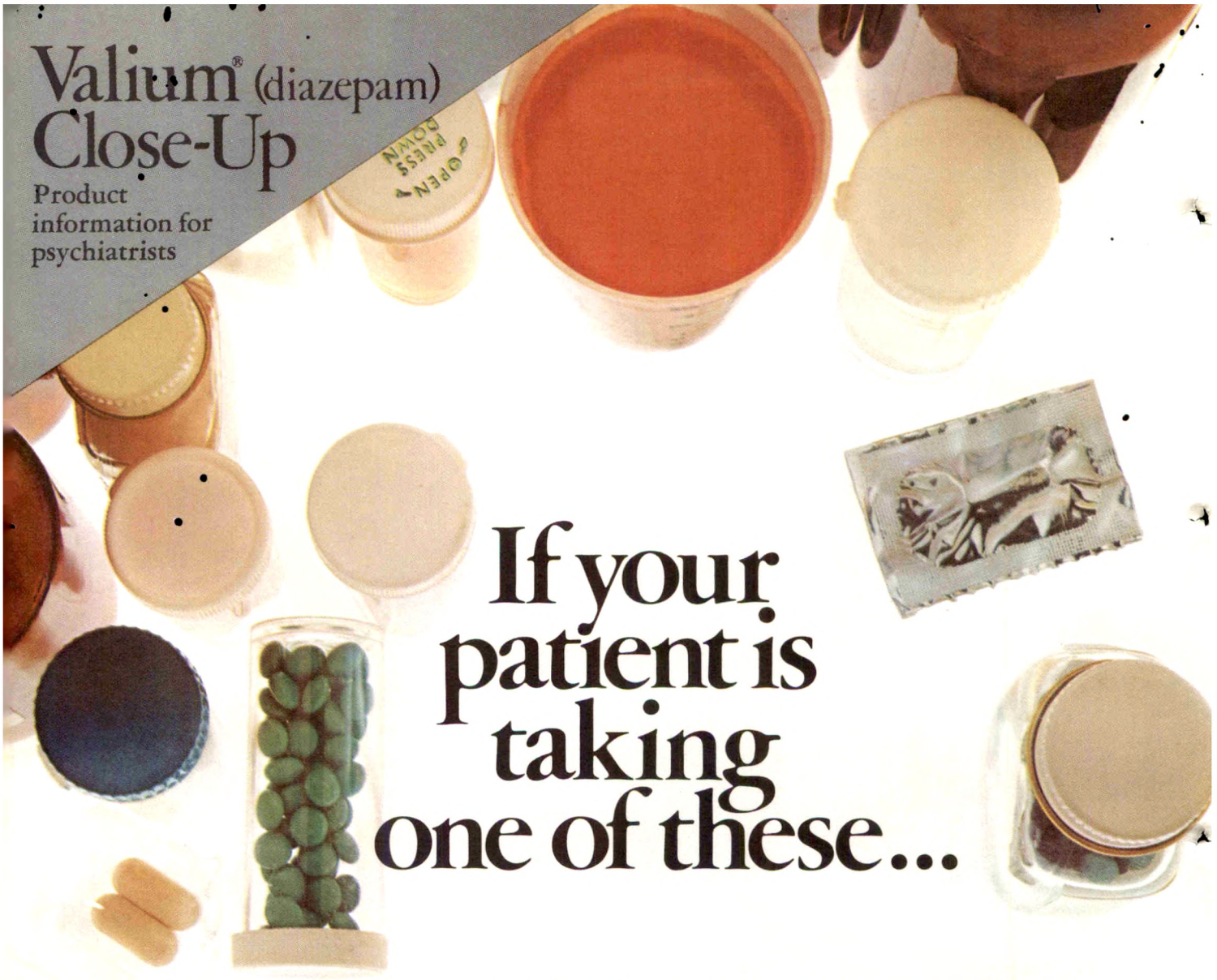
Other Models Still Available: The CW47, original therapeutic cerebral stimulator with only 3 of the 9 treatment current selections available on Model SOS.

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Valium® (diazepam) Close-Up

Product information for psychiatrists



If your patient is taking one of these...

Primary medications

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diuretics
vasodilators
anticholinergics

Psychotropics/CNS depressants

alcohol
phenothiazines
narcotics
barbiturates
hypnotics
MAO inhibitors
minor tranquilizers
antidepressants

Anticonvulsants

ROCHE



you should know this about Valium® (diazepam)

Valium is frequently used with most classes of these primary medications. Clinically significant drug interactions have not been reported to date.

Potentiation may occur with other CNS depressants. Careful consideration should be given to the pharmacology of these compounds when used concomitantly with Valium.

As with other agents which have anticonvulsant activity, the possibility of increase in frequency and/or severity of grand mal seizures may require an increase in dosage of standard anticonvulsants.

Valium® (diazepam)

2-mg, 5-mg, 10-mg scored tablets ℞

Before prescribing, please see following

Valium® (diazepam)

2-mg, 5-mg, 10-mg scored tablets

can relieve psychoneurotic anxiety and its associated depressive symptoms

Prompt, effective action. Valium works rapidly to relieve pronounced psychic tension in patients overreacting to stress and in psychoneurotic patients.

Wide margin of safety. Valium is generally well tolerated and in usual dosages rarely produces significant adverse reactions.

Dosage flexibility. Scored Valium 2-, 5-, 10-mg tablets give you dosage flexibility no tranquilizer capsule can match.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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Evaluation of Psychological Therapies

Psychotherapies, Behavior Therapies
Drug Therapies, and Their Interactions

edited by Robert L. Spitzer and Donald F. Klein

The contributors take a searching look at the efficacy and interaction of the most popular psychiatric therapies — individual psychoanalysis and psychotherapy, behavior therapy, and drug-psychological therapies — in treating depression, schizophrenia, behavioral disturbances of children, alcoholism, and various phobias. $17.00

Textbook of Disturbances of Mental Life

Johann Christian Heinroth

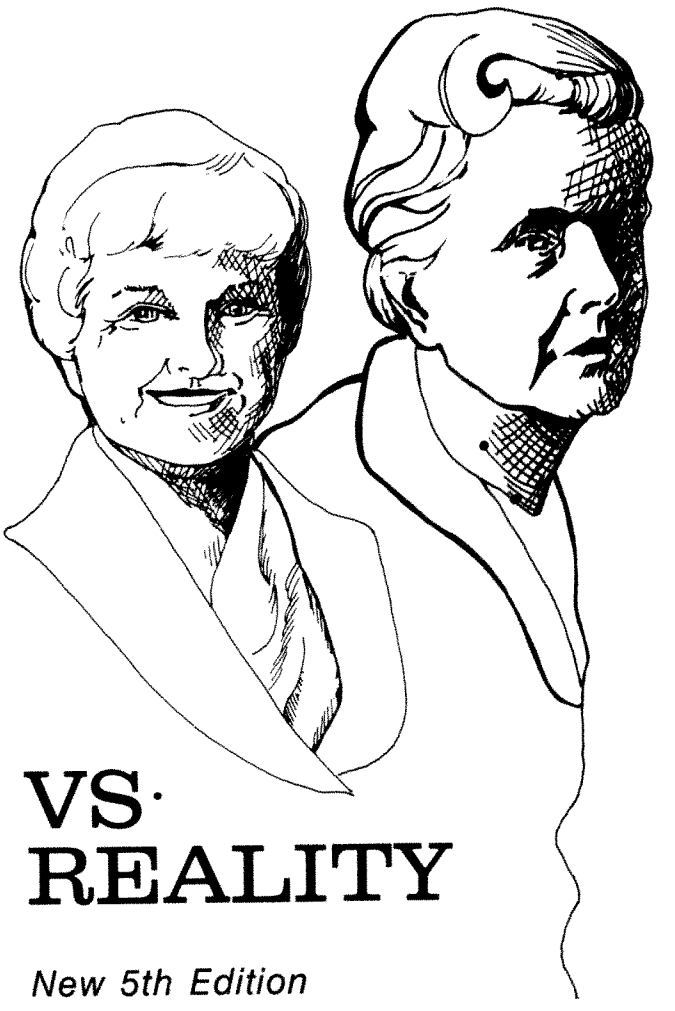
Preface by George Mora

Far in advance of his time in his enlightened and humane attitude toward mental illness, Heinroth helped firmly establish psychiatry as being the domain of the physician rather than the philosopher or the theologian. His most significant work in the field of mental pathology is now made available in English for the first time.
Two volumes. $17.50

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APPEARANCE



VS. REALITY

New 5th Edition

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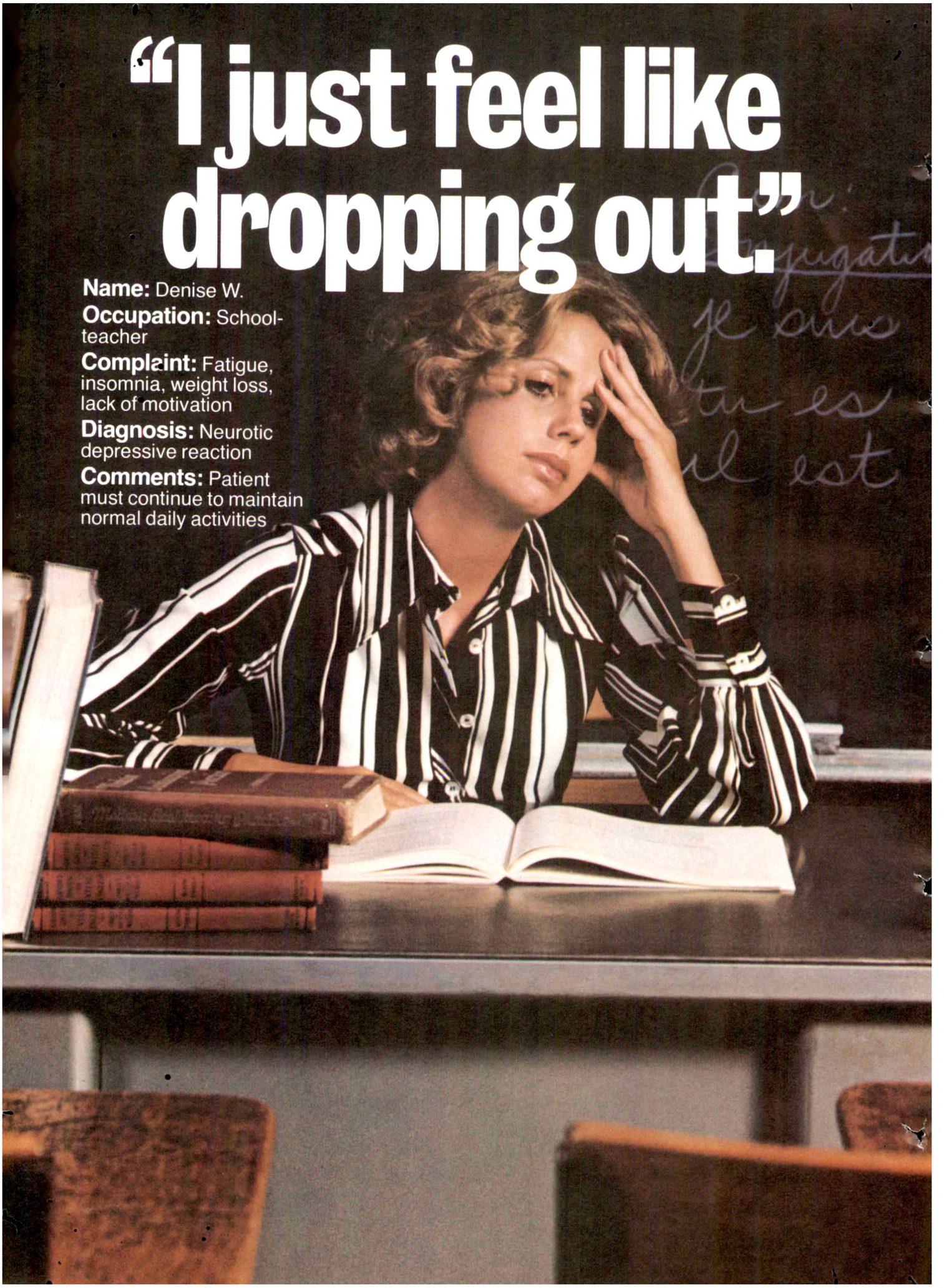
There is no cure . . . or social, medical, or legal facility that fully recognizes the pathologic disorders of the psychopathic personality (from the Preface). Dr. Hervey Cleckley, renowned psychiatrist and expert in understanding and defining the psychopath, explains the reality behind the mask of sanity worn by psychopaths. Now available in a new 5th edition, Dr. Cleckley's classic work presents careful, clinical observations and working hypotheses on psychopathy. Clinical anecdotes and updated case studies vividly illustrate the most current findings in the field. New references and a new bibliography are included. Major sections in the book outline the problem, present full and incomplete manifestations of the disorder, provide clinical profiles and comparisons, and reveal current controversies and possible rehabilitation.

By Hervey M. Cleckley, M.D. March, 1976. Approx. 544 pages, 6½" x 9½". About $14.95.

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"I just feel like dropping out."



Name: Denise W.
Occupation: School-teacher
Complaint: Fatigue, insomnia, weight loss, lack of motivation
Diagnosis: Neurotic depressive reaction
Comments: Patient must continue to maintain normal daily activities

In depression

Proven clinical efficacy

When patient complaints lead you to a diagnosis of depression, "...desipramine is an effective antidepressant...The most effective results occurred in those patients in whom the primary diagnosis was a psychoneurotic depressive reaction."[1] Such efficacy has been well documented[1-4] in over a decade of clinical use.

Prompt therapeutic response

Several studies have shown that Pertofrane may have a rapid onset of therapeutic action. This relatively rapid onset of action is particularly useful when patients must *continue* to function actively on their jobs or at home.

Mid-range sedation

"There is no problem of drowsiness."[1] Pertofrane shows good to excellent antidepressant effect yet is only mid-range in sedation[5]—especially important when patients must maintain normal levels of daily activity.

Compatible with tranquilizers/sedatives

When indicated, Pertofrane may be used with tranquilizers or sedatives.[1] This allows valuable flexibility in the individualization of therapy to the specific needs of the patient whose mild anxiety or agitation, which accompanies his depression, does not remit to Pertofrane alone.

References: 1. Barringer, T.J.: Psychosomatics 6:326, 1965. 2. Cammer, L.: in Depression: A Practical Approach (Davis, J.M., ed.), New York, Medcom, 1974, p. 26. 3. Lapolla, A.: Am. J. Psychiatry 121:1206, 1965. 4. Krakowski, A.J.: Am. J. Psychiatry 120:494, 1963. 5. Prange, A.J., Jr.: Psychiatric Ann. 3:56, 1973.

Pertofrane®

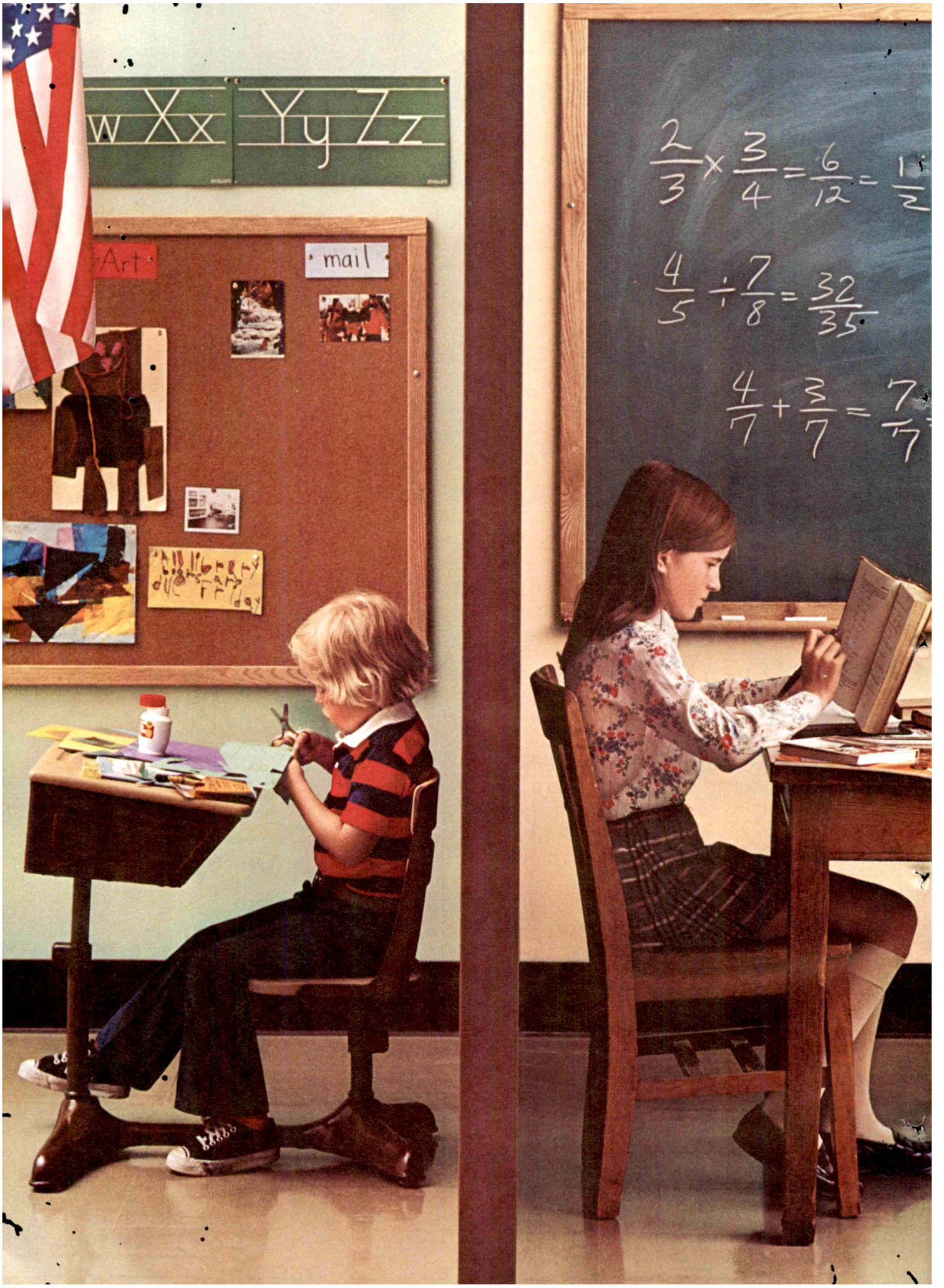
(desipramine hydrochloride NF)

When activity is important

USV PHARMACEUTICALS

USV Pharmaceutical Mfg. Corp.
Manati, P.R. 00701

Indication: For relief of mental depression. **Contraindications:** Do not use MAO inhibitors concomitantly or within 2 weeks of the use of this drug. Hyperpyretic crises or severe convulsive seizures may occur with such combinations; potentiation of adverse reactions can be serious or even fatal. When substituting Pertofrane (desipramine HCl) in patients receiving an MAO inhibitor, allow an interval of at least 14 days. Initial dosage in such patients should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated following recent myocardial infarction and in patients with a known hypersensitivity to tricyclic antidepressants. **Warnings:** Activation of psychosis may occasionally be observed in schizophrenic patients. Due to atropine-like effects and sympathomimetic potentiation, use only with the greatest care in patients with narrow-angle glaucoma or urethral or ureteral spasm. Do not use in patients with the following conditions unless the need outweighs the risk: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (may lower seizure threshold). This drug may block the action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents. Hypertensive episodes have been observed during surgery. The concurrent use of other central nervous system drugs or alcohol may potentiate adverse effects. Since many such drugs may be used during surgery, desipramine should be discontinued prior to elective procedures. Caution patients on the possibility of impaired ability to operate a motor vehicle or dangerous machinery. Do not use in women who are or may become pregnant, or in children under 12 years of age, unless the clinical situation warrants the potential risk. Because of increased sensitivity to the drug, use lower than normal dosage in adolescent and geriatric patients. **Precautions:** Potentially suicidal patients require careful supervision and protective measures during therapy. Prescriptions should be limited to small quantities. Discontinuation of the drug may be necessary in the presence of increased agitation and anxiety shifting to hypomanic or manic excitement. Atropine-like effects may be more pronounced (e.g. paralytic ileus) in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents). Prescribe cautiously in hyperthyroid patients and in those receiving thyroid medications; transient cardiac arrhythmias have occurred in rare instances. Periodic blood and liver studies should supplement careful clinical observations in all patients undergoing extended courses of therapy. **Adverse Reactions:** The following have been reported: *Nervous System:* dizziness, drowsiness, insomnia, headache, disturbed visual accommodation, tremor, unsteadiness, tinnitus, paresthesias, changes in EEG patterns, epileptiform seizures, mild extrapyramidal activity, falling and neuromuscular incoordination. A confusional state (with such symptoms as hallucinations and disorientation), particularly in older patients and at higher dosage, may require discontinuation of the drug. *Gastrointestinal Tract:* anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea. *Skin:* skin rashes (including photosensitization), perspiration and flushing sensations. *Liver:* rare cases of transient jaundice (apparently of an obstructive nature) and liver damage. If jaundice or abnormalities in liver function tests occur, discontinue the drug and investigate. *Blood Elements:* bone-marrow depression, agranulocytosis, thrombocytopenia and purpura. If these occur, discontinue the drug. Transient eosinophilia has been observed. *Cardiovascular System:* orthostatic hypotension and tachycardia. Carefully supervise patients requiring concomitant vasodilating therapy, particularly during initial phases. *Genitourinary System:* urinary frequency or retention and impotence. *Endocrine System:* occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement, and decreased libido and estrogenic effect. *Sensitivity:* urticaria and rare instances of drug fever and cross-sensitivity with imipramine. **Dosage:** All patients except geriatric and adolescent: 50 mg. t.i.d. (150 mg. daily). Dosage may be increased up to 200 mg. daily. Geriatric and adolescent patients should usually be started with lower dosage (25 to 50 mg. daily) and may not tolerate higher doses. Dosage may be increased up to 100 mg. daily. Lower maintenance dosages should be continued for at least 2 months after obtaining a satisfactory response. Mild anxiety and agitation which may accompany depression usually remit as the depression responds. Occasionally, however, a sedative or tranquilizer may be indicated. **How Supplied:** 25 mg. capsules (pink), bottles of 100 and 1000. Also, 50 mg. capsules (maroon and pink), bottles of 100 and 1000; single-dose blister packs, boxes of 500.


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Art
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$\frac{2}{3} \times \frac{3}{4} = \frac{6}{12} = \frac{1}{2}$
$\frac{4}{5} \div \frac{7}{8} = \frac{32}{35}$
$\frac{4}{7} + \frac{3}{7} = \frac{7}{7}$

The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline (primidone) for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards— used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate—As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail— A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

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Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

Mysoline® (primidone)

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initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

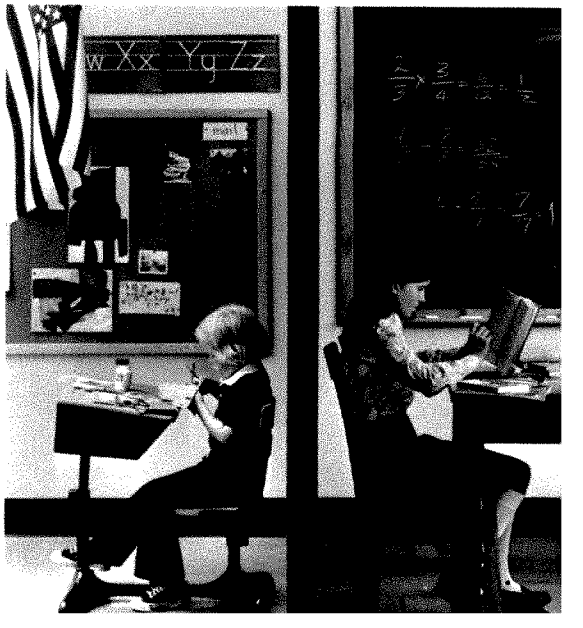
In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (teaspoonful) contains 250 mg. of primidone, in bottles of 8 fluid ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. *37*:49 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. *47*:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. *68*:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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I CLI ICALLY SIG IFICA T DEPRESSIVE NEUROSIS...







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RESULTS OFTEN SEEN IN A WEEK



Because the somatic and psychic complaints that accompany depressive neurosis can interfere with the treatment of the underlying psychopathology—rapid relief of these symptoms is often crucial to therapy.

In 14 double-blind studies of four weeks duration, 339 patients with depressive neurosis received Mellaril. In these studies, 55% of the overall improvement was observed by the end of the first week, and a total of 293 patients (86%) improved during the four weeks.*

Mellaril usually does not cause euphoria or undue sedation and is nonaddicting. Patients, therefore, generally remain alert and better able to respond to therapy. (The physician should, however, caution patients against participating in activities which require complete mental alertness, e.g., driving.)

*Data on file at Sandoz Pharmaceuticals.

MELLARIL® (THIORIDAZINE)

TABLETS: 25 mg and 50 mg thioridazine HCl, U.S.P.

for the short-term treatment of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling. The following reactions have occurred with phenothiazines and should be considered: *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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al and with medical ethics in particular. It seems to us that secular philosophers mostly raise questions, which is useful, but have no answers, while the theologians offer answers but not necessarily the right ones. Regardless of such reservations we recommend to anyone more seriously interested in ethics an introductory text such as Frankena's *Ethics* (12).

The most important recent publication in philosophical ethics, in our opinion, is Rawls's *A Theory of Justice* (13). It has many implications for the ethics of a health system. Rawls examined the concept of social justice designed to enhance fairness. To maximize liberty and to provide equal access to necessary services and commodities is the essential message of his text, which, as we stated before, is embodied in the right-to-health concept.

Another important contribution is Ramsey's analyses of medical ethics (14, 15), in which he defined ethical principles of behavior on the basis of the biblical concept of "covenant," which he saw as existing between physician and patient to maintain the "sanctity of life." Ramsey, a theologian, stated in his book *The Patient as Person* (15) that "caring," or "respect for human life," is the basic moral obligation binding individual medical decisions and the rules of medical practice.

Joseph Fletcher (16), a so-called situational ethicist, appealed to the medical profession through his pragmatism. We are also impressed with Clouser's attempts (17–19) to help physicians clarify their thinking about these issues.

Social and Behavioral Scientists

Important and extensive contributions to the field of medical and psychiatric ethics have been made by social and behavioral scientists. Their concerns are what values *are* rather than what they ought to be, what rules exist, how they are followed or not followed, and how they develop. In general, their point of view is closer to what psychiatrists espouse and can understand. We strongly believe that further important contributions will be made by behavioral and social scientists, particularly by economists, who are concerned primarily with what Dewey (20) called "instrumental values"—the vast array of technological tools and products—rather than with ideological values. Of course, the implementation of many ideological values depends on production, consumption, and priorities. Certainly this applies to medicine and psychiatry as well as to economics.

Psychoanalysts

One particular group that has explored values in depth is psychoanalysts, who have contributed major insights into preconscious and unconscious values. Sigmund Freud's concepts of the superego and the ego ideal (21) have been important and influential and have been taken over by dynamic psychiatry. Psychoanalysis has shaken older thought about rationality and free will—mainly through the introduction of the concept of unconscious conflict. In general, psychoanalysts, such as Hartmann (22) and Marcuse (23), maintain that psychoanalysis does not espouse any particular value system. This idea is not shared by all psychoanalysts, however. Fromm (24), Redlich (25), LaPiere (26), and others have stated that psychoanalysis has definite values and affinities to the values of certain cultures. It seems to flourish only in societies characterized by relative political and social freedom and is essentially elitist.

Lawyers

Finally, we would like to mention lawyers as a most influential group in the field of ethics, although they are not directly concerned with ethical issues unless controversy over such issues reaches courts of law. They certainly do not deal with trifling matters (*de minimis lex non curat*), but most of the major issues and problems in medical and psychiatric ethics sooner or later reach the courts, to be decided in ways that have a great deal of influence over the conduct of medicine and psychiatry. Examples are the decisions of Judges Bazelon and Johnson, which we will discuss further under the heading of the right to be treated.

Because most legislators are lawyers, legislation is another arena in which the legal profession exercises the greatest influence over policy in general and over medical and psychiatric policy in particular. We also want to point out that many consumer representatives and advocates are lawyers. The legal profession has clearly aided the civil rights movement in its defense of the rights and privileges of the committed patient. Some basic ideas on liberty and therapy and the civil implications of the "therapeutic state" have been expressed in the excellent book *The Right To Be Different* by Kittrie (27), who is a legal scholar.

From the preceding discussion it can be ascertained that defining the ethics of medicine and psychiatry today should be an interdisciplinary effort involving interdisciplinary societies and institutes. Scholarly work goes on in universities and in such free-standing institutes as the Hastings Institute of Society, Ethics, and the Life Sciences in Hastings-on-Hudson, N.Y., and the Society and Institute of Health and Human Values in Philadelphia, Pa. Publications of real interest to psychiatry are contained in the reports of the Hastings group, which has published the most comprehensive bibliography on medicine and bioethics to date (28); this bibliography includes a section on psychiatry.

THE RIGHT TO BE TREATED

The right to be treated has become a major and complicated legal issue. The right to treat is of equal complexity. We will not go into the legal complexities of these issues here, except to mention some important legal decisions that as precedents for further decisions will gradually establish some very definite rights of patients. We consider the right-to-be-treated issue far

more than a legal fiction. The array of legal and political problems involved in this issue has been masterfully described and analyzed by Stone (29).

The first major decision affecting this issue was made by Judge Bazelon (30), who ruled in *Rouse v. Cameron* (31) that a criminally insane patient who was committed to a mental hospital had the statutory right to be treated. Bazelon led the way for other landmark decisions by basing his ruling on the arguments of cruel and unusual punishment and the right to due process.

The second decision affecting the issue of the right to be treated was the ruling by Judge Johnson in *Wyatt v. Stickney* (32) that the State of Alabama must provide certain minimal standards of care in its mental hospitals or discharge its committed patients and that the state must appropriate funds for standards that the judge specified. This decision was appealed on the grounds of comity, or conflict between judicial and legislative discretion, and was initially not favored by the psychiatric establishment, which maintained that only a psychiatrist can define psychiatric standards.

The third decision involving the right to be treated was *Donaldson v. O'Connor* (33, 34), in which a paranoid patient who had been hospitalized for 14 years without treatment sued the physicians of the hospital for damages and demanded his discharge. When this case reached the Supreme Court it was ruled that patients who are not dangerous cannot be confined in institutions against their will if they do not receive treatment and if they could survive outside the institution with the help of friends or relatives. The court did not rule on the broader issue of whether all patients in a mental hospital have a right to treatment. The Supreme Court has not yet decided what the legal criteria for commitment are, nor has it upheld the *Wyatt v. Stickney* decision defining minimum standards for psychiatric care.

In another major case, *Robinson et al v. Weinberger et al*, which is pending, the Mental Health Law Project of Washington, D.C., asked a federal judge to order the District of Columbia to create adequate facilities in the community to permit the discharge of patients who do not receive adequate treatment from a state hospital—in this particular case St. Elizabeths Hospital, one of the nation's best. This case goes farther than the others because it does not merely suggest that patients should be "dumped." The ethical gist of the matter is that those few patients who therapeutically need to be confined should be treated adequately in institutions. Patients who are either not to be admitted or are to be discharged and treated in the community should be taken care of under proper conditions created for adequate care. In principle this goal is simple; in reality, due to the conflicting interests of patients, therapists, legislators, and lawyers, the problem is very complex.

Patients do not always want treatment; therapists want to treat, but only under conditions prescribed by them; and legislators may want to provide treatment but may not want to or are not able to provide funds for such purposes. These inequities have already produced some interesting legal opinions; more are undoubtedly to come.

Regrettably, the psychiatric establishment has assumed a conservative and defensive stance in these matters. First, it was not the psychiatrists but civil libertarian lawyers who pressed for changes. This created—to say the least—embarrassment and resistance among psychiatrists who worked in institutions; psychiatrists not associated with institutions still managed to maintain a detached apathy. Stone (29) strongly pleaded for more enlightened attitudes among psychiatrists, even for more enlightened self-interest. Legal precedents are leaning toward the legal and moral responsibility of the individual psychiatrist for the adequacy of treatment in public mental institutions. Legal suits have moved in the direction of holding individual psychiatrists responsible for the inadequate treatment in the public institutions they serve. Ultimately, of course, it is the patient's interest and not the expert's that needs to be considered and safeguarded.

THE RIGHT NOT TO BE TREATED

In general medicine and surgery in the United States today no competent adult patient can be treated without his informed consent. The parents' or guardians' consent is required for children; for adolescents the decisions are legally in the hands of parents or guardians, but, de facto, adolescents make their own decisions in most cases. Only psychiatric and mentally defective patients committed to institutions are (in most cases) denied this right (35). This means not only that they cannot make decisions about medical and surgical procedures, but also that they are forced to submit to them if this is deemed desirable by their physicians or the administrative head of the institution. They are not permitted to decide on their own whether they will be subjected to such serious and stressful procedures as ECT, insulin coma treatment, prolonged major psychopharmacological therapy, or psychosurgical procedures and conditioning treatment to modify behavior. Another procedure that all too often has been carried out without the patient's consent is sterilization. The restriction of the rights of these patients is based simply on the premise that a patient who has been committed to a psychiatric institution does not know what is best for him.

We contend that mental illness and commitment are not sufficient conditions for the restriction of the right to informed consent. To take away this right requires that evidence be presented in a court of law that the patient is incompetent. Of course, there is room for explanation and persuasion to induce a patient to consent to a medical, surgical, or psychiatric procedure that is in his interest, but there is no room for coercion and brainwashing. If a patient who is not declared incompetent does not want to be subjected to any particular treatment his judgment ought to be honored, even

if it is in disagreement with a clinician's judgment.

The right not to be treated was upheld in a federal court in *Winters v. Miller* (36). In reality, even voluntary patients in psychiatric hospitals are often subjected to strong pressures to accept treatment—a practice that we find objectionable from an ethical point of view even though the clinician's intent is benevolent. We know of patients who have consented to ECT and then—after being subjected to this severe treatment—have reversed their consent, but whose psychiatrists insisted that consent could not be reversed and treatment had to continue. We believe that this is wrong and that patients have the right to withdraw their consent at any time. In general, we feel that modern psychiatrists should be wary of taking on a parental and authoritarian function.

One important point, brought to our attention by Hardin Branch, is that if patients have the right to refuse treatment, individual psychiatrists as well as the profession of psychiatry must not be held responsible for social evils that may be the consequences of such refusal. Drunken drivers may refuse treatment by psychiatrists or by such peer groups as Alcoholics Anonymous; it is then up to society to devise other forms of control. It might be said that the control of deviance—largely forced upon psychiatry by society—is more of a curse than a boon to our profession and that we should be happy to leave it in the hands of the law for enforcement.

Halleck (37) discussed the following conditions under which coercive psychiatric treatment is justified: the patient must be dangerous to himself or to others, there must be reasonable probability that treatment will be beneficial to the patient or to others, and the patient must be incompetent to judge the effect of treatment. We agree, but we would go farther. Coercive treatment due to a patient's being dangerous or incompetent must be ordered by a court of law on the basis of evidence of such behavior and the value and availability of the treatment. In addition, treatment that benefits primarily others (presumably Halleck meant family, peers, or the community at large) should be conducted only if a court rules it. Actually, we cannot think of any good examples of such treatment unless it also benefits the patient.

One difficult problem is posed by patients who are dangerous to themselves. The question is, What is danger to oneself, what is self-destructiveness? We are not discussing minor patterns of behavior that are not in the patient's interest, but clearly suicidal behavior. The acutely suicidal patient may require prompt treatment that he does not wish to receive, although we recognize that there are normal persons who make a clear and cool decision to end their lives and who, in our opinion (though not in the opinion of many others), have a right to make such a decision. We know, however, that many severely depressed patients will not repeat suicide attempts once they are cured of their depression. In these cases a specific type of incompetence is involved. The judgment of such incompetence should be made by a court of law, if possible; we realize, however, that there are cases in which the legal approach would not be expedient and immediate action by psychiatrists is necessary.

RIGHTS OF PSYCHIATRIC PATIENTS

In general, the concept of informed consent has become the most important, most complex, and most confused concept in medical ethics. It essentially delineates or should delineate the patient's right to determine what kind of health-related procedures health personnel may or may not carry out. Many authors (15, pp 1–58; 38) also see this as the basic moral relationship underscoring all patient-physician contracts for medical treatment and human investigation. We believe that major changes regarding the rights of psychiatric patients that are comparable to the growing impact of effective psychopharmacology and the widening influence of psychotherapy on fairly large segments of the population are in the making. We do not wish to say, however, that a successful "rights" revolution has occurred. Many of our large public hospitals for chronic patients are still total institutions in the sense that they make all the decisions for patients in their care, even though on paper at least the general rights of patients have been spelled out.

With some shame we state that most of the changes in establishing patients' rights were not brought about primarily by psychiatrists but by civil libertarians led by lawyers, and that the most important decisions were made by enlightened judges. Of course, there have been superintendents of state hospitals who fought for more humane conditions in their institutions; concerned groups of psychiatrists; and, to mention one man in particular, the occasionally maligned Szasz, who unfortunately all too often has expressed good views in an unacceptable form.

Psychiatrists reject the idea that they are self-serving or that they are preserving an undesirable status quo. Indeed, we believe psychiatrists honestly think that in many cases compulsory treatment is more important for a given patient than his freedom. Psychiatrists in general opt for medical informality rather than for legal formality and for flexible medical therapeutic action rather than bureaucratic and legal surveillance. However, the tide of change has been in the direction of protecting the liberty of patients and also in the direction of growing legal and bureaucratic restrictions of the scope of psychiatric care. We will discuss the ethical but not the legal aspects of general rights of patients, commitment procedures, and the right to be treated and the right not to be treated.

General Rights

The changes in establishing patients' rights are to a certain extent expressed in statutes and regulations of the various state departments of mental health. These concern what one might call general rights of

patients (38)—such civil rights as the right to vote and to contract to hold or convey property. Only mental incompetence that has been proved and recognized by a court of law—not to be confused with the broader and different category of mental illness—ought to be a cause for the abrogation of these rights of patients in psychiatric institutions. It is specifically recognized that to deprive a patient of his belongings is apt to injure the patient's right to respect and dignity. Patients in mental institutions have the right to receive and send uncensored mail and to make and receive telephone calls, unless there are specific reasons—to be stated in writing by the administration of the institution—that make these privileges unwarranted for the patient or for society at large. Specifically, patients can be restrained or placed in seclusion only if they are dangerous to themselves or to others, and such restraints can be issued only with the consent of the chief administrator of the institution and must be constantly monitored and justified.

It is generally agreed that physical and chemical restraints ought to be used rarely and under no circumstances should they be used punitively. Another major ethical area of concern is the obligation to reduce the stigma of mental illness and, more specifically, to increase the patients' chances for fair employment and social interactions. Obviously, this is more easily said than done. In general, of course, we recognize that no legal or administrative provisions for these actions exist in many states and that even if they do exist they are not enforced or are circumvented.

Commitment Procedures

The most important right of the institutionalized patient is his basic right to the maximal degree of liberty that is compatible with his welfare and with the interests of society. Possible conflict between the individual's rights and the rights of society pose the most difficult problems in questions of treatment and research. These problems are particularly sensitive when the patients or subjects come from deprived or disenfranchised populations, e.g., psychotic patients, the mentally retarded, prisoners, and children. Much more careful work must be done before general ethical guidelines can be established.

These matters are regulated in all states by statutes defining various patterns of entry and discharge from institutions. In general we have observed in the last decade a very marked tendency to reduce the number of court commitments of psychiatric patients and to replace them with emergency and voluntary admissions. There has also been a tendency to discharge patients after a short period of hospitalization rather than keep them indefinitely. Psychiatrists generally do not like formal commitments. They consider it traumatic for most patients to be exposed to the stress of formal notice, representation by counsel, and cross-examination in court. They prefer the more informal emergency commitment.

In fact, psychiatrists feel threatened by legal challenges to their professional decisions. A recent study (39) suggested that this "professional" anxiety leads to poor clinical care: when psychiatric commitments were challenged by lawyers on behalf of their patient-clients in a public hospital in New York City there was a marked tendency for these patients to be discharged without a court appearance. This occurred in spite of the fact that the judges tended to universally support psychiatric decisions and the fact that the discharged patients were at least as sick as patients whose commitments were unchallenged and who remained hospitalized.

Libertarian lawyers are opposed to informal procedures, which in reality deprive the patient of his freedom, specifically his civil rights, without due process of law. Even so-called voluntary admissions, which have become the dominant mode of admission in many cases, "trap" the patient in a mental hospital because he is often not permitted to leave without giving written notice. One should realize, however, that it is not easy for a patient to leave a general hospital once he takes his shoes off; this is true even in shoe stores.

The only reason for formal commitment—and in our opinion for so-called emergency commitment as well—is danger to the patient or to others. Civil libertarian lawyers maintain that the need for treatment is not a sufficient reason for emergency commitment, a point of view that is not accepted by an appreciable number of psychiatrists. These lawyers also point out that it is difficult to predict dangerous behavior and that in the statutes of all states—with the exception of California, Massachusetts, and Maryland—the definitions of "danger" are very vague. One of the most difficult situations arises when a decision must be made to hospitalize a potentially suicidal patient against his will and to determine what type of admission he needs. As we mentioned before, we believe that suicide by patients with psychotic depression—presumably a true disease of unknown and complex etiology—ought to be prevented. Rapid commitment for these patients is essential, and therefore admission based on emergency certificate is not only clinically but ethically justified for them. The libertarian and clinical points of view are pitted against each other in two interesting articles, one by McGarry and Greenblatt (40) and one by Szasz (41). On the whole, we consider the present trend to be toward truly voluntary admissions.

During the last few years psychiatrists in the Western democracies have sharply criticized alleged psychiatric practices in the Soviet Union, where it is alleged that persons with politically undesirable actions and opinions who are not suffering from mental illness have been confined against their will in mental hospitals. Soviet psychiatrists have denied these charges. If they are true—and there seems to be some truth to these stories—this is a horrible abuse of the physician's powers and duties. As Judge Bazelon (42) and others (43) have pointed out, however, it is even more horrifying that large numbers of patients in the United States and in other democratic countries are deposited

in custodial welfare institutions, not because they suffer from mental illnesses, but because they are unwanted by their communities.

BEHAVIOR CONTROL

The problem of informed consent can be formulated as two questions: Who agrees to a decision? and Who decides what for whom? These questions are particularly pertinent for treatments that involve behavior control. According to London (44), who wrote an illuminating book on this topic, behavior control may be defined as attempts to influence persons to do somebody's bidding. It is a topic much broader than psychiatric therapy and includes vast segments of political propaganda, commercial advertising, moral and religious education, and rehabilitation of deviant persons.

The main problem in psychiatric behavior control is to delineate who has the right to set goals and approve methods and to safeguard those who do not wish to change their behavior or those who are not aware of what happens to them in the process. As methods of behavior change and behavior control become more efficient the problem is exacerbated. The reaction of concerned citizens inside and outside the profession of psychiatry to psychiatric methods of behavior control is strong, especially regarding those methods of behavior control which produce drastic, irreversible, and even dehumanizing changes.

The main categories of behavior change are the psychotherapies, the pharmacological therapies, the psychosurgical therapies, and the methods of genetic engineering. The impact of each of these methods is markedly different from the other because they vary in their capability to produce desired changes, their production of undesirable effects, their ease of application, their irreversibility, and the patient's awareness of their effects. Most important is the question of whether or not the patient is in a situation to give informed consent. That the patient should be protected from outright coercion may be a readily accepted idea and may be formalized in statutes and regulations, but that he should be protected from subtle pressures is a far more difficult idea to accept and to regulate. We know that individuals can be "brainwashed" to accept ideologies that a political regime considers desirable. The same is conceivable as far as psychiatric treatment is concerned.

Psychotherapy

The problems of behavior control vary with the different methods of therapy. Insight-producing psychotherapy as a method of behavior control incurs relatively few risks. The method is slow and limited to a relatively small target population. When behavior changes are produced the accompanying increased awareness enables the patient to monitor the behavior changes, at least to some degree. The more persuasive psychotherapy becomes, the less likely is such monitoring.

In conditioning psychotherapeutic methods the patient is less able to check undesirable changes and side effects. The gruesome movie *A Clockwork Orange* showed how an extremely aggressive asocial teenager turned for a while into a passive and helpless person. The danger of creating dehumanizing effects in a patient who has not given truly informed consent is a serious ethical breach, especially if the target symptom is undesirable political behavior and the technique is aimed to produce mind control. As far as we know this has not happened, but conceivably it could happen.

Pharmacotherapy

The risk of dehumanization is potentially even greater in behavior change produced by drugs. At present the power to change behavior by means of drugs is limited because the drugs used are relatively inefficient (although by no means harmless). To overtranquilize a patient is certainly to threaten his human freedom and dignity, apart from the danger of incurring the serious side effects that all tranquilizers produce. There is good reason for concern for the future, when there will be more powerful drugs with specific effects. At present our society is involved in exercising strict control over all drugs that are potentially harmful. Such control, particularly over the pleasure-producing drugs, is difficult and can be maintained only at high economic and social cost, as the addiction problem in many countries demonstrates.

Psychosurgery

The gravest concerns have been expressed over the use of psychosurgical techniques to produce behavior changes. The reason for this apprehension is the irreversibility of the changes produced by neurosurgical lesions and the patient's inability to protect himself against the consequences of such lesions once they have been produced. This is equally as true for the crude lobotomies that were carried out on a large scale in the 1940s and 1950s, which had definite dehumanizing effects in addition to their therapeutic results, as for today's refined microsurgical techniques. The fact that it takes highly skilled personnel to carry out such operations keeps the number of patients at risk relatively small; however, there is no statistical justification for harmful and unethical procedures.

Genetic Engineering

We also want to mention eugenic measures or methods of genetic engineering to alter human behavior (45). Since the disastrous and inhuman involuntary sterilizations that occurred under National Socialist rule in Germany, no serious scientist—except the physicist and Nobel prize laureate Shockley—has endorsed compulsory eugenic sterilization. There has been some discussion (46–48) about future approaches, such as cloning, to improve the human race. How-

ever, this is one problem that today's psychiatrists need not worry about yet.

A policy position of the National Institute of Mental Health on this subject (49) highlighted the wide and increasing use of behavior modification, including behavior change by drugs and neurosurgical techniques. At present, according to this publication, 600,000 persons receive such treatment. NIMH recognized the value of behavior modification but cautioned therapists and patients that therapists are accountable to patients and their representatives for the effects of such treatment; they must weigh the risks and benefits and inform the patients about the effects. This is particularly important if patients are in mental hospitals, if they are senile, or if they are prisoners because the imbalance of power between these patients and their therapists warrants particular caution and careful monitoring.

CONFLICTS OF INTEREST IN THERAPEUTIC GOALS

The right to be treated or not to be treated, in general, and the right to agree to or to decline behavior modification treatment, in particular, often raise questions of conflicts of interest. Does the therapist satisfy the interest of the patient or his own interest? To a certain extent the therapist always satisfies his own interests to make a living, to gain prestige, to fulfill his moral need to help, or to increase his knowledge; within certain limits this is entirely permissible and desirable. However, in many situations the therapist serves a third party, particularly when he is an employee of an institution whose interests are not necessarily identical to the interests of the patient. Third-party interest can also occur in solo private practice when a third party requests the treatment or pays for it. This third party may then overtly or covertly, grossly or subtly, influence the psychiatrist to do his bidding. This is often the case when children or adolescents are in treatment or when a patient's spouse pays for therapy. It also happens when persons on whom the psychiatrist depends, or to whom he is or wishes to be obligated, influence treatment goals. The most malignant of these instances concern the individuals who are imprisoned in psychiatric institutions for political reasons (9). We believe that subtle forms of pressure by third parties are applied often and everywhere—not only under dictatorships. We are also convinced that psychiatrists are not sufficiently aware of these pressures.

Conflict of interest is one facet of the wider problem of what values, especially what conflicting values, are served by psychiatry and how the values of therapists, patients, third parties, and society at large may differ markedly from one another. The problems, particularly in prisons or in relatively monolithic organizations such as the armed forces, may be insoluble. What we plead for is greater awareness of these value conflicts. Unfortunately, psychiatrists have a marked tendency to assume that they operate in a valueless frame of reference; although they keenly notice their patients' conflicts, they are less inclined to see their own. The psychiatrist, no matter what his theoretical orientation, is almost always a party to conflict, and he needs to be aware of this (50).

PRIVACY AND CONFIDENTIALITY

Privacy is a pervasive ethical concept with wide personal, political, and legal implications. It is of central importance for psychiatry. Greenawalt (51) defined it as the right to be left alone. To exclude the public to a certain extent is one of the great values of the free world, according to Westin (52). Privacy refers to the control of the flow of information about a person to others, to the invasion of one's private sphere (one's home and possessions—as in searches and wiretapping), and to the right within limits to live according to one's own wishes. These concerns, particularly the intrusion into and public revelation of a person's inner world (his conscious and preconscious thoughts, feelings, and volitions), are extremely important in psychiatric treatment. One important aspect of this larger issue of privacy, with the implied right of the patient to accept or reject treatment, is the narrower issue of confidentiality.

Private Practice

The concept of professional confidentiality refers to the obligation of certain professions to respect information revealed in professional activities. Professional secrecy in medicine is mentioned in the Hippocratic Oath. It is protected in many states and countries by privileged testimony in courts of law in recognition of the fact that information obtained in the relationship of physicians and their patients is particularly sensitive and may lead to serious occupational and social damage if revealed in public.

Again, in this article we are not concerned with the legal complexities of privacy and confidentiality but with basic ethical considerations. Are there situations in which confidentiality ought to be relaxed in the interest of the patient or in the interest of others and society at large? Benefits, risks, and potential harm must be balanced against each other, and no general valid ethical guidance can be offered, although most frequently psychiatrists will act according to what they consider to be in the best interest of the patient.

The problems of confidentiality are highlighted in the "warning" case of *Tarasoff v. Regents*, which is pending in California. This case involves a therapist who revealed the possible risk of homicidal behavior in a dangerous patient to the police, who could not act upon this information, but not to the victim, who was later killed by the patient. The psychotherapist and the police were subsequently sued by the family of the victim. Although this case is unresolved, the current tendency of the courts is not to make public disclosure of confidential patient information mandatory. This legal situation intensifies the ethical dilemma for the psychi-

atrist in this type of case, in which the welfare of society is clearly balanced against personal privacy. This is an area in which professional ethics must provide the ultimate guidance and in which the best interest of the patient as an ethical touchstone comes under the greatest strain.

Psychiatric Research

Both privacy and confidentiality are invariably tested in the context of psychiatric research. In most types of clinical psychiatric and psychological research as well as in standard clinical observation the patient is subjected to more or less intensive and extensive exploration of his personality and social relations. Even when this is done with the patient's explicit consent, he is hardly ever aware of the depth and meaning of such explorations—especially in more thorough investigations. If this is done in the interest of the patient (as it is in most therapeutic encounters), no conflict will arise. If the revelations are used for research and publication, even with the explicit consent of the patient, there may be ethical problems, such as there were in the case of *Doe v. Roe*, which was recently returned undecided to a lower court by the Supreme Court.

Investigators and clinicians who report their data in journals and at teaching conferences try to conceal the identity of their patients. Usually these disguises are rather ineffective, especially today when the "nonreading" charity patient—on whom much of the research has been done in the past—has virtually disappeared. It is much more likely today that psychiatric patients will read full accounts or vignettes of their case histories. The patient may find it difficult to accept the published findings of the investigator, or he may find the conclusions outright damaging and then sue, especially if he feels hostile toward the psychiatrist.

Group and Couples Therapy

The problem of confidentiality is particularly sticky in group and couples therapy. Patients are under no professional ethical obligations of confidentiality. Although the leader of a therapeutic group may stress the desirability of not discussing the content of the therapy outside the group, he cannot enforce this effectively. Members of groups ought to be at least apprised of this risk. In contrast to ex-President Nixon's practice, psychiatrists have learned to be scrupulous about obtaining permission to record by sound, videotape, or film. Such documents ought to be used with great care and tight security controls.

We would also like to refer to the type of breach of confidentiality that should be the easiest to control—professional gossip. It is our impression that many members of the mental health professions talk too loosely. Otto Pötzl once remarked that any psychiatric diagnosis is a form of slander and ought to be defended in court. Has the stigma of having a mental illness changed, except among a small group of intellectuals? Shouldn't we exercise more restraint?

Data Systems

There has been considerable concern over leakage of information through computerized data systems. With national health insurance and, perhaps, national health data systems just around the corner, this concern seems justified. Experts in the field, such as Westin (53) and Sawyer and Schechter (54), seem less worried about such dangers. Actually, the dangers already exist in traditional records. However, with appropriate legislation it would not be difficult to make these data sufficiently secure and to prevent unauthorized access to them. It is also of importance to destroy data that are not essential and to record only minimal data if they are likely to become public.

HUMAN EXPERIMENTATION

Ethical rules and statutes governing biological and psychosocial research have changed radically since the trials of the National Socialist experimenters accused of crimes against humanity. The general deliberations of the Nuremberg Court have become known as the Nuremberg Code. The rules and regulations that govern research in the United States have been patterned after the 10 points of that code and have had profound influence on attitudes toward biomedical and psychiatric research (55). All government-supported research in the United States must abide by these rules. In our opinion, these rules have greatly helped to protect the patient and have made investigators more sensitive to the experiences of their subjects, although, in the opinion of some, they also have had an inhibiting effect on human research.

Although the committees of medical centers scrutinize applications for research, they almost never monitor the ethical conduct of the research. Psychiatric patients, particularly institutionalized patients suffering from severe disorders, are more vulnerable than other medical and surgical patients. They require greater protection and special considerations in relation to investigative procedures. The three basic considerations are free and informed consent of the patient, the weighing of possible benefits and risks, and the avoidance of suffering and danger. Psychiatric patients who have been declared incompetent are not able to consent freely to research. Consent has to be given for them by relatives or guardians. Most concerned experts assume under these circumstances that research should be carried out only if it benefits the patient directly, if it holds promise that the patient might benefit in the foreseeable future, or if it benefits at least a group of patients suffering from an identical or closely related disease or disorder.

The informed consent rule also eliminates observations by concealed observers. Caudill's research in the early 1950s would not be approved under current rules (56): he stayed on a psychiatric ward under the pretense of being a patient. This holds also for the

more recent investigations of Rosenhan (57), who had a group of people enter a mental hospital as "fake" patients, even though his observations were of considerable value and could not be duplicated in their entirety by a regular participant-observer. The research of Milgram (58), who deliberately misinformed paid volunteers to test their capacity to obey cruel commands, would also not be allowed.

One particular problem in judging and balancing positive and negative effects of research is posed by research that investigates organizations and institutions. Consent for research is usually given by the leadership of an organization. The rank-and-file members of the organization are usually not given the chance to agree or disagree. Because one usually cannot predict the impact that the research will have on the organization or its members, the participants may have accepted research that proves to be highly critical of the organization and damaging to its members. Probably the best way to safeguard the interests of organizations and their members is to let them scrutinize scientific reports before publication. This will at least give them a chance to correct potentially damaging or insensitive statements. It will, on the other hand, curb freedom of research. Just as psychosocial research on underprivileged minorities in ghettos or on people in developing countries is critically reviewed before consent is given, psychosocial research in organizations and in the community should be subject to more stringent examination before it can take place.

THE ETHICS OF POLICY DECISIONS

We discern a definite trend in medical ethics to move from considerations of the physician-patient relationship to broader ethical considerations of policy decision making. The right-to-treatment problem and the regulations governing experimentation are pertinent examples. In general there was little broad and systematic policy planning outside the public mental hospital sector until World War II. Most of the planning until then had been haphazard, without clear formulations of goals, methods, or costs. During World War II and in the period following the war, when NIMH emerged, planning became quite deliberate.

From the very beginning NIMH established its main goals on the basis of a philosophy embodied in public health. These goals included broader and more equal access to mental health care and a program of prevention. The former was a laudable but difficult to implement goal because of its cost and a lack of manpower; the latter was utopian in the face of the amount of knowledge about preventing mental illness. There is little doubt that after a good start much of the community mental health movement has slowed down.

The point we wish to make is that certain ideological values guided the first professional planners in psychiatry. They knew that implementation of their goals would require vast economic resources, which were available only to a limited degree because the political leadership had other priorities than health and mental health. To persuade legislators to change their value priorities became a crucial task.

Possibly the most important clarification of psychiatry's ideological and technological values would be to define with the national leadership what the practical job of psychiatry is: to diagnose, treat, and prevent a relatively defined number of psychoses? to perform this task on a number of neurotics? to make unhappy and incompetent persons happy and competent? or to tackle poverty and civil and international strife? We are convinced that psychiatrists must beware of diffusion, collusion, and utopian endeavors and state their tasks in the form of clear priorities.

Today, on the eve of the initiation of a national health insurance program, one of the principal policy questions for psychiatry is to what extent psychiatric services, training, and research will be paid for and who gets paid for them. Undoubtedly this will be of the greatest importance for patients and practitioners alike (59). APA is keenly aware of this and is trying to persuade the nation's legislative and administrative leadership to enact proper legislation and allocation of funds. The arguments for such steps are largely ethical in terms of the needs of suffering populations and in terms of fairness and justice.

PSYCHIATRY AND CHANGING MORAL VALUES

Professional persons, including psychiatrists, do not make moral rules or change them. They accept or reject the rules of their culture and interpret them for themselves and their patients. In a pluralistic society this is a complex and often confusing process. Psychiatrists vary widely in their own moral views, as Rogow (60) has shown. Some are unusually flexible and permissive; others are remarkably authoritarian and paternalistic. We mentioned the current general trend in industrialized democracies to decrease authority and to interpret ends and means pluralistically (61). We have also referred to many aspects of a new balance between consumer power and professional power —the former is increasing and the latter is decreasing. Underprivileged minorities, particularly blacks and Spanish-speaking Americans, have gained much in status and rights. Women have moved toward socioeconomic equality. These changes in attitude are paralleled by a trend toward equality of opportunity for psychiatric treatment.

There have been marked changes in the prevalent morals in North America since the immediate post-World War II period. The attitudes toward self-destructive acts and victimless crimes have become more relaxed and less punitive. In psychiatry these changes in attitude have affected alcoholics, who are now considered in need of control and treatment rather than punishment. Less change of opinion has taken

place in regard to users of other drugs (62); in general, society continues its vendetta against even harmless addiction. Some psychiatrists have feebly expressed their opposition to such repressive and punitive points of view, with little avail.

Remarkable changes of attitude have been taking place in the sexual mores of large segments of the population of the United States. Greater permissiveness concerning the varieties of sexuality and sexual behavior outside of marriage is obvious. Sexuality—including masturbation—is considered to be, in the words of Masters and Johnson, a natural act.

• After a referendum APA struck homosexuality from its diagnostic nomenclature and replaced it with the term "sexual orientation disturbance." Obviously, one can no more vote on what a disease is than one can vote on whether the moon is made of green cheese, but one can vote on rights and sanctions. The question is not whether homosexuals are sick or deviant but whether they ought to have the opportunity to work toward sexual change if they choose to and whether they ought to be punished and discriminated against. Most psychiatrists would agree that homosexuals ought to have the opportunity either to attempt to change or to adjust optimally to their condition. They also ought to be protected from socioeconomic discrimination and legal harassment. With some clearer ethical thinking, APA's embarrassing referendum could have been avoided.

COMMENT

There is one area in which a psychiatrist made a fundamental contribution to ethics: Freud's discovery and formulation of unconscious motivation and conflict. The question of the limits of free will and of responsibility were raised once more with Freud's discovery, and the complexity and confusion became greater than ever. According to most ethical doctrines, one cannot be responsible for one's unconscious acts. However, every act, whether unconscious or not, has antecedents and consequences. It is the primary task of psychiatry to study antecedents and consequences of behavior that we call abnormal and to apply methods of intervention to alter such behavior. In pursuing this task we will learn more about the limits of will and responsibility. If we do not expect quick solutions to confusing problems and pseudoproblems, we may be able to make our contribution to an age-old theoretical and practical question.

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Current Trends in the Training of Family Therapists

BY EDWARD W. BEAL, M.D.

The author describes and compares training programs for family therapists at 15 centers in 9 major U.S. cities, using a scale that reflects the spectrum of theoretical approaches to family therapy. At one end of the spectrum are the centers with an experiential orientation, at the other end are those with a structural orientation, and in the middle are those which combine these orientations. He concludes that although creative theoretical advances are being made at a few of these centers, the quality and design of training at the remaining centers suffer from the absence of an adequate diagnostic nomenclature for family therapy and from an insufficiently designed conceptual framework.

THE DIVERSE NATURE of family therapy training programs is related to the evolution of the "family therapy movement" during the past 20 years. At the end of World War II psychiatry was dominated by a theory that was oriented toward the understanding of individual psychological functioning. The family of an individual in therapy was important only as it affected the relationship between the individual and the therapist. Collateral visits with family members were occasionally held, but these were not the focus of treatment.

The interest in group psychotherapy that emerged after World War II represented a major change in the theory and technique of understanding psychological functioning. Although the focus on the therapist-patient relationship was then diluted and the therapy process was shared by other group members, the family of the patient in group therapy still remained therapeutically in the background.

A significant technical change for the family therapy movement occurred with the birth of family group therapy, when the practice of conducting group psychotherapy with naturally occurring groups rather than with artificially composed groups began. This change was primarily technical rather than theoretical. Therapeutic intervention was then focused on the relationships among individuals related by family ties rather than on the relationships among individuals united by various categories of psychological functioning.

From these technical and theoretical changes has emerged a large body of information on group process that has been used by family therapists who focus on the group process within a family. Since 1960, multiple-family group therapy has emerged as a logical progression of the treatment of the individual family as a group (1).

As the theory of group process and its technical application to family groups has emerged, increasing efforts have been made by psychoanalytically oriented psychiatrists to study the relationship between an individual's psychological functioning and the environment of his family. A significant effort has been made to extend individual psychoanalytic theory toward an understanding of the individual in his relationships with his family.

Thus far in this paper the focus has been on those forms of family therapy in which the practice and techniques rather than the theory of family therapy distinguish family therapy from other therapeutic procedures. During the past 20 years, however, a major theoretical shift has occurred in which family therapy has become more a new theoretical orientation toward the understanding of emotional functioning and less a method or technique defined by the number of people in a room. This new theoretical understanding of emotional functioning and of family therapy has been influenced by communications theory, general systems theory, and family systems theory. Multiple-family group therapy, in this theoretical frame of reference, focuses on family emotional processes rather than on group process as the method of therapeutic intervention.

CLASSIFICATION OF FAMILY THERAPY THEORY

Any attempt to assess current trends in the training of family therapists must consider the significant differences in theoretical viewpoints and technical applications of family therapy between and within various training programs.

To compare the diverse theoretical viewpoints of family therapy training centers, I have used a modified form of a scale published by the Group for the Advancement of Psychiatry (2). This scale is designed to reflect the spectrum of theoretical approaches to the understanding and treatment of families. It demonstrates contrasting ways of understanding the ex-

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Dr. Beal is Clinical Instructor, Department of Psychiatry, Georgetown University Medical School, Washington, D.C., and Chief Child Psychiatrist, Community Psychiatric Clinic, Bethesda, Md. Address reprint requests to him at 8830 Cameron St., Suite 603, Silver Spring, Md. 20910.

pression of emotional tension or affect in a family. As one moves from the A to the Z end of the scale, less theoretical and technical emphasis is placed on the direct expression of affect and more on the structure of family relationships. The theory at the A end of the scale is categorized as more experientially oriented, the theory at the Z end as more structurally oriented, and the theory in the middle (M) as a combination of these two orientations. The experiential-versus-structural approach as a method of comparison has been increasingly used in the past few years. This method is one of many ways of evaluating the field of family therapy.

This study is designed to illustrate similarities and differences that exist among theoretical approaches and training programs. It is not designed to evaluate the superiority of one theory or therapeutic approach to another. My research has been based on the assumption that training methods and techniques follow logically from theoretical understanding. In addition to comparing these theoretical understandings, I attempted to answer the basic questions of who is being trained, by whom, what is being taught, where is it occurring, and how it is being accomplished.

I made no attempt to consider all of the family therapy training centers in the United States. Rather, I selected a cross section of 15 centers in 9 major cities on the basis of the prestige of individual teachers at these centers, the number of publications produced by authors from the centers, and the frequency with which symposiums, seminars, or workshops were offered. The information contained in this study was gathered through correspondence with directors of education of family therapy programs, innumerable personal conversations with trainees and teachers, many site visits, and the reading of training brochures.

The criteria I used in comparing the training centers included the following: theoretical orientation, supervisory methods, technical interventions, trainees, trainers, curriculum, location, and methodology.

CLASSIFICATION OF TRAINING CENTERS

A: Experiential Orientation

Centers that fall at the A end of the theoretical scale include the following: the Menninger School of Psychiatry, Topeka, Kans.; the Family Institute of Chicago, Ill.; the University of Wisconsin Medical School, Madison, Wisc.; the University of Colorado Medical School, Denver, Colo.; and the University of Minnesota Medical School, the Midwest Family Therapy Institute, and the Amherst H. Wilder Child Guidance Clinic in the Minneapolis-St. Paul, Minn., area.

The assumption of therapists at the A end of the scale is that relationships are significantly influenced by the expression or inhibition of affect or emotional tension. Direct expression of this tension or affect is therefore appropriate and indicated for the maintenance of healthy relationships in a family. Therapists at the A end of the scale use group psychotherapy theory and techniques. The modality for emotional growth is considered by these therapists to be the therapeutic relationship between therapist and patient. They emphasize the subjective experience of therapy and the subjective awareness and intuition of the therapist to guide the therapy. Some of these therapists conceptualize family therapy as a technique to facilitate individual psychotherapy. The A therapists also consider indications and contraindications for family therapy.

The process that is primarily studied by therapists at the A end of the scale is the one that exists among members of the family group in therapy and between each of them and the therapist. A variety of techniques is used within this framework, including family sculpting, psychodrama, Gestalt therapy, behavior modification, encounter group techniques, interpretation of unconscious conflicts, and group process techniques. Teaching methods at the centers named above include lectures, literature seminars, small group discussions, experiential techniques, and live and videotaped demonstrations of family interviews by faculty and students.

Teachers at these centers are more often from psychiatry and social work than from other disciplines. The trainees in the medical school programs are psychiatry and psychology residents, medical students, and practitioners from the community. The family therapy institutes tend to accept only postgraduate students for course work and supervision on a part-time basis. Supervision of trainees is frequently done through live or videotaped observation of the trainee's sessions with families or through group discussions; rarely does it involve the traditional one-to-one type.

Carl Whitaker, M.D., of the University of Wisconsin Medical School, is the most noted professional from any of the centers at the A end of the theoretical spectrum named above. Unlike some leaders in the family therapy movement, Whitaker is less theoretically oriented; his outstanding strength is his creative, intuitive, and personal approach to treatment. He has an intrusive but gentle style with families and assumes a variety of roles as a therapist. He also makes ingenious use of cotherapists. Trainees are fascinated by his technical skill, but many state that his style seems too unpredictable to be learned. Whitaker is particularly skillful in his use of extended family members, especially grandparents, whom he encourages to attend family sessions.

The training at the remaining centers is done by individuals who are practicing family therapists. These therapists teach from their own experience, by reviewing the appropriate literature, and by attending workshops. The quality and design of the training at these centers appears to suffer from the absence of an adequate diagnostic nomenclature for family therapy as well as from an insufficiently designed conceptual framework.

Z: Structural Orientation

Centers that fall at the Z end of the theoretical scale include the following: the Philadelphia Child Guidance Clinic in Pennsylvania and the Georgetown University Postgraduate Program in Family and Systems Theory and Family Psychotherapy and the Groome Child Guidance Center in Washington, D.C. The assumption of therapists at the Z end of the scale is that emotional tension leads to a process that manifests itself in the structure of family relationships. They believe that this tension is maintained or resolved by the nature of the communication pathways or the relationship system through which it is transmitted. Some Z therapists believe that changing communication pathways and feedback systems is the appropriate method of intervention in a dysfunctional family. Others believe that a family relationship system can be modified by changing the part an individual plays in the relationship system.

Group psychotherapy theory is seldom if ever used within the Z structural orientation. Indications and contraindications for family therapy are not considered to be relevant by Z therapists. The course of therapy is defined more by predetermined theory than by the therapist's subjective feelings. The Z therapists appear to be oriented more toward theory and research than toward therapy.

Major training efforts at these 3 centers are focused on professionals with advanced degrees, primarily in psychiatry and social work. The Philadelphia Child Guidance Clinic and the Georgetown program also place significant emphasis on the training of residents in psychiatry and interns in psychology as well as medical students. The Philadelphia clinic has designed and implemented a unique 40-hour-a-week, two-year family therapy training program for nonprofessional high school graduates who have lived in the area serviced by the clinic.

The primary teachers at Georgetown and at the Philadelphia Child Guidance Clinic, Murray Bowen, M.D., and Salvador Minuchen, M.D., respectively, have extensive psychoanalytic training. However, they deny the value of this theoretical framework for their functioning as family therapists. Minuchen and Bowen clearly identify themselves with the structurally oriented Z therapists, but each has developed different concepts of family theory and structure and different technical interventions and training methods. Their different training methods appear to follow logically from their respective theoretical premises.

Minuchen (3) defines the structure of the family by the patterns of coalitions and splits among family members. These are reflected in the communication pathways that family members follow. A primary objective of Minuchen's structural family therapy orientation is to modify the existing patterns of family coalitions, splits, and communication pathways (4). One of the most characteristic technical features of this orientation, according to Camp (4), "is the deliberate employment of commands, directions, and suggestions as the therapist takes self-conscious responsibility for the direction in which he is pushing the family." This technical approach is specifically designed to modify the feedback system reflected in the communication pathways of the relationship system of the family.

Supervision and training methods at the Philadelphia Child Guidance Clinic are logically designed to make the therapist aware of the feedback mechanisms through which family members organize communication. Therapists in training have feedback mechanisms from their supervisors built into the very architecture of the therapy rooms, which are equipped with one-way mirrors, one-way sound, a telephone line between observation room and interviewing room, and appropriate videotape facilities. This equipment allows constant or periodic monitoring of the therapist-family communication pathways by the supervisor. Immediate consultation can be initiated by either supervisor or therapist; this allows the therapist to readjust his approach to the feedback system within the family.

The Philadelphia Child Guidance Clinic offers family therapy training for individuals who have already been trained in a mental health profession. Periodic workshops focused on specific issues are offered for mental health professionals who are not employed by the clinic. New staff at the clinic are taught by Dr. Minuchen in a two-hour seminar once a week. Additional supervision and observation for up to about five hours a week are provided during family therapy when necessary. Each successive year a junior staff member receives less supervision; after three to four years he is free to work on his own.

Although both Bowen and Minuchen are at the Z or structural end of the scale, their theories, therapeutic interventions, and training methods are markedly different. One of the cornerstones of Bowen's theory is the concept of differentiation of self, which is a measure of the degree of fusion between the intellectual and the emotional systems within an individual. It is also a measure of the degree of unresolved emotional attachment between an individual and his parent. Bowen believes that this emotional and intellectual fusion within the individual and between an individual and his parents manifests itself in the manner in which the individual functions in the relationship system in his nuclear or extended family. He also believes that this pattern of fusion in the family relationship system is so predictable that it can be traced through generations.

According to Bowen, the range of relationship patterns in the system between parents and children is as follows: 1) marital conflict, 2) physical, emotional, or social dysfunction in one spouse, 3) projection of parental problems onto one or more of the children, or 4) a combination of all three patterns. The way these patterns are processed is explained by Bowen's concept of the triangle. His theory assumes that a two-person system is basically unstable and that in an emotional field two people involve a third to make a triangle in an attempt to resolve the tension. This concept describes

the way any three people relate to each other and involve others in the emotional issues among them. The predictable moves in the triangle have been used to develop a system of therapy designed to modify the triangular emotional system.

Therapy within this framework focuses on the most important triangles in the family. It is designed to help one or more family members to become aware of and modify their emotional responsiveness to triangular moves by other family members. Bowen's therapy is basically designed to modify the family relationship system by modifying the part an individual plays in the triangles of the family emotional system. He clearly distinguishes himself from other family theorists by attempting to remain in contact with but outside of the family emotional system.

According to Bowen,

> When one person in the triangle can control self while still remaining in emotional contact with the other two, the emotional tension between the other two subsides. When it is possible to modify the central triangle in a family, the other family triangles are automatically modified without involving other family members in therapy. (5, p.382)

Bowen's therapeutic method is clearly designed to place "the two most important family members [parents] into therapy with the therapist, which makes the therapist the target for family efforts to involve a third person" (5, p.385). Progress in therapy depends on the therapist's ability to maintain meaningful contact with but remain outside of the emotional processes operating among family members. Bowen encourages family members to make contact with as many members as possible of their extended family system to learn more about the emotional processes passed down to them.

Bowen's teaching places strong emphasis on theory and its technical application. Trainees are encouraged to understand their emotional functioning in their own family. Meeting for three hours a week, his postgraduate program has a general portion that stresses theory and technique and a research teaching portion. More advanced therapists meet with Dr. Bowen in other research and teaching conferences held each week. The length of either program is a function of the trainee's progress as a therapist and work in his own family system. The annual fall Georgetown symposium, now in its 11th year, is the major teaching and research event of the year. A two-year family fellowship for third- and fourth-year psychiatry residents began in 1975.

M: Combination of A and Z Orientations

The following institutes fall at the M position on the theoretical scale: the Boston Family Institute in Massachusetts; the Center for Family Learning, New Rochelle, N.Y.; the Bronx Psychiatric Center, Albert Einstein College of Medicine, New York, N.Y.; the Nathan W. Ackerman Family Institute, New York, N.Y.; and the Family Institute of Philadelphia, Pa. The type of family therapy practiced by the faculty at these centers has characteristics of both the A and Z ends of the spectrum. The orientation of individual faculty members at some of the centers falls more clearly at one or the other end of the scale. The overall theoretical orientation of these centers, however, is a mixture of family and individual concepts with a wide variety of techniques. Two factors that appear to influence an individual therapist's place on the scale are his motivation for theory and research and the geographical and professional environment in which he works.

The trainees at all of these institutions are fully trained mental health professionals. The Boston Family Institute also includes teachers, homemakers, and B.A. candidates among their trainees (6), and the Bronx Psychiatric Center includes undergraduates from the various mental health disciplines. The Center for Family Learning appears to be the only institution with an integrated program to make basic knowledge of family process and function available to the nonprofessional community.

The format for training at most of these institutions includes periodic and annual workshops with noted family therapists as well as year-long courses that may be held for two to four years. Supervision is frequently done through live or videotaped observation of the trainees' sessions with families or through discussions in small groups. Literature seminars are offered on the nature of the clinical interview, the study of family process, and in various theories of family therapy. Family therapy techniques used by the faculty at these institutions include psychodrama, Gestalt therapy, behavior modification, structural therapy, family reconstruction, group dynamics, network therapy, modeling, family sculpting, and multiple-family group therapy.

The therapists at some of these centers, especially the senior faculty of the Center for Family Learning, have moved toward the Z end of the theoretical spectrum with its emphasis on studying one's own functioning in one's nuclear and extended family system.

DISCUSSION

There are no central or comprehensive sources of information about family therapy training in the United States. Most training centers and many mental health professionals are unaware of what other centers are doing in their training programs. Although the Group for the Advancement of Psychiatry's report *The Field of Family Therapy* (2) dealt with conceptual approaches, treatment techniques, and the actual practice of family therapy, it did not address itself to the issue of training family therapists. In this study I have attempted to describe, assess, and compare a selected group of training programs.

Creative and innovative theoretical advances are being made at a few of these training centers. Disciples of these innovators are attempting to teach and in-

tegrate these varied concepts at other centers. However, the quality and the design of the training at most of the centers suffer from the absence of an adequate diagnostic nomenclature for family therapy as well as from an insufficiently designed conceptual framework.

Family institutes tend to be centers where multiple theoretical approaches are taught. Smaller clinics often follow the theoretical orientation of one creative individual. Supervision almost always involves live or videotaped observation of family therapy sessions or group discussions; rarely does it involve the more traditional one-to-one type. A few institutions are now beginning to heavily emphasize the idea that trainees need to study and understand their functioning in their own nuclear and extended families as well as develop a system orientation to life situations.

The introduction of family therapy training into psychiatric centers organized along traditional lines in which emphasis is placed on psychoanalysis and individual psychotherapy often creates disequilibrium and dysfunction. It takes the highest order of understanding and skill to achieve the integration of family therapy and training into these systems. This integration is one of the things the mental health profession hopes to accomplish in other kinds of systems that are dysfunctional, whether an individual, a family, a school, a company, or a community. It would be ironic if the mental health profession could not achieve some integration of family therapy with other modalities of treatment to the enrichment of all treatment methods.

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Crime and Violence Among Mental Patients

BY ARTHUR ZITRIN, M.D., ANNE S. HARDESTY, PH.D., EUGENE I. BURDOCK, PH.D., AND ANN K. DROSSMAN

The authors studied the arrest rates of 867 patients from the Bellevue catchment area who were discharged from the psychiatric division of Bellevue Hospital. They found that the arrest rates of these patients for two years preceding and two years following their admissions to the study were higher than the arrest rates for the general population of the Bellevue catchment area as well as those for 4,601 cities in the United States. They discuss the implications of this finding for the postdischarge care of mental patients, particularly in the light of current discharge policies.

THERE HAS BEEN a marked reduction in the number of patients in mental hospitals in recent years (1, 2). In California the mental hospital census has dropped from about 30,000 to 7,000 over the past five years; in New York State the census has declined from 70,000 to 35,000 during the same period. This phenomenon and the enactment in some states of legislation that narrows the criteria for involuntary hospitalization of the mentally ill have focused the attention of psychiatrists and the public on the problems engendered by large numbers of former mental hospital patients in the community (3) and, in particular, on the relation between mental illness and criminal behavior (4).

The literature on crime and mental illness is sparse and contradictory. Studies done in 1922 (5), 1930 (6), and 1938 (6), which were based on New York State hospital statistics, reported lower arrest rates for discharged patients than for the general population. A study done in Connecticut in 1945 (7) agreed with the findings for New York. In the Connecticut study the arrest rate for felonies, calculated over a two-year period, was 4.2 per 1,000 for ex-patients and over 27 per 1,000 for the general population. Comparable results were found for misdemeanors.

An extensive study of a population of 10,247 male patients over the age of 16 who had been released from New York State hospitals between 1946 and 1948 was reported by Brill and Malzberg in 1962 (8). These authors obtained the arrest records for 5,354 of these patients for the whole period before discharge and for the five years after discharge and determined indirectly the arrest rates of the remainder (N=4,893) of their sample. They reported that the overall arrest rate for ex-patients was 122 per 10,000, while that for the general population was 491 per 10,000. However, they also found that the arrest rate for felonies was higher for the ex-patients than for the general population (54.70 per 10,000, compared with 32.83 per 10,000). The higher rate of arrests for felonies among the ex-patients, according to the authors, "was found to be due to the presence among the patients of a disproportionate number with a previous arrest record. Because of the universally high rate of arrest among the latter group, a small excess of their number has a strong influence on the total figure" (8).

In 1965, Rappeport and Lassen (9) reported their study of the arrest records for periods of five years before and five years after hospitalization of all male patients over the age of 16 who had been discharged from all psychiatric hospitals in Maryland during 1947 (N=708) and 1957 (N=2,152). Focusing only on arrests for serious crimes against persons (murder, negligent manslaughter, rape, robbery, and aggravated assault), they found that the arrest rate for robbery was significantly higher for both populations of ex-patients than for the general population. Arrest rates for the other crimes against persons were slightly higher for the ex-patients than for the general population. Schizophrenic patients accounted for more than 30% of the arrests before and after hospitalization in the 1947 group and for about 20% in the 1957 group.

In 1966 Rappeport and Lassen (10) reported that discharged female psychiatric patients had a higher arrest rate for aggravated assault than did women in the general population.

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The authors are with New York University School of Medicine, 550 First Ave., New York, N.Y. 10016, and the Bellevue Hospital Center, New York, N.Y. Dr. Zitrin is Professor of Psychiatry, Dr. Hardesty is Research Associate Professor of Psychiatry (Biometrics), Dr. Burdock is Professor of Psychiatry (Biometrics), and Ms. Drossman is Project Assistant. Dr. Hardesty is also Associate Director and Dr. Burdock is also Director, Biometric Laboratory.

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In 1967 Giovannoni and Gurel (11) reported on a group of 1,142 psychotic patients whom they followed for four years after discharge from 12 Veterans Administration hospitals. These ex-patients had significantly higher arrest rates for serious crimes (assault, homicide, and robbery) than did the general population. Ninety-five percent of these patients were schizophrenic; a majority had abused alcohol.

The frequency of involvement of schizophrenic patients in serious crimes remains in dispute. In a 1974 report on psychiatric outpatients, Guze and associates (12), who relied on the patients' own accounts of felony convictions, concluded that schizophrenic patients were infrequently involved in serious crimes. In a response to that paper, Paull and Malek (13) offered evidence from a psychiatric court clinic that schizophrenic patients frequently commit both misdemeanors and felonies.

In 1973 Häfner and Böker (14) reported a study done in Germany. They concluded that, although "crimes of violence committed by the mentally ill and the retarded were proportional to the number of such crimes charged to the general population," schizophrenic patients were far more likely than other mentally ill patients to be arrested for violent offenses.

METHOD

In this report we present the results of a study of the arrest records of a sample of patients admitted to the psychiatric division of Bellevue Hospital from Bellevue's own catchment area. At the time the sample was selected (July 1969–June 1971) this area consisted of a strip of Manhattan between 14th and 42nd Streets extending from the East River to the Hudson River, with a population estimated to have been approximately 150,000. Only 20% of Bellevue's psychiatric admissions were from this catchment area; most came from other parts of Manhattan.

During the two-year period July 1969–June 1971 a study sample was assembled from consecutive admissions from Bellevue's catchment area for the purpose of determining the efficacy of a community-oriented mental health program. Although there were some 4,800 admissions from the catchment area in the course of this interval, only about 2,000 of these fell into the sample. Some patients had multiple admissions; the number of admissions ranged from 1–22 per patient over the two-year period. Because of these multiple admissions, the number 4,800 corresponds to only 2,140 individual patients.

Our sample comprised all those consecutively admitted patients who could be screened for catchment area membership, who could be interviewed for a baseline measure of psychopathology by the research staff, and for whom a bed was available on one of the three wards designated for the study. However, because of the high frequency of multiple admissions the 2,000 admissions correspond to only 867 individual patients.

The number of admissions and the number of patients cannot be specified exactly because some patients did not or could not give their names and were listed as unknown; others gave names born of delusions. The identities of some of these patients were never determined because they eloped from the hospital before identification could be established. In addition, many patients gave different names on different occasions; some of these were later identified by ward personnel if they recognized them. However, the study sample of 867 was painstakingly investigated. Therefore, it can be asserted that, with one possible exception, the sample represents a group of individual patients. The exception is a case involving multiple admissions under different names: at one time the admissions were thought to be of 1 person, but later they were considered to represent 2 persons; only 1 person could be found on follow-up, however.

In addition to the sample of 867 patients, we also had data on 977 patients from the catchment area who were screened in the psychiatric admitting office but who did not become inpatients. Some of these individuals came to the admitting office more than once during the study period. As can be seen in table 1, the nonadmitted patients were very similar to the admitted group in sex and age. There were more men than women in both groups of patients, in contrast to the catchment area population, which had more women (see table 1). The age distributions of the two groups of patients are quite similar, with the highest proportion of patients in the 20–50-year-old range. In contrast, the catchment area shows a relatively uniform distribution of ages from 20 to 70.

Among the data subsequently collected were the arrest records for each patient in the inpatient sample (N=867) for the two years before admission to the study and the two years following that admission. The

TABLE 1
Comparison of 867 Patients Admitted to Bellevue, 977 Patients Treated and Released, and General Catchment Area Population (N=130,397), by Sex and Age

Item	Catchment Area Population		Patients Treated and Released		Patients Admitted	
	Number	Percent	Number	Percent	Number	Percent
Sex						
Male	59,686	46.0	644	66.0	496	57.2
Female	70,711	54.0	333	34.0	371	42.8
Age (years)						
0–9	8,999	6.9	0	—	0	—
10–19	10,630	8.2	62	6.3	42	4.8
20–29	26,004	19.9	291	29.8	273	31.5
30–39	18,301	14.0	240	24.6	228	26.3
40–49	17,619	13.5	203	20.8	175	20.2
50–59	18,850	14.4	83	8.5	89	10.3
60–69	17,694	13.6	55	5.6	43	4.9
70–79	9,494	7.3	17	1.7	12	1.4
80 or more	2,806	2.2	4	0.4	1	0.1
Unknown	0	—	22	2.3	4	0.5

types of offenses for which patients in the sample had been arrested have been divided into violent offenses and nonviolent offenses. Violent offenses have been further subdivided into those involving direct bodily harm and those involving the potential for harm.

Under the category of violent offenses involving bodily harm were murder, assault, rape, and sexual abuse. Violent offenses with the potential for harm included robbery, burglary, possession of weapons, possession of burglar's tools, arson, and inciting to riot. Nonviolent offenses (exclusive of traffic violations) included, in order of their frequency, public intoxication, possession or sale of drugs or of a hypodermic instrument, disorderly conduct, loitering, resisting arrest, possession of stolen property, harassment, larceny, menacing behavior, and others.

RESULTS

Of the 867 mental patients in our sample, 202 (23.3%) had been arrested at least once during the two years before and the two years after their admission to the study; 117 of these 202 patients (13.5%) had been charged with a nonviolent offense only, and 85 patients (9.8%) had been charged with a violent offense.

Figure 1 shows the age distributions of the patients who had been arrested for at least one violent offense during the study period, those arrested for a nonviolent offense only, and the total sample of 867 patients. The age distribution of the total sample shows a wider dispersion than the age distribution of either of the two groups of patients who had been arrested. This reflects the fact that most arrests of either type were of patients in the 20–40 age range. The age distribution of the group arrested for a violent offense peaks in the 20–30 age interval, whereas more of the patients arrested for a nonviolent offense were in the 30–40 age interval. Moreover, the curve for the group arrested for a violent offense does not extend beyond the 50–60 age interval.

Table 2 shows the distribution of diagnoses among the total admission sample, patients who had been arrested for a violent offense, and those arrested for a nonviolent offense. The patients were classified according to the primary diagnosis recorded at the time of discharge from the hospital. In the sample as a whole, as well as in the group arrested for a violent offense, almost half of the patients were schizophrenic. On the other hand, the proportions of alcoholic and drug-dependent patients who had been arrested for a violent or a nonviolent offense were twice as large as the corresponding proportions in the total sample.

Table 3 shows the diagnoses and arrest records of the 30 patients who had been charged with at least one violent offense involving bodily harm as well as those of the 55 patients who had been charged with at least one violent offense involving the potential for harm. Twenty-one of the patients in each group were schizophrenic. The schizophrenic patients arrested for at least one violent offense involving bodily harm had a mean number of arrests for all offenses greater than that of any of the other diagnostic groups, but the mean number of arrests for all offenses of schizophrenic patients for whom the most serious charge involved potential for harm was lower than that of all but one of the other diagnostic groups.

Table 4 details the offenses charged to the 85 patients who had been arrested for at least one violent offense of either subtype according to the patients' diagnoses. Approximately half of these 85 patients were schizophrenic. Schizophrenic patients were charged with 51% of the violent offenses and 52% of all offenses, compared with 11% and 9%, respectively, for alcoholic patients, 15% and 21% for drug-dependent patients, and 23% and 18% for patients with other diagnoses.

However, only 10% of the schizophrenic patients in the total sample of 867 patients were arrested for violent offenses, compared with 16% of alcoholic

FIGURE 1

Age Distributions of Total Admission Sample (N=867), Patients Arrested for a Violent Offense (N=85), and Patients Arrested for a Nonviolent Offense (N=117)

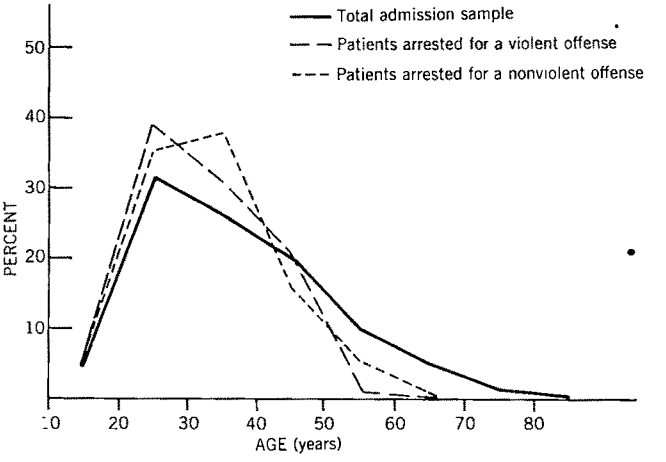


TABLE 2

Comparison of Total Admission Sample (N=867), Patients Arrested for a Violent Offense (N=85), and Patients Arrested for a Nonviolent Offense (N=117), by Diagnosis

Diagnosis	Total Admission Sample		Patients Arrested for a Violent Offense		Patients Arrested for a Nonviolent Offense	
	Number	Percent	Number	Percent	Number	Percent
Schizophrenia	403	47	42	49	44	38
Alcoholism	64	7	10	12	23	20
Drug dependence	51	6	15	18	17	14
Neurosis	71	8	6	7	11	9
Other	278	32	12	14	22	19

TABLE 3
Arrest Records and Diagnoses of 30 Patients Arrested for a Violent Offense Involving Bodily Harm and 55 Patients Arrested for a Violent Offense with Potential for Harm

Item	Diagnoses					
	Schizophrenia	Drug Dependence	Alcoholism	Neurosis	Other	Total
Patients arrested for a violent offense involving bodily harm						
Number of patients	21	2	2	3	2	30
Number of arrests for violent offense involving bodily harm	30	2	3	3	4	42
Number of arrests for violent offense with potential for harm	14	1	0	1	1	17
Number of arrests for nonviolent offense	84	3	1	4	2	94
Total number of arrests for all offenses	128	6	4	8	7	153
Mean number of arrests for all offenses	6.1	3.0	2.0	2.7	3.5	5.1
Patients arrested for a violent offense with potential for harm						
Number of patients	21	13	8	3	10	55
Number of arrests for violent offense with potential for harm	22	18	10	3	19	72
Number of arrests for nonviolent offense	36	55	21	4	25	141
Total number of arrests for all offenses	58	73	31	7	44	213
Mean number of arrests for all offenses	2.8	5.6	3.9	2.3	4.4	3.9

TABLE 4
Offenses Charged Against 85 Patients Arrested for a Violent Offense, by Diagnosis

Item	Number of Arrests, by Diagnosis				
	Schizophrenia (N=42)	Alcoholism (N=10)	Drug Dependence (N=15)	Other (N=18)	Total (N=85)
Violent offenses					
Murder	1	0	0	0	1
Aggravated or simple assault	24	3	2	6	35
Rape	3	0	0	1	4
Sexual abuse	2	0	0	0	2
Robbery	15	5	3	6	29
Burglary	10	4	8	10	32
Possession of weapons	17	5	5	3	30
Possession of burglar's tools	6	0	5	7	18
Arson	0	0	0	3	3
Inciting to riot	1	0	0	0	1
Total	79	17	23	36	155
Nonviolent offenses					
Public intoxication	52	5	0	5	62
Possession or sale of drugs or of hypodermic instrument	16	3	31	11	61
Disorderly conduct	38	4	4	6	52
Loitering	17	6	18	11	52
Resisting arrest	16	4	4	4	28
Possession of stolen property	9	0	9	7	25
Harassment	16	1	3	2	22
Larceny	13	1	1	6	21
Menacing behavior	8	3	1	1	13
Other	26	5	21	12	64
Total	211	32	92	65	400
Total	290	49	115	101	555

patients, 30% of drug-dependent patients, and 5% of patients with other diagnoses.

Of the 42 schizophrenic patients among the 85 arrested for a violent offense, 20 had a history of alcohol abuse, 8 were at some time drug dependent and alcoholic, and 2 others had also been diagnosed as drug ad-

dicted. Of the 15 patients with a primary diagnosis of drug dependence, 2 had also been diagnosed as schizophrenic and 7 were abusers of alcohol. Of the 10 alcoholic patients in the group arrested for a violent offense, 4 had been diagnosed as schizophrenic on at least one occasion and 1 was drug dependent.

Table 5 shows when the arrest for a violent offense occurred with reference to the time of admission into the study of the 85 patients. There are three groups represented here: those who had been arrested during the two-year period preceding admission into the study but not during the two-year follow-up period, those who had been arrested during the two years following admission but not during the two years preceding admission, and those who had been arrested during both of these periods. Arrests for violent offenses involving bodily harm occurred 1½ times more frequently during the two-year period after admission, but arrests for violent offenses with potential for harm and arrests for nonviolent offenses occurred at about the same frequency before and after admission.

In table 6 the arrest rates per year for major crimes are shown for our sample of 867 patients for the two years before and the two years after their admissions in 1969–1972, for the population of the Bellevue catchment area as a whole in 1972, and for 4,601 cities in the United States with populations over 2,500 in 1972.

In the preparation of this table, the population data for the 4,601 cities were reduced by the proportion of children under age 15 (calculated from table 6 of *Statistical Abstracts of the United States:1971* [15]), and the rates were recalculated from table 27 of *Crime in the United States, 1972* (16, p. 120), with the arrests of children under age 15 omitted. The population figures for the three police precincts comprising the Bellevue catchment area were taken from a study prepared by the Rand Institute and the *New York Times* and reported in the *Times* of July 30, 1973 (17). This population was also reduced by the proportion of children under age 15 in the catchment area, as reported in the tables of the New York City Department of City Planning.

Since each patient's arrest record covers a four-year period, the total number of arrests for each violent offense for the sample was divided by 4 to obtain an annual rate. This method yielded a conservative estimate. Had the arrest rates for the sample been calculated for the single year in which the maximum number of patients was under study, the rates would have been higher. Other factors contributing to the likelihood that the rates given in table 6 are minimal include the following: 1) the death rate in the sample was higher than that in the catchment area, 2) some patients in the sample may not have been in the city during the two years before their admission to the study or may have left the city following discharge from the hospital, so that any arrests they incurred in other cities would not have been counted, 3) periods of hospitalization precluded arrests, 4) a portion of the arrest records for the year 1973 (the terminal year of follow-up) was not available from the New York City Police Department at the time of this writing.

1970–1972 was the period during which the major portion of our sample was under study. 1972 was chosen for the comparison of other urban populations and the Bellevue catchment area population because it was the year with the highest frequency of arrests for crimes of violence in the United States during 1970–1972 (16, p.4). Nevertheless, the frequency of arrests for each of the major crimes listed in table 6 was con-

TABLE 5
Time of Arrest in Relation to Admission to Study of 85 Patients Arrested for a Violent Offense, by Type of Offense

Item	Time of Arrest				
			During Both Periods		
	During Two Years Before Admission	During Two Years After Admission	Before Admission	After Admission	Total
Arrests for violent offense involving bodily harm					
Number of patients	9	10	—	—	11
Number of arrests	11	11	5	15	20
Arrests for violent offense with potential for harm*					
Number of patients	20	14	—	—	30
Number of arrests	22	16	20	31	51
Arrests for nonviolent offense**					
Number of patients	11	10	—	—	33
Number of arrests	26	18	100	91	191
Arrests for all offenses					
Number of patients	28	21	—	—	36
Number of arrests	59	45	—	—	262

* Includes some patients from the category of violent offense with bodily harm.
** Includes some patients from the categories of violent offense with bodily harm and violent offense with potential for harm.

TABLE 6
Age-Adjusted Arrest Rates per 1,000 Persons for 4,601 U.S. Cities in 1972, the Bellevue Catchment Area in 1972, and the Bellevue Admission Sample of 1969–1971 (N=867), by Offense Charged

Offense Charged	U.S. Cities	Bellevue Catchment Area	Admission Sample
Murder	0.15	0.45	0.30
Rape	0.17	0.57	1.15
Robbery	1.06	8.81	8.38
Aggravated assault	1.44	5.81	7.02
Burglary	2.31	5.33	8.67

TABLE 7
Proportions of Men and Women Arrested for Major Violent Offenses in the United States in 1972 and in the Bellevue Admission Sample of 1969–1971 (N=867)

Offense Charged	Proportions in United States		Proportions in Admission Sample	
	Percent of Men	Percent of Women	Percent of Men	Percent of Women
Murder	84	16	0	100
Aggravated assault	86	14	91	9
Rape	100	0	100	0
Robbery	93	7	83	17
Burglary	95	5	97	3

siderably higher among our sample and in the Bellevue catchment area than in urban areas of the United States as a whole. Moreover, except for murder[1] and robbery, the rates of arrest for serious crimes for the sample of mental patients were even higher than the rates for their community.

Table 7 shows a comparison of the proportions of men versus women in our sample of 85 patients who were arrested for a major violent offense and shows a similar comparison for the United States as a whole. The data for the United States are from table 35 of *Crime in the United States, 1972* (16, p. 130). The largest difference occurs for the crime of robbery, for which our mental patients showed a higher proportion of women than did the United States as a whole. The proportions for murder in the sample should be interpreted with caution because they are based on a single case.

DISCUSSION

The commonly held belief that the mentally ill commit fewer crimes than the general population is not supported by our study. Indeed, the arrest records of these 867 psychiatric patients from the Bellevue Hospital catchment area show that their rates of arrest for criminal behavior, including violent offenses, are higher than the corresponding rates in their community. Our study was based on admissions to an acute psychiatric service of patients from Bellevue Hospital's catchment area. That the conclusions are not in agreement with earlier reports based on state hospital patients (5–8) may be due in part to differences in methodology. Our results are in general agreement with the reports of Rappeport and Lassen (9) and Giovannoni and Gurel (11).

The presumption that the mentally ill commit fewer crimes than the general population is one factor that has been invoked in support of the current discharge policies of mental hospitals. Because there is a lack of accurate predictive indicators of violent behavior in mental patients, we do not believe that our findings, even if confirmed, will effect any major change in these policies. The prediction of dangerousness, the single most common criterion for involuntary psychiatric hospitalization, is generally conceded to be highly unreliable (18), except perhaps in samples of selected persons at particularly high risk of injury to themselves or others. We are now examining our data on the patients in our study to determine whether we can identify any factors that distinguish patients who were arrested for crimes from those who were not. However, the findings presented here have implications for the postdischarge care of mental patients, particularly if current discharge policies are continued.

The provision of suitable community facilities for treatment, supervision, or follow-up has not kept pace with the needs generated by the discharge of large numbers of mental hospital patients and by more restrictive admission policies. On the other hand, a consequence of the voluntary status of most mental hospital patients today is that they are not required to attend the clinics that are available. It is likely that a system of conditional discharge of some classes of patients with required attendance at outpatient clinics would make possible timely interventions that might prevent criminal behavior or abort full relapses. There is a need to identify those patients for whom such a plan would be useful. To oppose mandatory posthospital care for patients who are known to be at increased risk of dangerous behavior if not supervised is misguided civil libertarianism. Patients who abuse alcohol or other drugs, regardless of the nature of their concomitant or associated mental illness, appear from our data as well as from the work of others (11) to need such care.

The increasing diversion of arrested persons from the criminal justice system to mental hospitals is another factor that might be responsible for the apparent rise in criminal behavior in mental patients. The arrest rate among former hospital patients increases when those who were placed in mental hospitals after an arrest are discharged. Records of convictions of mental patients are less accurate as an index of criminal behavior than records of arrests because many offenders are found in-

[1]Three arrests for murder in our sample have not been counted because they occurred outside of the four-year period of the study.

competent to stand trial or are taken out of the court system before that stage of the legal process takes place. Conviction records would therefore give misleadingly low figures on the criminal activity of the mentally ill.

The treatment of the mentally ill offender has long been a vexing problem. Correctional institutions with psychiatric services have been notoriously ineffective. In New York State all but convicted and sentenced mentally ill offenders are now treated in civil state hospitals. The transfer of mentally ill prisoners from correctional hospitals to civil state hospitals after completion of their sentences, a result of the landmark *Baxstrom* decision (19), has been hailed as a highly successful development in the treatment of this group of patients (20).

However, some recent evaluations of the adjustment of these patients in civil hospitals require a qualification of that judgment. For example, one investigation determined that patients transferred from the correctional hospital at Matteawan were found to have "economically, physically or sexually abused other patients" in the civil hospitals (21). Another study found that some mentally ill offenders in civil hospitals formed a "rapacious subculture" that terrorized other patients and exploited the "staff's tendency to 'treat' people rather than 'punish' them" (22). It seems clear that, although a serious inequity was corrected by removing prisoners who had completed their sentences from correctional institutions, the change in the place of their detention has had multiple consequences, not all of them beneficial.

Although our focus has been on criminal and violent behavior shown by mental patients in the community, these patients are often the victims of crime and exploitation or suffer in other ways from inadequate attention. In pursuit of the laudable goal of removing patients from institutions and placing them in the community, the discharge of the mental patient from the hospital has become for some the standard of successful psychiatric therapy and for others the triumph of individual liberty over social control. However, many of these patients are discharged to fend for themselves—to find lodging in the cheap single-room-occupancy hotels and rooming houses in decaying sections of our cities (23). Few halfway houses are available for discharged patients; moreover, residents of middle-class communities, alleging lack of supervision of the patients, often resist the opening of such facilities in their neighborhoods (24). Instances of kidnapping, imprisonment, and mistreatment of former mental patients by rooming house owners have recently been documented in a New York State court (25).

The special vulnerability of mental patients can be measured in other ways as well. In the follow-up of the 867 patients in our sample we found a death rate more than double the expected rate in the population of the Bellevue catchment area across age categories (24.9 deaths per 1,000 for our patients, compared with 11.2 deaths per 1,000 in the catchment area). The suicide rate was 5.1 per 1,000 for our sample, compared with .08 per 1,000 for New York City as a whole. There were 2 victims of murder among our patients.[2] This would correspond to a rate of 1.15 per 1,000, compared with a rate of .20 per 1,000 for the city as a whole.

COMMENT

This study has not provided answers to the difficult questions of why behaviors that lead to arrest occur among discharged mental patients or how to predict and prevent them. However, the discharge of patients from mental hospitals without provision for decent housing and humane follow-up care for those who require it is regressive. It is not the great social and psychiatric advance that it has been claimed to be.

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Change in 1976 Annual Meeting Site

The APA Executive Committee decided to move the May 10-14, 1976 APA Annual Meeting from Atlantic City to Miami, Florida, after hearing the APA Meetings Management Department report that it had ascertained that a major Atlantic City hotel had inadvertently committed several hundred rooms to another organization during the week the APA meeting was scheduled. This would have left APA with the unacceptable alternative of using a myriad of scattered small hotels in the Atlantic City area. It has been confirmed that Miami Beach can accommodate the meeting.

Further arrangements and developments will be reported in future issues of *Psychiatric News*.

Cluster Analysis Profiles of Suicide Attempters

BY ARI KIEV, M.D.

The author reports the results of a three-stage cluster analysis of data on 298 patients who attempted suicide. Patients were allocated by the analysis to seven identifiable profile types that correlated with six outcome variables. These profiles include data on the social setting and the seriousness of the attempt and judgments about overall functioning and interpersonal conflict as well as the input of the patients' significant others. The author states that these profiles have prognostic significance as well as implications for the treatment of suicide attempters and the education of emotionally involved significant others.

THE DIFFICULTIES INHERENT in developing valid criteria for estimating the prognosis of suicidal crises stem from the fact that the psychological and social factors leading to the suicide attempt have often passed or subsided by the time evaluation occurs. The psychiatrist must deal with probabilities on the basis of factors that cannot be observed but can only be inferred by the patient's history and the accounts of emotionally involved significant others.

Factor analysis of the 56-item Life Crisis Inventory, which was developed for a one-year follow-up study of 300 patients who attempted suicide (1), pointed to the prognostic importance of three factors: interpersonal conflict, the social setting of the attempt, and the attitudes of significant others.

This paper examines the results of a hierarchical clustering technique designed to condense nine Life Crisis Inventory factor scores and four variables regarding significant others for each patient in a sample of 298 suicide attempters (the number of the original cohort of 300 for whom sufficient data were available) into clinically relevant profile or prognostic types.

METHOD

The analysis involved three stages: pattern extraction, or R analysis (2), to compress the patient-variable dimensions; Ward's hierarchical grouping technique (3, 4) to define the symptom types of attempters; and a "scrubbing" procedure (5) to test the fitness of each person to this cluster, to reassign misfits and overlaps, and to eliminate isolates.

RESULTS

Of the total sample examined (N=298), 245 patients (82%) could be allocated to seven clearly identifiable types. Misfits were reassigned to the cluster they most suitably fit, and isolates and overlaps were identified and omitted from the remaining analysis. Outcome profiles for each of these cluster types on six outcome variables were then established by means of a discriminant analysis. The resulting function discriminating the cluster types shares 41.3% of its variance with the six outcome variables. (The computer program used to calculate the function, DISCRIM, also prints the canonical correlation [.541, p=.006].)

The following descriptions characterized the seven profile types.

Type I: Suicidal Gesture

Type I, the largest clustering, consisted of patients with characterological problems who experienced few symptoms of anxiety, guilt, or depression; minimal dysfunctioning and minimal interpersonal conflict; and alcohol and/or drug abuse. Denying the presence of psychiatric difficulties or attributing their problems to situational factors, patients who fit this profile type made the least severe attempts of all seven groups. They reported no plans for the attempt, minimal suicidal intent, no real desire to die, minimal suicidal thoughts, and minimal discomfort before the suicide attempt. They attempted suicide with pills or by slashing their wrists in the presence of others or with others nearby, although no significant others were available for interview at the time of admission. Despite repeated low-risk attempts, these patients tended to have good general outcomes; they showed little increase in conflict or in symptoms at one- and two-year follow-up.

Type II: Acute Depressive Reaction

Type II, the second largest clustering, consisted of acutely depressed patients who believed themselves to be incapable of coping with objective life stresses and attempted suicide with irreversible means in a setting

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Dr. Kiev is Director, Social Psychiatry Research Institute, and Clinical Associate Professor of Psychiatry (Social Psychiatry), Cornell University Medical College, New York, N.Y. Address reprint requests to him at 150 East 69th St., New York, N.Y. 10021.

that held minimal possibilities of rescue.

These patients reported the highest scores on the somatic symptoms of depression, including appetite, sleep, and sex disturbances, as well as high scores on anxiety, tension, fear, and remorse. They reported minimal interpersonal conflict with significant others, who tended to be supportive.

The social setting of the suicide attempts of this group of patients—at a distance from others and with minimal possibility of rescue—despite the supportive attitudes of significant others, earmarked this high-risk group. In the year following the attempt, these patients showed the highest scores on increased conflict, the second highest scores on subsequent hospitalization, relatively high scores on increased symptoms, and some subsequent attempts.

Hospitalization should be seriously considered for this group of patients. They require prompt reduction of depressive symptoms with ECT or psychopharmaceuticals. Special precautions should be taken when a patient who fits this type appears to improve but has not yet come out of his depression.

Type III: Passive-Aggressive and Passive-Dependent Personality Disorder

Type III patients reported a minimum of symptoms and interpersonal conflict. Judged by significant others to be quite dysfunctional, these patients had been hospitalized in the past more often than any of the other groups. They attempted suicide in a remote and private way, using irreversible means and making no effort to obtain help. In the year following the attempt, they made the fewest major changes in their lives and were judged generally to have the worst clinical outcomes, despite the absence of repeated attempts. They scored none or minimal on ratings of suicidal preoccupation and obsessive thoughts or acts. They showed little evidence of emotional or physical blunting of affect and received the lowest scores on unusual thoughts and behavior of any of the groups. They also had mild and minimal ratings on gradiosity, hallucinatory behavior, and bizarre thought content. They were rated as minimal to mild on anxiety, guilt, tension, and depressive symptoms.

A history of a serious suicide attempt in an isolated social setting without the seeking of help and a discrepancy between these patients' views of their problems (they minimized them) and the views of significant others (who may have been indifferent, rejecting, punitive, or overcontrolling in their concern for the patients' welfare) made this a high-risk group.

Treatment goals for patients who fit this type must be designed to reduce their high rate of dropping out of treatment. Brief hospitalization should be considered to reduce the pressures brought by significant others and to arrange less noxious living arrangements for these patients. Long-range supportive programs may prove to be the most satisfactory way of keeping contact with them. At the same time, significant others should be helped to reduce their excessive guilt and sense of responsibility.

Type IV: Anxiety Reaction with Interpersonal Conflict

Of the seven groups, type IV patients were judged to have the highest scores on anxiety and guilt and the second highest scores on tension, suicidal threats, and interpersonal conflict, despite reports of significant others that they were supportive of the patients and nonthreatened. While patients who fit type IV attempted suicide in close proximity to others with reversible methods—e.g., lacerations and pills—they were judged to be above average in suicide potential.

Type IV patients received average scores for unusual thoughts and emotional and physical blunting and the lowest scores on depressive symptoms and overall dysfunctioning. They seemed to be making good adjustments.

The most prominent feature of this group appeared to be the marked discrepancy between the patients' high scores for interpersonal conflict and the significant others' accounts of being supportive, which no doubt contributed to the persistence of high levels of interpersonal conflict, the highest incidence of repeated attempts, and the second highest scores on symptoms in the year following the attempt. These patients reported considerable interpersonal conflict with significant others, who viewed themselves as supportive and minimized the patient's distress. The discrepancy between the patient's subjective sense of rejection and the reports of the significant others' indifference led to repeated attempts, a persistence of symptoms, and continued interpersonal conflict.

Treatment for this group of suicide attempters must ultimately focus on the resolution of interpersonal conflict and reduction of related anxiety and tension. Outpatient treatment and family therapy may be most relevant because hospitalization is unlikely to resolve the problem.

Type V: Socially Isolated

Type V patients, who were characterized by very high scores on interpersonal conflict, suicide potential, and unusual thoughts and behavior, attempted suicide at a distance from others and made no effort to be rescued. Despite this, significant others judged their functioning to be unremarkable, a finding with substantial prognostic implications. Of interest and possibly related to their social isolation, type V patients (like type VII patients, described below) could not be personally evaluated at the time of the one-year follow-up.

The inclination of this group of patients to drop out of treatment should be recognized. Efforts should be made to establish a long-range supportive and contact program for these patients, with simultaneous efforts directed toward the education of significant others, who must be helped to understand the seriousness of the problems of these patients.

Type VI: Suicidal Preoccupation

Type VI patients constituted a group who were diagnostically similar to type I patients except for high scores on unusual thoughts and behavior and the highest score of any group on suicide potential. These patients reported more obsessive thoughts and interpersonal conflict than type I patients, although their scores were close to the mean. They experienced little anxiety, guilt, or depressive symptoms and only minimal dysfunctioning. They were close to the mean on emotional withdrawal and physical blunting, and they attempted suicide in close proximity to others.

At one year, type VI patients had made the most major changes. They had high scores on increased symptoms but had made no subsequent suicide attempts.

In essence, type VI patients presented more serious characterological problems than type I patients as well as the highest scores on the factor measuring suicide potential. They made relatively nonserious attempts in close proximity to others in the face of situational problems but appeared to have good one-year outcomes, probably related to the major changes they were able to make in their lives.

Type VII: Chronic Dysfunctional

Type VII patients resembled type III patients in that they had low scores on anxiety and guilt and attempted suicide at a distance from help, using irreversible means. They also had high scores on interpersonal conflict, suicide potential, and significant others' ratings of overall dysfunctioning and rejection.

They differed from type III patients in that they had higher scores on obsessive thoughts, suicidal preoccupation, and overall dysfunctioning, the highest scores on emotional and physical blunting, and some evidence of depressive symptoms.

DISCUSSION

Threatened or attempted suicide cannot be lightly dismissed as attention-seeking behavior. People who attempt suicide often come close to death. Those who threaten suicide or who make less serious attempts may kill themselves in subsequent attempts.

To the extent that suicide does not occur unpredictably it can be prevented if proper attention is paid to long-range prognosis and degree of risk.

The results described here of a cluster analysis underscore the importance of evaluating the effects of discrete variables that may act simultaneously and perhaps synergistically to increase suicidal risk. Particular emphasis must be given to the views of significant others regarding the patient's symptoms and need for treatment, the presence of interpersonal conflict, and the discrepancy between the attitudes of the patient and of significant others.

The groups at highest risk of suicide appear to be patients suffering from psychoneurotic illnesses with depressive or anxiety symptoms who are involved in interpersonal conflict and patients with longstanding characterological problems associated with nonfunctioning and considerable interpersonal conflict. Socially isolated patients, who tend to disappear from follow-up, represent an additional high-risk group.

The attitudes of significant others toward the patient, his illness, and his suicide attempt have major significance in assessing prognosis. Patients with the highest scores on depressive symptoms and serious suicide attempts reported considerable conflict with significant others at the end of one year after their suicide attempt, suggesting that increased interpersonal conflict subsequent to an attempt may be reflected in rehospitalization, increased symptoms, and subsequent attempts.

In contrast to depressed patients, those with substantial anxiety reactions reported considerable interpersonal conflict, which contrasted markedly with the accounts of significant others, who considered themselves to be supportive and nonthreatened by the attempt. The discrepancy between the views of patients and those of significant others may have been reflected in the high incidence of repeated attempts and the increase in symptoms in this group of patients.

The significant others in the passive-dependent, passive-aggressive group conveyed indifferent and rejecting attitudes, which were at considerable variance with the average scores on interpersonal conflict reported by the patients, whose isolated attempts were above average in intention and lethality.

The significant others of the socially isolated patients also reported indifferent and rejecting attitudes, but this corresponded to the perceptions of the patients, who had the highest scores on interpersonal conflict and who attempted suicide in isolation.

Psychiatric illness may too often be mistakenly rationalized as attributable to factors other than mental disease. Failure to recognize the patient's need for treatment may lead to an escalation of conflict and stigmatization of the patient unless appropriate efforts are made to educate both patients and their significant others as to the nature of the problem.

As has been shown in previous reports (1, 6, 7), the attitudes of significant others appear to relate to the outcome of suicidal patients—whether in terms of their ignoring the presence of a treatable illness or in terms of their creating a nonsupportive, punitive, or noxious environment for the patient. These factors have relevance for assessing the immediate risk following a suicide attempt and for the continuing management of the patient. If symptoms improve but negative attitudes persist, prognosis must necessarily be guarded.

COMMENT

The clinical descriptions of the seven profile types given here illustrate the relevance of considering the

simultaneous interaction of these variables in determining prognosis for suicide attempters. These profiles held up over the period of follow-up. They demonstrate an empirical consistency and validity that gives them prognostic significance. The high correlation between initial and outcome variables further supports the validity of these profiles, which have been theoretically and factor-analytically determined.

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APA Grant Program for Minority-Group Medical Students and Residents

A small number of qualified minority applicants (including but not limited to American Indians, Asian Americans, black Americans, and Spanish-speaking Americans) will receive residency stipends, assistance in selecting a residency program, expenses to a yearly meeting with other minority fellows to share experiences and administrative help through APA's Office of Minority Affairs. The program is sponsored by APA, with funds provided by the National Institute of Mental Health.

Applications are being accepted now for residencies beginning in July 1976 and subsequent years. Applicants must be U.S. citizens and must have graduated from a U.S. medical school or possess the ECFMG certificate.

For further information write Jeanne Spurlock, M.D., Office of Minority Affairs, American Psychiatric Association, 1700 18th Street, N.W., Washington, D.C. 20009, or call (202) 232-7878.

The Behavior Therapies: Therapeutic Breakthrough or Latest Fad?

BY ARTHUR K. SHAPIRO, M.D.

The author critically examines the evidence for the effectiveness of the behavior therapies as described in the American Psychiatric Association's task force report on behavior therapy. He concludes that current attempts to evaluate behavior therapy suffer from inadequate methodology and that the claims for its therapeutic efficacy are excessive and unsupported by the controlled clinical evidence.

THE BEHAVIOR THERAPIES, increasingly used in the treatment of emotional disorders, have influenced treatment by psychiatrists and are one of the major therapies used by psychologists. If the trend continues, behavior therapy[1] may become the predominant therapeutic modality taught to and practiced by psychologists, replacing insight-oriented, client-centered, rational, and other types of psychotherapy. This trend is evident in the many training programs that have been established throughout the country (1) and in the exponential increase in the publication activity in the field of behavior therapy, which now yields more publications than any other therapy (2). Further serious consideration of behavior therapy was stimulated by the favorable report of the American Psychiatric Association's Task Force on Behavior Therapy (3).

This new development in psychotherapeutics raises important scientific and pragmatic issues and questions. If the theory is correct, and if it can be substantiated by appropriate clinical evidence, two important alternatives must be considered. Either psychiatrists will have to spend considerable time and energy in mastering the theory and practice of behavior therapy, or this therapy will increasingly be conducted by psychologists.

The question of whether behavior therapy is a therapeutic breakthrough or a recent fad is therefore an important scientific and pragmatic one. Because the APA task force report is an important summary document that has had and will continue to have an important influence on psychiatry, this paper will focus on and be limited to the advantages of behavior therapy as described in nine points in this report. The nine points will be discussed serially as they appear in the report.

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Dr. Shapiro is Professor of Clinical Psychiatry and Director, Special Studies Laboratory, Payne Whitney Clinic—Cornell University Medical College, 1300 York Ave., New York, N.Y. 10021.

[1]The term "behavior therapy" is used in this paper in the broad sense of the heterogeneous group of behavior therapies.

ADVANTAGES OF BEHAVIOR THERAPY

Behavior therapy developed largely from experimental psychology in the laboratory and is distinguished from alternative clinical approaches because it is based on experimental behavioral science. Behavior therapists claim superiority for their technique because their therapy is based on theory derived from the laboratory.

But let us not forget the harmful effects of overelaborate theoretical formulations in medical history (4). Medical history is in part the succession of overdeveloped theory and practice, corrected by a simple empiricism that, in turn, becomes overelaborated (5). In the past erroneous theory imposed on treatment often led to clerics, empirics, apothecaries, and charlatans being more correct than physicians (6). Although theory derived from experiments in the laboratory may be an important lead for clinical practice, it may be clinically irrelevant. For example, placebo effects occur less frequently in laboratory experiments than in clinical situations (7). In addition, quantifiable and reliable laboratory tests of suggestibility do not correlate with placebo effects in clinical situations or with subsequent clinical course, whereas placebo effects do tend to correlate with clinical course (8–10). An established principle in pharmacology is that the data derived from the laboratory (e.g., in vitro, specific species, or animal studies) cannot be safely generalized to man. The hypotheses derived from such data have to be repeated in man.

Reppucci and Saunders (11) came to a similar conclusion. They stated, "Although claims of the success of applied behavior modification as a vehicle of institutional and individual change are widespread, convincing empirical evidence is limited to laboratory and highly financed, small demonstration projects. Thus, the usefulness of current behavior modification techniques in natural settings is unclear." (p. 658)

Behavior therapy methodology requires and leads to the clinical behavior therapist objectively analyzing,

describing, and quantifying the clinical problem, treatment, and evaluation.

This claim is made repeatedly by behavior therapists. However, all clinicians make this claim, and behavior therapists are merely repeating it, using research instead of clinical jargon (12–14).

Most of the reported case histories and behavioral analyses are very complicated, primarily unverified suppositions, and merely stated to be objective by the behavioral analyst. Supporting data such as studies of reliability, validity, and the psychometric properties of the concepts and their measurement are generally absent from the behavior therapy literature, and when present are limited to behavioral observations and target symptoms. I suspect that the reliability and validity would be as low for behavioral analyses as they are for psychiatric diagnosis and psychodynamic formulations.

Behavior therapy is applicable to both psychological and organic illnesses.

This concept is untrue, adherence to it may be harmful and occasionally lethal, and it is often pragmatically irrelevant. At a minimum it should be used only after careful consideration of other alternatives. Many organic illnesses are influenced by stress and psychological factors, but no one today would treat syphilis except with penicillin, pellagra except with nicotinic acid, diabetes with anything but insulin, and so on. However, behaviorists mistreat and harm patients with Gilles de la Tourette's syndrome when they use behavior therapy instead of haloperidol (15, 16) and when they interpret the symptoms as voluntary, manipulative expressions of anger (17–20). It is questionable whether teaching children to behave or improved management of mentally retarded children should be classified as a psychiatric, medical, or psychotherapeutic treatment.

Patients with chronic disabling problems who have failed to respond to other forms of psychotherapy have shown well-documented and substantial improvement with behavioral procedures.

It is curious that this claim is made by all psychotherapeutic schools, as well as by charlatans whose patients have failed to respond to other therapies. It should not be forgotten that despite the tendency to glamorize and sentimentalize medical history, until recently medical treatment could be characterized as the history of the placebo effect, since almost all medications were effective primarily because of the placebo effect (5). Placebo effects have been appreciated throughout medical history but are most easily appreciated only when attributed to others. It is impressive that one's sensitivity to the placebo effects of others is no guarantee against developing beliefs in one's own placebo effect. There are many historical illustrations, few or no exceptions (5, 6), and at least 3 studies that demonstrate this kind of defensiveness (12–14, 21).

Evaluation of outcome is more common in behavior therapy than in other kinds of psychotherapy.

This claim is without substance, since most clinicians claim that they continually evaluate outcome, reformulate hypotheses, and reevaluate outcome. More important are the specific procedures that are used and the type of evidence collected to assess outcome.

The APA task force report goes on to describe three types of research designs that have been used to assess the effectiveness of behavior therapy.

The individual subject, or own control design has shown the efficacy of behavior therapy in treating a variety of disorders.

This efficacy was illustrated by several studies of changes in the ward milieu and social behavior of psychotic patients (22, 23), and claims were made for similar effectiveness in neurotic and mentally retarded patients.

The multiple-baseline design is considered an important and frequently used method.

In this study design, several patient problem behaviors are measured and the therapeutic intervention is used for only one of the behaviors. If that behavior changes, it is argued that the treatment intervention is specific. If all of the behaviors change, it means that the effect is nonspecific, i.e., it is a control for the placebo effect of treatment.

Let us consider the two previous designs together by briefly discussing the data in the literature on the placebo effect (8). All clinical studies using placebos as controls report positive, neutral, and negative placebo effects. Placebo effects occur more frequently in clinical situations than in laboratory experiments. These two well-established observations suggest that placebo effects would be highly likely concomitants of behavior therapy. The own-control and multiple-baseline designs lead to the *post hoc, ergo propter hoc* fallacy. It is an elemental and generally accepted principle but usually applied to someone else's theory or therapy and frequently overlooked about one's own (12, 14). It is common in medicine among clinicians and especially psychotherapists and psychoanalysts. Many behavior therapists, including many experienced researchers, commit this error.

Behaviorists point out that therapeutic change during psychoanalysis cannot safely be attributed to insight into the unconscious. Without additional evidence, it must also hold that therapeutic change cannot be attributed to conditioning procedures.

My impression, from reading many case reports and from clinical contact and consultation with behavior therapists, is that much more than the application of conditioning principles goes on in the treatment of patients by behavior therapists. In fact, the actual conditioning seems to account for only a very small part of the treatment compared to various other potential "placebogenic" factors, such as support, education, persuasion, exhortation, drugs, and many other non-

specific factors used to one extent or another in all therapies. The recent trends toward treating the whole patient or family and broad spectrum behavior therapy are testimony to the use of many modalities. In fact, as I read and hear reports of the treatment of patients, I am impressed that behavioral procedures are very similar to my own, except that the descriptive jargon differs from my jargon.

I was disappointed by the claim of behavior therapists that their therapy is superior to other therapies, a claim that is based on their self-assessed clinical reports compared with the self-assessed clinical reports of others. Behavior therapists rigorously criticize clinical studies reporting success in psychotherapy, but the same rigor is not applied to studies of successful behavior therapy.

But all of these speculations are unimportant compared to the definitive basis for evaluating the effectiveness of a treatment, that is, the data from carefully controlled studies, as described in the following section.

The experimental group/control group design can provide useful information on the efficacy and cost-effectiveness of behavior therapy.

This design, used extensively and successfully in drug studies (24–26), would compare behavior therapy with a control that may be an inert procedure, an active but nonspecific procedure, or another traditional or standard therapy. Although establishment of efficacy requires this kind of evidence, the task force report describes only 2 frequently cited studies of this type (27, 28). Paul's study (27) of desensitization therapy was seriously flawed because the subjects were nonclinical volunteers with anxiety in public speaking situations, and his findings could not be replicated in a clinical study of the treatment of children with phobias (29). The other research cited was Gelder and Marks' study (28) of classic phobias in outpatients who were treated weekly for about a year with desensitization, group, or individual therapy. Desensitization therapy led to the most rapid and best improvement. These 2 studies are inadequate evidence for, and do not warrant, the sweeping conclusions of the task force report about the efficacy of behavior therapy.

As part of a comprehensive review of the evidence for the effectiveness of psychotherapy, Luborsky (30) compared behavior therapy with psychotherapy. In his review he included only studies with genuine patients and excluded those with student volunteers, subjects with habit disorders such as bed-wetting, individuals with addictions such as alcoholism, and subjects with other bad habits. Nineteen controlled comparisons in 12 studies between behavior therapy (primarily desensitization) and individual therapy met minimum standards for inclusion in the review. Thirteen of the comparisons showed no difference in efficacy, 6 favored behavior therapy, and none favored individual psychotherapy. Five of the 6 comparisons favoring behavior therapy were of poor research quality and received the lowest ratings for adequacy of methodology. In addition, the therapies were brief, most of the studies were conducted by behavior therapists, and there was a tendency for benefits to appear early with behavior therapy and later for psychotherapy. This last finding may be related to the fact that behavior therapy is more structured and time-limited than psychotherapy. Luborsky could not draw any conclusions from these data.

In an important recent study (31) 94 psychiatric outpatients with anxiety neurosis or personality disorder were treated for 4 months by senior behavioral or psychoanalytically oriented therapists or assigned to a waiting list control. At 1- and 2-year follow-up assessment with multiple, comprehensive measures, all groups were equally and significantly improved.

In another study (32), hyperactive children were treated for 8 weeks with methylphenidate alone, behavior therapy and methylphenidate, or behavior therapy and placebo; they were evaluated with multiple measures during therapy and 4 weeks after therapy. In a preliminary report for 36 subjects, significant improvement occurred with all of the treatments but was significantly greater and less costly using methylphenidate alone.

Some studies have questioned the results of aversive conditioning in alcoholism (33), implosive therapy and flooding procedures (34), the clinical significance of self-control of cardiac functioning (35), and the evidence supporting the presumed specific or essential procedures used in systematic desensitization (36).

Wolfe (37), in a study of the major findings from 20 review articles in the literature, concluded that

> 1) The research on the behavior therapies is surprisingly lacking in rigor. Pawlicki (1970) estimates that only 4 percent of the studies met Eysenck's three criteria of control group, baseline measurements, and follow-up assessments.
>
> 2) There appears to be an increasing awareness on the part of behavior therapy practitioners of the complexity of clinical disorders. This is evidenced by the fact that most of the reviews discussed ways of making a particular therapy more effective by combining several techniques, by focusing more on imagery techniques, and by emphasizing the patients' responsibility in developing their own behavior program.
>
> 3) While one or another behavior therapy has achieved some positive results for some patients with specific problems, generally these therapies produce part-successes, alleviating only some aspects of the patient's problems. Furthermore, a problem which afflicts all of the behavior therapies is their typical failure to produce results which generalize beyond the treatment setting.[2]

Another theme in the history of medicine is apparent: first the optimists, then the skeptics, and finally, one hopes, a reality synthesis.

[2]Reprinted by permission from "The Success of the Behavior Therapies: A Report on 20 Reviews," by Barry E. Wolfe (unpublished manuscript), 1974. Copyright 1974, Barry E. Wolfe.

Behavior therapy is highly effective, ameliorative, or promising in the treatment of many disorders.

The task force report concludes that behavior therapy can be used in the treatment of phobias, anxiety, enuresis, stuttering, obsessive-compulsive behavior, hysteria, encopresis, psychological impotence, transvestism, exhibitionism, gambling, obesity, anorexia, insomnia, nightmares, temper tantrums, head bumping, thumb-sucking, excessive scratching, and excessive verbal demands. The report concludes that it is also useful in treating rebellious behavior, sibling rivalry, isolate behavior, elective mutism, hyperactivity, difficulties in social interaction, classroom disruption, failure to study, low academic performance, social behavior in chronically ill mental patients, verbal and nonverbal behavior of psychotic and schizophrenic children, and the tics associated with Gilles de la Tourette's syndrome.

My response to these claims is to refer to Garrison (38), America's major medical historian, who was able to discern some lawfulness in the historic sea of medicine's ineffective remedies. He observed that "Whenever many different remedies are proposed for a disease, it usually means that we know very little about the disease, which is also true of a . . . [remedy] when it is vaunted as a panacea or cure-all for many diseases" (p. 25).

Contemporary psychotherapy, including behavior therapy, is consistent with this history. Many psychotherapists claim superior results in the treatment of psychological illness, and each tends to claim cures for many illnesses (12–14, 39).

The statements in the APA task force report about the effectiveness of behavior therapy in Gilles de la Tourette's syndrome may illustrate the tendency to uncritically exaggerate outcome data. The effectiveness of behavior therapy in treating this syndrome is referred to three times in the report, with four supporting references:

> behavior therapy techniques have . . . been shown to be effective in reducing the characteristic multiple tics [in Gilles de la Tourette's syndrome] (19, 40). (p. 29)
>
> Behavior therapy has been highly effective with . . . tics associated with Gilles de la Tourette's syndrome (19, 40). (p. 36)
>
> [Negative practice] . . . has been successfully used with some patients in whom the tic was part of a Gilles de la Tourette's syndrome (20, 41). (p. 22)

My subsequent comments are based on my experience with and study of over 250 patients and treatment of about 150 patients with Gilles de la Tourette's syndrome (16, 42, 43).

All of the case reports referred to in these references (19, 20, 40, 41) were carefully reviewed. A detailed review will be published elsewhere (43). Of the 17 patients, 1 had blepharospasm (41), 2 did not fulfill the criteria for a diagnosis of Gilles de la Tourette's syndrome (20), and for 6 the information was inadequate to make the diagnosis (20, 44). Of the 6 with inadequate information, 4 had remissions that were incomplete or not generalized, 2 were treated with concomitant chlorpromazine or amobarbital. Of the remaining 8 patients with adequate information for the diagnosis of Gilles de la Tourette's syndrome, 1 withdrew from treatment (20), 1 failed to improve (20), and 3 were reported to have improved at 4-year follow-up; unfortunately, two of these reports were based on self-reports from the patients (20). The results for the remaining 3 patients were unconvincing and not supported by the data; 1 patient was treated with concurrent haloperidol (40), another with thioridazine (19), and the third patient with extensive psychotherapy and residential treatment for 2 years (19).

Thus of 8 patients with adequate information for the diagnosis of Gilles de la Tourette's syndrome, 2, or 25%, may have improved. This amount of improvement is exactly the same as that obtained by our research group for patients who were evaluated 7 months to 8½ years after their initial evaluation (16). The average improvement for patients not receiving haloperidol was 25%, which contrasts with a 75% average improvement for patients receiving haloperidol. These trends were confirmed by two studies (45, 46) of single patients treated with massed or negative practice and aversive stimulation. Symptoms in both patients were decreased only during the period of treatment and were markedly and consistently reduced in 1 patient (45) after treatment with haloperidol.

Our research group has studied over 250 patients with Gilles de la Tourette's syndrome. About 38 were previously treated with various behavioral procedures in other centers, and 2 patients were similarly treated, unsuccessfully, in our laboratory. Many patients reported that their symptoms decreased while they concentrated on the procedure (all such patients report that their symptoms decrease when they are absorbed in nonanxious tasks), but none reported any permanent, lasting, or generalizing effect outside of the treatment situation. Although we may be seeing a biased sample of failure (the successes may not get to us), I think it unlikely that behavior therapy has any substantial beneficial therapeutic effect on the symptoms of Gilles de la Tourette's syndrome. Considering the extensive negative clinical experience, the inadequate evidence from the literature about the effectiveness of behavioral techniques, and especially the successful use of haloperidol (15, 16), I would consider the recommendation for and use of behavior therapy in treating this syndrome as indefensible at the present time, except perhaps as an experimental procedure requiring informed consent by the patient.

Yates (20), who is frequently referred to and extensively quoted in the APA report and who has studied and treated tics quite extensively, is more thorough, cautious, and tentative about his conclusions than the members of the APA task force. He has stated,

> Virtually all of the studies reviewed are seriously defective in the adequacy of follow-up data. (p. 201)

> Thus the results reported by behavior therapists must be treated with extreme caution and should be regarded for the present as experimental studies of the control and manipulation of these forms of maladaptive behavior, rather than as therapeutic in intent or result. (p. 202)
> One of the major problems of the behavior therapist relates to the transfer of results obtained in a laboratory to real-life situation. . . . Results produced in an impersonal laboratory situation are not very likely to transfer very strongly outside the laboratory. Hence, the strictly laboratory-based studies must be regarded as a preliminary to the attempt to abolish the behavior outside. (p. 203)
> [such studies have] . . . only begun to be applied to the control and modification of tics. (p. 204)

I wonder how many of the other enthusiastic claims in the task force report would weather a critical, in-depth analysis of outcome.

CONCLUSIONS

My impression is that the APA task force report is a well-written, concise summary about behavior therapy that successfully serves to inform psychiatrists unacquainted with the principles of behavior therapy. However, I do not believe that the evidence supports the extensive claims made about the clinical effectiveness of behavior therapy. The arguments, in general, are similar to the claims of all clinicians, past and present, except that they are couched in experimental research parlance.

Despite these limitations, enough clinical evidence has been amassed to seriously consider behavior therapy as a therapeutic breakthrough. However, it is a promise as yet unfulfilled. A major potential asset of behavior therapy is the tendency by psychologists (especially experimental psychologists), who use it most, to study and evaluate their procedures and results. Unfortunately, the studies have not been very successful and suffer from inadequate methodology. The therapeutic claims for behavior therapy are excessive and have outdistanced the controlled clinical evidence. Behavioral procedures are being used in an undisciplined way, too extensively, and by inadequately trained clinicians. In this sense behavior therapy may be the latest fad.

Sufficient data have been obtained to suggest that clinicians should become further acquainted with behavior therapy. I would consider using it and would refer patients to skilled therapists for treatment of selected clinical problems. I would also recommend that psychiatric residents interested in learning about and using behavior therapy have available to them a skilled behavior therapist for teaching and supervision. However, I would consider it premature to replace other more traditional (especially data-oriented) therapeutic techniques with behavior therapy until evidence of its effectiveness, in the form of controlled comparative studies, becomes available. I am somewhat concerned and would recommend to behavior therapists that standards of education and clinical experience be established to prevent unqualified and potentially dangerous treatment for unwary patients. It should be appreciated that these remarks, although directed to behavior therapy because it is the subject being discussed, apply equally to all schools of psychotherapy (8).

The question of whether behavior therapy is a therapeutic breakthrough or the latest fad can best be answered in the future only after adequate evidence in the form of well-controlled studies has accumulated. Unfortunately, current attempts to evaluate the behavioral therapies have not been very successful and suffer from inadequate methodology. At the present time, therefore, the balance is more in the direction of the behavioral therapies being the latest fad rather than a therapeutic breakthrough.

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Psychiatric Aspects of Dwarfism

BY JAMES S. BRUST, M.D., CHARLES V. FORD, M.D., AND DAVID L. RIMOIN, M.D., PH.D.

Sixteen adult dwarfs–11 with achondroplasia and 5 with hypopituitarism–were studied by means of psychiatric interviews and psychological tests. There were no significant differences between the two groups; in general, the subjects had achieved a satisfactory life adjustment despite the stress of having bodies uniquely different from those of the general population. They had secure identities as "little people" and successfully used coping mechanisms such as a sense of humor and a pleasant interpersonal style. Male dwarfs tended to experience more emotional distress than female dwarfs.

ALTHOUGH INTENSIVE medical research has provided much information regarding the diagnostic classification, etiology, and, more recently, treatment of dwarfism (1–3), there has been relatively little investigation into the psychological and emotional aspects of these disorders. This report represents a review of the literature and a clinical study of the psychological status of two groups of adult dwarfs. One group consisted of subjects with achondroplasia, a disorder that results in disproportionate short stature: the arms and legs are small in proportion to the trunk. This disorder is inherited as an autosomal dominant trait, but the majority of the cases are sporadic and presumably represent new mutations. The second group consisted of subjects with hypopituitary dwarfism, a disorder that results in proportionate short stature and may be associated with other pituitary hormonal deficiencies, particularly of the gonadotropins.

The authors are with the School of Medicine, University of California, Los Angeles, where Dr. Brust is Assistant Clinical Professor of Psychiatry, Dr. Ford is Adjunct Associate Professor of Psychiatry, and Dr. Rimoin is Professor of Pediatrics and Medicine. Dr. Ford is also Head Physician, Psychiatric Liaison Service, and Dr. Rimoin is Chief, Division of Medical Genetics, Harbor General Hospital, Torrance, Calif. Address reprint requests to Dr. Ford, Department of Psychiatry, Harbor General Hospital, 1000 West Carson St., Torrance, Calif. 90509.

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REVIEW OF THE LITERATURE

Of those psychological elements which have been studied in dwarfs, intelligence has received the most interest. Most authors have reported that dwarfs have normal intelligence with a normal distribution (4–10); however, Spencer and Raft (11) found an average IQ of 83 in two subjects with hypopituitary dwarfism. There has also been some evidence of a verbal-performance disparity in subjects with pituitary (12) and achondroplastic (4) dwarfism. Studies concerning level of school performance have reported conflicting findings (9, 10).

Many authors see a potential for the development of emotional difficulties in individuals of dwarfed stature. This follows from the observations that merely the small size attracts unusual attention and that there is a tendency for the dwarf to be treated as if he were younger (4, 5, 7, 9, 12–18). Adolescence has been regarded as an especially difficult period (14). Personality traits are not stereotyped, but the frequent occurrence of some traits has been noted. These include immaturity and dependency (7, 14), extroversion (12), latent depression (7, 12, 15), and the absence of overt aggressive behavior (15). Ego-defense mechanisms include denial (14, 16, 17), exaggeration (7, 14), obsessive-compulsive defenses (15), and withdrawal or social isolation (4, 17, 19). Other efforts that dwarfs use to achieve mastery over their specific situation include adopting the role of mascot (14, 17), the use of humor (14), and the use of scholastic achievement (10). Special note has been made by Money and associates (4, 17) of "chronic cheerfulness" and "optimism" in children with achondroplasia.

Money and Pollitt (16) have investigated the psychological response of hypopituitary dwarfs to treatment with human growth hormone. It was their feeling that the degree of psychological maturation in the dwarfed child under treatment related not so much to the amount of growth, but rather to the ability of the parents and other adults to treat the child according to his age instead of his size. They have also described a "readjustment syndrome" reflecting a range of difficulties in accepting the treatment. Symptoms varied from feelings of pressure to perform better to outright refusal of the treatment. This seems to be due to an identity crisis brought about by the prospect of change from a familiar, albeit abnormal, situation. Another

response to such treatment has been decreased denial and immature behavior coupled with increased overt depression and anger (7).

METHOD

The Medical Genetics Clinic at Harbor General Hospital treats a large number of dwarfed patients. From the census of this clinic, patients with a definite diagnosis of either hypopituitarism or achondroplasia were asked to volunteer for the study. Ninety percent of the persons approached agreed to participate.

The sample consisted of 4 men and 7 women with achondroplasia and 3 men and 2 women with hypopituitary dwarfism. The subjects ranged in age from 19 to 80 years, with an average age of 33 for the dwarfs with achondroplasia and 47 for the hypopituitary dwarfs. The height for both groups ranged from 42 to 57 inches, with an average of 51 inches, which is typical for persons with these disorders (1).

Each subject was seen one or two times in an individual psychiatric interview by one of us (J.S.B.). The interview consisted of a structured portion to obtain certain specific information and a nonstructured portion to allow the development of personal information. The structured portion of the interview included queries into demographic information, aspects of the stress of being a dwarf, and childhood history. Interview data were recorded on a standardized protocol form.

In addition, each subject was given a battery of written psychological tests to be taken home, completed, and mailed back to the examiner. These tests included the Comrey Personality Scales, Shipley-Institute of Living Scale for Measuring Intellectual Impairment, Multiple Affect Adjective Check List, and a modified form of the Draw-a-Person test. On this last test, the subject was asked to draw a picture of himself or herself, then turn the paper over and draw a picture of a normal-sized person.

RESULTS

Since no significant differences were found between the hypopituitary and achondroplastic subjects, results are reported for the total sample.

Marital Status and Living Situations

Eight of the 16 subjects were married and living with their spouses, 3 of whom were of normal size. Four subjects were living with their childhood families, 2 with other adults, and 2 were living alone. Within the total sample there had been one divorce and one annulment of marriage. Fourteen of the dwarfs were living productive lives of employment, school attendance, and/or homemaking. Of the 2 who were not productive, one immature woman was supported by her family, and a man with painful spinal disease was disabled. Four of the subjects were parents.

Childhood Histories

Twelve of the 16 dwarfs had lived in intact families until at least the age of 16, and only 3 of the sample described excessive strife in their childhood families. Four (25%), all with achondroplasia, had one or more relatives who were also dwarfs.

In response to questions about their childhood personalities, 12 (75%) described themselves as happy, 9 (56%) as outgoing as opposed to shy, 8 (50%) as obedient, and 5 (31%) as mischievous. Fourteen of the subjects (88%) described one or more habit disturbances during childhood, including nail-biting (8, or 50%), thumb-sucking (6, or 38%), and enuresis past age 5 (5, or 31%).

All subjects noted that to some extent they had been teased as children. Ten of the 16 (63%) had dealt with the teasing in predominantly passive ways, usually by ignoring it or walking away. Only 6 (38%) had made any sort of an active response, and this was invariably a verbal rather than a physical one.

The majority of the subjects (12, or 75%) remembered liking school, and 10 described themselves as above-average students. Only 2 had failed to graduate from high school, and 8 of the 16 had education beyond the high school level.

Although over half of the subjects (11, or 69%) began dating later than their peers, most of the subjects (11 of 16) eventually made a satisfactory sexual adjustment. Most had at least some dates with people of average height.

Stress Situations Unique to Dwarfism

Nine of the 16 subjects reported that they were not really aware of being different from other people until after they had started school, and some reported no awareness of this until their teens or 20s. Of the 6 subjects to whom the question was applicable, 5 reported no special feelings when a younger sibling passed them in height. Twelve stated that they were treated in accordance with their apparent age, not their actual age.

All agreed that many things in the environment were designed for taller people, and this caused them personal discomfort or inconvenience. Most frequently mentioned were public telephones and shelves in stores. The men commented on the height of urinals in public restrooms.

All of the subjects agreed that job discrimination was a problem. Most felt that it was a combination of the employer's feeling uncomfortable with them and their appearance and the employer's fear that they could not complete the duties of the job because of either their size or intelligence.

All of the subjects were cognizant of the popular expectation that dwarfs are supposed to be "jolly." When questioned about this, all of them gave an answer that approximated, "Some are, some aren't, and some act that way." They did not believe the jolly

quality to be typical and felt that it often represented role playing rather than true feelings.

When questioned about their attitudes concerning children, 9 of the 16 had a firm preference for having short-statured children rather than children of normal height. They cited a variety of reasons for this preference, including difficulties in disciplining a normal-sized child, a greater feeling of identity with a dwarf child, and concern that a normal-sized child would be embarrassed by his dwarf parent. Also mentioned was the somewhat illogical notion that the dwarf parent is better able to help the short child adjust to his height (of course, a normal child would not require such help). The 5 subjects who favored normal-statured children frequently cited less emotionally charged reasons such as, "I'd rather my child not have achondroplasia because people with achondroplasia develop bone and joint disease." Two subjects had no preference regarding this issue.

Exploring the relative merits and disadvantages of proportionate and disproportionate dwarfism, most of both the hypopituitary and achondroplastic dwarfs preferred their own group but thought that each group had certain advantages. The person with achondroplasia was viewed as physically stronger, certain to be sexually mature, and more likely to look his age. The advantages attributed to hypopituitarism included a more attractive physical appearance, greater ease in obtaining clothing that fit, and the increased probability of obtaining jobs in show business. (However, only 3 of the total sample, all hypopituitary dwarfs, were deriving their principal income from show business.) Another advantage of pituitary dwarfism, which was not mentioned by any of the subjects, is that it can be treated if detected early. (There is no treatment for achondroplasia.)

In response to the hypothetical question "Would you accept treatment to make you taller if such treatment existed?" only 3 subjects expressed a willingness to accept such hypothetical treatment, and even they acknowledged that it would create an adjustment problem.

In regard to the overall adjustment to being unusually short, 6 subjects actually viewed shortness as an asset, usually pointing out that it made them "special." Nine dwarfs saw problems in being so short but felt they were able to cope with them. Eleven subjects (69%) identified prejudice and interpersonal difficulties as their major problems, while 5 (31%) were most concerned with physical inconveniences. One dwarf had no opinion on this subject.

Clinical Evaluation

When asked to assess their own mood, 15 of the subjects described themselves as happy or content, and 6 of these qualified their statements by adding that they were occasionally depressed or irritable. One subject described himself as depressed.

On the examiner's assessment all demonstrated appropriate affect. There was no euphoria. There was some evidence of depression in 3 subjects, one of whom also evidenced some anxiety and hostility. None of the subjects was psychotic.

Ten subjects were judged to be mature as evidenced by good judgment, realistic goal formation, effective planning, and adequate self-esteem. Five others were judged less mature because of their greater than usual dependency upon their parents or their continuing interest in issues generally associated with adolescence. One subject with hypopituitarism was grossly immature, with childish speech patterns and self-image. She displayed a clinical picture consistent with 3 cases reported by Spencer and Raft (11).

Ego-defense mechanisms readily identified in the clinical interview or by assessment of behavior included humor, altruism, suppression, and passive-aggressive behavior. These terms are used as described by Vaillant (20). Humor was employed by 13 of the subjects; for example, one of them commented, "The shorter the dwarf, the longer the name of the diagnosis." Thirteen of the 16 used suppression to minimize discomfort; for example, a typical response to a question about what it is like to be a dwarf was, "It's not bad." Altruism was identified in 8 subjects. An example is a high degree of activity in recruiting members for the Little People of America, a self-help organization. Passive-aggressive behavior was noted in 8 subjects. A typical example was a subject who would repeatedly postpone the interviews and then delay returning the psychological test material despite professed interest in and appreciation for the study.

Psychological Testing

Fifteen of the subjects completed the psychological test battery. Because there were completed test materials for only 4 subjects in the hypopituitary group, the 2 diagnostic groups were not compared for statistical significance. Sight evaluation, however, did not suggest any significant differences.

Anxiety, hostility, and depression as measured by the Multiple Affect Adjective Check List indicated that the group as a whole was representative of the general population.

The Comrey Personality Scales compare a variety of personality factors including trust versus defensiveness, social conformity, and masculinity versus femininity, among others. Averaged test scores for these factors indicated that the female dwarfs appeared to be quite average compared with the norms. However, the men had more abnormal scores, and their average was more than one standard deviation from the norm on those scales indicating a tendency toward decreased masculinity, increased conformity, and a tendency to answer questions in a socially desirable manner.

On the modified Draw-a-Person test the subjects correctly perceived themselves as smaller than normal persons and, in fact, they tended to draw themselves as even smaller in proportion to normal persons than they actually were. The average ratio of the subjects'

height to the normal average height was .77, but the ratio of their height to the normal height in the drawings was .72 (where the height of the normal person equals 1.0). Achondroplastic dwarfs have disproportionate bodies. Their heads and trunks are normal-sized while their limbs are considerably shortened because of bone dysplasia. Their heads may appear to be even larger because of frequent frontal bossing. The ratio of the upper body segment to the lower body segment, which in normal persons is slightly less than 1.0, is increased in persons with achondroplasia. The achondroplastic subjects correctly perceived this, drawing the normals with an upper-lower body segment ratio of .96 and themselves with a ratio of 1.27. In addition, the subjects with achondroplasia drew themselves with larger heads than the normals. The ratio of head height to total height was .25 in self drawings and .17 in drawings of normals (where total height equals 1.0). This is consistent with the actual appearance of persons with achondroplasia.

Average IQ, as measured by the Shipley-Institute of Living scale, was 120 for the entire group (125 for the achondroplastic dwarfs and 105 for the hypopituitary dwarfs). These figures must be evaluated with caution since the tests were self-administered, but the clinical impressions did support the finding of above-average intelligence.

DISCUSSION

The degree of psychological well-being and confident self-identity of these adult dwarfs was striking. Their positive adjustment to life despite the stress of a major physical deformity was demonstrated by their ability to maintain productive lives including employment, marriage, and parenthood. Psychological testing confirmed the clinical impression of a relative lack of psychiatric symptoms, excessive anxiety, or depression. A secure sense of identity was suggested by the large number of dwarfs who regarded themselves as "special," would prefer a short to normal-sized child, and who would reject a hypothetical treatment to make them average height. The Draw-a-Person test data confirmed that they had a realistic acceptance of the difference between themselves and others.

We emphasize that what we observed is related to the successful employment of learned coping mechanisms. Our data indicate that as children, the subjects experienced increased inner tension, as evidenced by habit disturbances such as nail-biting and thumb-sucking. They also used denial and repression, as indicated by their failure to comprehend that they were different from other children and their failure to react when a younger sibling passed them in height. Passivity and suppression were used to deal with teasing from other children.

However, by adulthood most dwarfs had learned that the important issue was not their height, but rather the way in which their height affected others. Accordingly, one of the most important coping mechanisms demonstrated was the ability to set others at ease. This was accomplished through a pleasant interpersonal style with frequent use of humor. Participation in the self-help organization, Little People of America (19, 21), assisted some subjects in their identification as a small person and allowed expression of the healthy ego mechanism of altruism. Negative feelings were generally expressed by means of passive-aggressive behavior, and in view of the size differential between themselves and normal people, this appears to be a reasonable solution.

Our finding that adult male dwarfs seemed more disturbed than adult female dwarfs, as evidenced by the results of the Comrey Personality Scales as well as the interviews, can be explained by cultural factors. Masculinity is often symbolized by size and power. Men are expected to be taller than women. The male dwarf finds that not only is he shorter than other men, he is also shorter than almost all women. We hypothesize that it is easier for the female dwarf to accept her shorter stature because it more closely approximates cultural attitudes.

The impression of Money and associates (4, 17) that children with achondroplasia are almost always cheerful and optimistic was not shown to be carried into adulthood. Although many of the dwarfs did appear to be cheerful by either their own report or objective evaluation, this was not felt to be chronic or universal. We suggest that the dwarf's cheerful demeanor is a part of learned coping mechanisms aimed at placing others at ease and therefore facilitating interpersonal relationships. Our data did not support the concept of constitutional euphoria.

The subjects for this study do not represent a random sample of the dwarf population, since we studied only those persons available to us through a medical clinic. It is entirely possible that these people represent a more active and psychologically healthy group and that there are withdrawn and isolated individuals who are not a part of the population from which we could draw.

What is important to stress is that there does exist a sizable group of dwarfs who function well as psychologically healthy and productive members of the community.

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Viral Encephalopathy Mimicking Functional Psychosis

BY LAWRENCE G. WILSON, M.D.

Encephalitis, particularly herpes simplex encephalitis, frequently presents as a disorder with puzzling psychiatric symptoms before frank evidence of central nervous system involvement is apparent. The author describes three cases of encephalitis characterized by abrupt onset of bizarre psychological disturbance in the absence of gross neurologic dysfunction. Each patient was initially diagnosed as schizophrenic but later became critically ill and recovered only after a long and chaotic hospital course. The author warns psychiatrists and staff on psychiatric impatient units against mistakenly diagnosing cases of early encephalitis as functional psychoses.

CASES IN WHICH a patient presenting with a psychiatric problem is later found to have an organic illness that may explain the entire clinical picture are common in psychiatry. Herpes simplex encephalitis, the most common form of nonepidemic fatal encephalitis in the United States, frequently presents just this situation. The difficulty of characterizing the symptoms of herpes simplex encephalitis is such that it has been ranked with syphilis as a "great imitator" (1). One review of herpes encephalitis cases (2) reported that all of the adult patients displayed largely psychological symptoms in the early stages of illness, with neurologic symptoms developing later. In another report (3), 10 cases of encephalitis were described in which psychiatric symptoms were the most prominent aspect of the early stage of the illness. Other clinicians have noted that the early course of encephalitis specifically simulates that of a schizophrenic illness (4–6). Weinstein and associates (7) described 6 cases of encephalitis (probably postinfectious type) in which the initial symptoms were very similar to those of many early schizophrenic psychoses. It is of interest that in 4 of the 6 cases severe emotional stress preceded the onset of the disorder.

Thus, patients with this illness frequently are seen by a psychiatrist or admitted to a psychiatric service and diagnosed as having schizophrenia or another psychiatric disorder. Accurate diagnosis of herpes encephalitis is exceedingly difficult (8) and in the past was mainly of academic interest, since supportive care was all that could be offered. With the investigation of several agents (idoxuridine and cytosine arabinoside) that are possibly effective in the treatment of herpes encephalitis (9), differential diagnosis has become more important, since the reported mortality of untreated cases has varied from 50% to 70%. In addition, survivors have frequently suffered neurologic impairment that sometimes resulted in institutionalization. This devastating mortality and morbidity might be substantially reduced if treatment were begun early.

The psychiatrist is sometimes confronted with a patient who responds atypically to treatment for the presumed psychiatric condition but for whom a diagnosis of encephalitis cannot be ruled in or out. Hollander and associates (1) described one case in which neurologists and psychiatrists debated over the diagnosis for a number of days before encephalitis became clearly manifest. In such situations it is important to examine the individual case in detail to determine the presence or absence of differentiating signs of psychiatric and neurologic disease. Even with a detailed family and personal history and adequate physical and laboratory examination, it is often not possible to make a diagnosis. In such cases the patient must be cared for by an interdisciplinary team to give him maximum medical and psychiatric support.

The following 3 case reports demonstrate the difficulties with diagnosis and early management of encephalitis. In all 3 cases, psychiatrists were the key persons involved in management until definite neurologic symptoms developed.

CASE REPORTS

Case 1. The patient was a 30-year-old divorced mother of two who was brought to the hospital by her mother. She was unable to relate a coherent history but did say "I'm crazy—maybe I've been crazy all my life." She said she was "living in a dream" and had felt "unreal" for 2 weeks. The mother said the patient had become despondent and agitated about 2 weeks before and had poor appetite and sleep. She had reportedly lost 10 pounds over this period and had become more disorganized and confused.

The patient had been involved in an affair with her employer and expected to marry him after he was free from his wife. However, several weeks before the patient's admission he had taken his wife and family on an expensive vacation to

Dr. Wilson is Staff Psychiatrist, Veterans Administration Hospital, 4435 Beacon Avenue South, Seattle, Wash. 98108. He is also Assistant Professor, Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, Wash.

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the "island paradise" that the patient expected to move to with him when they were married. Her symptoms began at that point, and her mother stated "she acted like her dreams were shattered." She was described as previously being "bull-headed" and "high-strung" but free of major emotional problems. There was no history of psychosis in the family, but there was a history of suicide and alcoholism in previous generations.

On examination the patient appeared frightened and agitated. Formal mental status testing was difficult since her answers to questions regarding time and place were flippant, tangential, or inappropriate. Her speech at times was incoherent, at other times scanty and disorganized. There was no evidence of hallucinations or delusional ideas. She was preoccupied with being "crazy" but later also claimed that everyone was crazy but her. She appeared fearful but responded well to firm direction, relating to the examiner in a childlike manner.

Physical examination disclosed a pulse rate of 104 per minute and normal temperature. The ocular fundi revealed no abnormalities. Neurologic examination was normal with the exception of symmetrically increased deep tendon reflexes. Admission hematologic examination, urinalysis, serum electrolytes, and screening blood chemistry studies were within normal range.

The patient was hospitalized with a provisional diagnosis of acute schizophrenic episode, with seemingly clear-cut precipitating stress in her personal life. She was begun on 400 mg chlorpromazine (Thorazine) per day. After several days of increasing medication she began to display intermittent mutism, staring, exaggerated poses, and immobile postures. This behavior was mixed with babbling and giggling. Medication was further increased after the appearance of waxy flexibility, and ECT was considered because she refused to eat. Ward nurses noticed the patient occasionally "dragging" her right leg, but cursory examination was unremarkable. The patient remained afebrile. On the 10th hospital day she began drooling and later began to choke and aspirate her secretions.

A neurologic consultation was obtained, and the patient was found to have an absent gag reflex and difficulty swallowing but no other specific neurologic abnormalities. An electroencephalogram (EEG) showed a diffusely abnormal pattern compatible with metabolic encephalopathy, with very slow delta wave activity predominating. Chlorpromazine was discontinued and the patient was transferred to the neurology service. The patient was thought to be suffering from an acute metabolic encephalopathy secondary to chlorpromazine or from some type of intracranial pathology with resulting organic brain syndrome.

On the neurology service the initial cerebrospinal fluid examination was normal. Skull films and brain scan showed no abnormality. The patient began to have a mild temperature elevation to 38.5° C (101.3° F) and continued to be agitated and exhibit catatonic signs. She also began to have bizarre, rhythmic tonic and clonic contractions of all extremities that were accompanied by gasping respiratory movements. A second cerebrospinal fluid examination again showed normal chemistry and no cells. The patient developed aspiration pneumonitis and became more severely ill. A third cerebrospinal fluid examination was also normal.

Because of the continuing downhill course and the inability to delineate a specific etiology for the illness, catatonic schizophrenia was thought the most likely diagnosis. While in the intensive care unit the patient was given five electroconvulsive treatments over a 2-day period, with brief improvement after each treatment. ECT was discontinued because of electrocardiographic changes and the development of gram-negative sepsis.

Additional history obtained from the employer revealed that both he and the patient had had symptoms of herpes labialis about 2 weeks prior to her admission to the hospital. She had complained of headache, and psychological disorganization subsequently became obvious. At this time a diagnosis of herpes simplex encephalitis was considered. A fourth cerebrospinal fluid examination (on the 22nd hospital day) showed 30 lymphocytes per mm^3, the first evidence in the spinal fluid of a central nervous system infection (see table 1). Spinal fluid protein and glucose levels remained normal. At the height of the illness a serum antibody titer for herpes simplex virus showed an 8-fold increase, later rising to a 64-fold increase. Viral cultures were obtained, the results of which were not available during the patient's severely ill phase. Herpes simplex virus, presumptive subtype I, was isolated from throat cultures, but cultures of spinal fluid were negative for the virus. Brain biopsy was considered but not done because it was late in the course of the illness. No specific treatment for the encephalitis was given, although the patient did require tracheostomy.

The patient's clinical course gradually improved after the aspiration pneumonitis and gram-negative sepsis were adequately treated. As she became more alert to her surroundings, the unusual bilateral dystonic movements ceased and she became combative and abusive to nursing staff. After further improvement she was returned to the psychiatric service. Motor functions were unimpaired, but she was quite emaciated and weak. At this time the patient was speaking coherently but in a rambling, disjointed fashion, with clang associations and perseveration. She showed confusion and disorientation to time and place but never displayed evidence of delusions or hallucinations. On several occasions she had brief depersonalization episodes, which were apparently precipitated by mildly stressful psychiatric interviews. Her behavior initially was similar to that of a 9-or 10-year-old child and later to that of an early adolescent. Repeated EEGs showed gradual improvement but a continuing generalized abnormality. The patient was given no medication in the recovery phase. She gradually improved in orientation and general behavior and eventually a mild memory defect was the only observable residual of the illness; she was discharged after being hospitalized 3 ½ months.

The patient was seen in follow-up over the next 36 months. Her personal involvement with her employer continued, and she remained hopeful that he would eventually marry her. Follow-up treatment consisted of regular reexamination of neurologic functions and supportive psychotherapy oriented around domestic and romantic stresses. The patient consistently appeared bland and used denial to a rather extreme degree. However, there was never any evidence of delusional thinking, inappropriate affect, hallucinations, or other symptoms suggestive of thought disorder. She had a mild memory defect for the first few months after discharge. She felt that her memory for names and numbers was mildly impaired until about 2 years after discharge, when she felt completely normal. Extensive psychological testing 33 months after discharge gave no evidence of organic impairment. Her psychological profile on the Minnesota Multiphasic Personality Inventory was within normal range.

Case 2. This patient was an adopted 17-year-old high school girl who was transferred to a university hospital from a private community hospital. She had been hospitalized for 7

TABLE 1
Results of Cerebrospinal Fluid Examination in Three Cases of Presumed Herpes Simplex Encephalitis

Patient	Hospital Day	Opening Pressure (mm of water)	White Blood Cells (count per mm³)	Percent of Lymphocytes	Protein (mg/100 ml)	Glucose (mg/100 ml)	Culture
1	12	160	1	100	33	104	Negative
	15	260*	0	—	20	76	Negative
	20	150	—**	—	24	88	Negative
	22	170	30	95	15	88	Negative
2	8	50	0	—	15	107	Negative
	10	360	10	100	14	88	Negative
	16	130	8	100	11	80	Negative
	29	130	2	100	15	70	Negative
	41	220	0	—	15	60	Negative
3	4	270	0	—	—***	—***	Negative
	27	425	7	80	14	80	Negative
	30	195	4	100	17	75	Negative
	33	150	8	100	11	80	Negative
	40	470	1	100	12	91	Negative

* Patient straining.
** Bloody tap with clear supernatant.
*** Within normal limits.

days because of the acute onset of bizarre behavior and subsequent lethargy and mutism. The referring psychiatrist believed that catatonic schizophrenia was the diagnosis but sought neurological consultation before consideration of ECT.

The patient had been in good health and had been an active competitive swimmer in high school. There was no history of psychiatric illness in the patient or her adoptive family. She had begun acting bizarrely 3 days before admission, when she began taking notes on family conversations and television programs. She also made many uncharacteristic comments and at times had garbled speech. This sort of worrisome behavior occurred intermittently over a 3-day period. After experiencing chills one night and claiming that "Satan is taking me over," she was taken by her parents to a hospital for evaluation.

Complete records from the first hospitalization are not available. The patient was diagnosed as having an acute psychotic reaction and was initially treated with 7 mg of haloperidol (Haldol) and .5 mg of benztropine mesylate (Cogentin) per day. After several days she became dysarthric and later became mute. Her mother noticed her "limping" with her right leg, "like she'd had a stroke." After continuing this deteriorating course and spiking a fever to 38.9° C (102° F), she was transferred to the university hospital psychiatric service.

Examination showed an uncommunicative young girl who followed the examiner with her eyes but produced only gutteral sounds and occasional garbled speech. Temperature was 38.9° C (102° F) and pulse was 100. Pupils were equal and reactive and the fundi were benign. She showed increased muscle tone of the right extremities and intermittent clonus of the right ankle. Plantar reflexes were flexor. There was a maculopapular rash of the perioral region with some vesicles present. Further history from the parents revealed that the patient had been troubled with a rash on the chin and perioral area for about 1 month.

The patient was transferred to the neurology service immediately after initial examination. An EEG showed very severe generalized disturbance of brain function. A spinal tap revealed normal opening pressure, no white cells, 7 red cells, and normal protein and glucose values. Brain scan and echoencephalogram were normal, as were initial hematologic, serum chemistry, and urine values. In spite of the normal spinal fluid findings, the patient was thought to be suffering from herpes simplex encephalitis. However, a brain biopsy was negative for cell changes typical of herpes, and multiple cultures of spinal fluid, brain, pharynx, and rectum failed to recover any viral, bacterial, or fungal agent. Serological tests for a wide variety of viruses (including herpes, Coxsackie, influenza, mumps, and cytomegalic inclusion virus) showed no titer rise indicative of infection.

The patient continued on a downhill course, at first receiving only supportive care. She developed aspiration pneumonitis but responded to antibiotic therapy. Four additional diagnostic spinal taps showed small numbers of mononuclear cells but normal glucose and protein values (see table 1).

After several weeks of hospitalization, the patient was more responsive to her environment, smiling and reacting to her doctor's comments. Gradually she began speaking recognizably, but repeated words or phrases in an echolalic fashion. She was transferred to a rehabilitation unit where she continued to improve her moderately impaired motor skills. Therapy with 300 mg of diphenylhydantoin (Dilantin) daily was begun because of the appearance of epileptic foci in the EEG. When she was discharged after a total of 60 days' hospitalization she was able to follow simple commands and take care of her personal needs with supervision. She continued to speak in a rambling and irrelevant fashion and was quite distractible in a childlike manner.

After the patient returned home she required close supervision but continued to improve. She pursued her swimming interests and returned to high school, where she received above average grades. She continued to manifest short-term memory disturbance and occasional impulsive behavior 6 months after discharge. Follow-up EEG at 6 months showed continuing abnormalities in both temporal regions. The

patient gradually regained almost complete recent memory function, graduated from high school with a "B" average, and began college. An EEG 16 months after discharge showed only very mild abnormalities.

Case 3. The patient was a 20-year-old unmarried male who lived with his parents. He had been in good physical health until he "passed out" while with a girlfriend at a bowling alley. He was described as having a "spaced-out" appearance, with some possible seizure activity. There was incontinence of urine and a suggestion of head-turning to the right. He expressed fears of being poisoned or "possessed" by his girlfriend.

The patient was hospitalized on a neurology ward of a military hospital for investigation; while there he had probable minor seizures. While hospitalized he had a normal neurological examination, normal EEG, and normal brain scan. He was discharged after several days with only phenobarbital prescribed. In the 24 hours after discharge, he displayed bizarre behavior, disorientation and inappropriate laughing and crying. He also apparently showed clumsiness of his right hand and had some perseveration of motor movements.

The patient was readmitted to the neurology service and extensive investigation was undertaken. A brain scan, two EEGs, and blood and liver chemistry studies were normal. A lumbar puncture showed elevated opening pressure (270 mm of water versus a normal of under 170 mm), but the spinal fluid smears and cultures were negative. The patient's behavior continued to be bizarre, with babbling and incoherence. He was transferred to the psychiatry service after 10 days on the neurology service because a definite neurologic diagnosis could not be made. A diagnosis of catatonic schizophrenia was made, and treatment was begun with chlorpromazine, haloperidol, and benztropine mesylate.

In spite of doses of haloperidol up to 80 mg per day, the patient's behavior deteriorated. He became delusional and claimed he was dead; later he claimed that the devil was in possession of him. He began to show posturing and would hold his extremities in unusual fixed positions for long periods of time. He later became "stuporous," holding his body in an opisthotonic position. Because of the development of pneumonitis, his inability to take food, and the continuing downhill course, he was transferred to a nearby university hospital for electroconvulsive therapy of the presumed catatonic schizophrenia.

On arrival at the university hospital psychiatric service, the patient had a temperature of 38.4° C (101° F). He would occasionally open his eyes and make brief eye contact but was unresponsive to verbal or painful stimuli. The neck was rigid in a mildly extended position and the legs rigidly extended. There was clonus of the right ankle and the gag reflex was absent. The patient was transferred to the neurology service. He began to have prolonged seizures of a temporal lobe status epilepticus type that were very difficult to control. There were no localizing signs on neurological examination and the EEG was suggestive of encephalitis.

Subsequent lumbar punctures (see table 1) showed increased opening pressure and small numbers of lymphocytes. Spinal fluid cultures were negative for bacteria, viruses, and cryptococcus. Another brain scan was normal, as was an echoencephalogram. Tests for porphyria were negative. Serologic tests for a large variety of viruses did not indicate current infection. However, the attending neurologist considered herpes simplex encephalitis the most probable diagnosis. Brain biopsy was considered but was not performed.

The patient developed severe pneumonitis and needed a tracheostomy to control secretions. His seizures were finally brought under control but rigid muscle tone and waxy flexibility remained. However, he became more responsive to the environment and after about 30 hospital days he was able to walk and talk. His intellectual and motor function slowly returned and after about 45 hospital days there were no specific motor, sensory, or reflex deficits. The patient showed very poor short-term memory and decreased intellectual function compared with his pre-illness state. He was transferred to a rehabilitation service for work with his minor speech deficits and to await a return of his short-term memory functions. He remained only on anticonvulsant medications. He was discharged after 2 months of hospitalization to a special school program. An EEG 3 months after discharge was essentially normal, but at 5 months after discharge there was still definitely decreased short-term memory function.

DISCUSSION

Encephalitis has long been noted to produce psychological disorganization in its early stages. As in these 3 cases, differential diagnosis is usually difficult, with signs of brain disease frequently being ambiguous, late, or both. The importance of early diagnosis is clear from these cases. All 3 patients developed probable aspiration pneumonitis and 2 required tracheostomies, which earlier intensive nursing care (usually not available on a psychiatric ward) might have prevented. Intensive medical management of the commonly associated cerebral edema (for example, with corticosteroids) might prevent some of the morbidity of the illness. Although idoxuridine was recently reported to be both ineffective and severely toxic in a large interhospital study (10), early diagnosis may also allow the use of some specifically effective drug agents.

These cases demonstrate the need to continually reevaluate the patient with presumed psychiatric illness who responds atypically to psychiatric management during hospitalization. Once a patient is diagnosed as having a psychiatric problem, ward staff frequently tend to minimize observations that ordinarily indicate organic dysfunction. In our cases the previously diagnosed psychiatric conditions diminished the impact of observations like "limping" and "dragging a leg." In any psychiatric patient with vague or soft neurologic signs or mild disturbance of sensorium (no matter how well explained by supposed severe psychiatric illness) careful observation should be the watchword. The most underutilized of the psychiatrist's techniques, the mental status examination, should be carefully repeated in these cases. This procedure alone would often indicate the neurologic nature of the disorder early in its course. In all of our cases, the severe disturbance in sensorium would probably have become more obvious and the "thought disorder" less impressive with serial testing. Repeated neurologic examinations and special neurologic tests (EEG, brain scan) add another diagnostic dimension. In addition, an amobarbital (Amytal) interview has been found to be valuable as an aid to differentiating

psychiatric from neurologic disorders (11, 12). In catatonia, amytal will usually result in the patient's beginning to talk, and psychotic speech or delusional ideas will become obvious. In encephalitis or other brain disease, the neurologic or communication deficits will usually become more prominent.

The difficulty with diagnosis in our cases was compounded by the late development of evidence of infection in the spinal fluid. Normal spinal fluid findings until the 15th or 20th hospital day are unusual but not unknown in cases proven by brain biopsy to be herpes encephalitis. All 3 cases were diagnosed as catatonic schizophrenia; 2, both before and after neurologic consultation and work-up, and 1 before neurologic consultation. One patient received ECT after intensive neurologic investigation was inconclusive. The 2 others were referred to the university hospital for ECT for their presumed catatonia; both were immediately recognized on admission as having probable neurologic disease. One case had serological evidence (64-fold increase in antibody titer) and positive throat culture for herpes; the other 2 cases had clinical evidence of herpes encephalitis but no specific laboratory evidence. Two of the 3 patients became very seriously ill, with mixed coma and seizure states. All had very slow recoveries with recent memory disturbances many months after discharge. All 3 patients had the same team of attending neurologists involved in their cases and it was their consensus that herpes simplex encephalitis was the most likely diagnosis.

Another problem in the differential diagnosis of encephalitis is the frequent occurrence of psychological or social stress factors in the patient's life. Often these will appear to the examiner to be precipitating causes of a functional psychosis. Patient 1 particularly draws our attention to this factor, since her symptoms began at a time of severe personal disappointment. Did she experience an acute psychotic reaction and *then* develop encephalitis, or did she develop a psychosis during the prodromal phase of her central nervous system viral infection, with the romantic disappointment being merely an interesting, but chance, concurrent event? It is often assumed that the onset of organic illness is related to physical conditions, whereas the onset of psychiatric illness is related to life change or life stress. However, there is increasing evidence (13) that both psychiatric and medical illnesses become manifest during major life changes, disturbing life events, or certain psychological states such as depression. Schmale (14) found 41 of 42 unselected hospitalized patients with medical illness to have evidence of loss (either actual, threatened, or symbolic) and feelings of hopelessness and helplessness prior to the onset of the disease. In another study, Greene and his colleagues (15) found that a high percentage of persons who died suddenly and unexpectedly had been previously depressed.

Some recent work (16) has strongly linked stress, emotions, and immunity. The immunosuppressive qualities of adrenocortical hormones, whose levels are responsive to stress, may be important in the pathogenesis of infectious illness. A variety of viral infections have been related to stress in animals. Certain frustrating laboratory situations have caused increased susceptibility to herpes simplex infection in mice (17, 18). A clinical finding related to this was made in a group of patients with psychotic depression (19). This group had significantly higher levels of antibody to herpes simplex virus as compared with a group of persons with other psychiatric disorders and healthy controls. The investigators considered the possibility that the herpes infection might disturb brain chemistry and lead to depression. They did not, however, consider the possibility that depression and its associated endocrinologic changes might predispose to increased infection with the ever-present herpes virus. Another group has suggested that brain changes as a result of mild or inapparent herpes infection of the central nervous system may result in pathological conditions that lead to aggressive psychopathic behavior. In a small group of patients, Cleobury and associates (20) found that psychopathic persons had higher levels of serum antibody to herpes virus type 1 than patients with other psychiatric disorders or control patients in a general hospital. Thus, there is some evidence that a combination of stress and exposure to virus (or a latent carrier state) may lead to encephalitis and that the impact on behavior of even mild infection may be considerable.

The 3 cases presented illustrate that frequently a diagnosis of schizophrenia is made for an acutely psychotic patient without careful consideration of alternative explanations. It is becoming clear that more than a cross-sectional view of a person is needed for this diagnosis. Some recent work on diagnostic criteria in psychiatry (21) has suggested that the clinician must consider presenting picture, past functioning, and family history before making a diagnosis of schizophrenia. Too quick a reliance on a diagnosis of schizophrenia often precludes further investigation of the etiology of a psychotic state.

Although cases of sporadic encephalitis are infrequent, psychiatrists must be careful to investigate fully any case of psychosis that does not follow a typical course. Inpatient psychiatric staffs must make careful observations to detect at an early stage those cases of encephalitis which initially present as psychiatric disturbances.

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Prompt Publication Policy

The *Journal* would like to remind authors who are particularly interested in early publication (whether to establish priority of an idea, reveal innovative results of new research, or for other pressing reasons) of its prompt publication policy. Under this policy, short (maximum of six double-spaced pages) manuscripts will be considered for publication in the Brief Communications section of the first available issue. These manuscripts must still undergo expert scrutiny before being accepted, and two months must be allowed for the printing process. However, the time required for publication of these short articles can be lessened considerably.

Authors who would like to have their manuscripts considered under this policy should indicate this desire, as well as the reasons for it, when they submit their manuscripts.

Introduction

BY RICHARD JED WYATT, M.D.

SINCE THE SCHIZOPHRENIC syndrome was first described around the turn of the century, there have been many false starts at defining its symptoms, course, and etiology, as well as determining the best way to treat it. While most observers believe the syndrome to be real, extremely painful, and debilitating, our knowledge has not advanced proportionately to the efforts we have made. The Symposium on Current Concepts of Schizophrenia held at the 128th annual meeting of the American Psychiatric Association was designed to present reviews of some of the many current areas of investigation in schizophrenia.

My reaction to the symposium was that, although we do not yet have the answers, our techniques, understanding of experimental controls, organization of research, and general objectivity are advancing rapidly.

Dr. Carpenter describes a diagnostic method that may eventually be used internationally. The method is based largely on current symptoms and signs, but he suggests that longitudinal information is also needed for more rigor. Dr. Kohn's review of the literature on social class indicates that schizophrenia occurs most frequently in the lowest social classes, a finding that can probably be considered a fact at this time. The reasons for this relationship are not yet known definitively; however, Dr. Kohn presents interesting hypotheses.

Dr. Reiss' review of family studies leads him to conclude that there are abnormalities in the interaction of families of schizophrenics and that these abnormalities may precede the onset of the disorder. While it is not yet clear what role these abnormalities play in the etiology of the illness, Dr. Reiss proposes methods for examining this role and recommends rapprochement and joint endeavor as an alternative to the relatively entrenched schism among the social, psychological, and biologic approaches to the study of schizophrenia.

Perhaps the major research advances have come from adoption studies, which demonstrate the importance of genetic factors in the etiology of schizophrenia. Drs. Matthysse and Kidd use mathematical models to assess the magnitude of the genetic contribution—a question that the studies of adoptees and twins have not answered. They conclude that the figure may exceed 47%, a percentage derived from the concordance rate for monozygotic schizophrenic twins.

Dr. Meltzer imaginatively and diligently follows up an observation that creatine phosphokinase (CPK) is transiently elevated in the serum of acutely disturbed psychotic patients. While intramuscular drug injections and nonspecific factors such as activity or stress may account for some of the increases, they cannot account for the majority. The functional significance of this finding, particularly in light of the fact that increased CPK activity is present in both schizophrenia and the affective disorders, is not known. However, Dr. Meltzer's work demonstrates a systematic methodology for examining such an observation.

Dr. Snyder reviews the most exciting current hypothesis of schizophrenia: that the schizophrenic behaves as if there were an excess of dopamine in the brain. The evidence is circumstantial at this point, but information generated from investigations in this area is rapidly expanding. Drs. Gillin, Kaplan, Stillman, and Wyatt review criteria by which biochemical hypotheses of schizophrenia can be tested. To illustrate the application of these criteria, they examine the short-acting hallucinogen, *N,N*-dimethyltryptamine (DMT), which meets some but not all of the investigational criteria. Finally, Dr. Davis, who frequently uses the library as his laboratory, provides us with some new ways to look at the neuroleptic drugs.

Of course, there are other important areas of recent investigation of schizophrenia that could not, because of space limitations, be represented in this symposium. There has been much research on the level of the

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Dr. Wyatt is Acting Chief, Laboratory of Clinical Psychopharmacology, Bldg. 10, Rm. 4N206, National Institute of Mental Health, 9000 Rockville Pike, Bethesda, Md. 20014.

enzyme monoamine oxidase in the platelets of chronic schizophrenic patients (1–4), the controversy over megavitamin therapy has received considerable attention (5–8), and there have been investigations of disordered cognition and perceptual defects in schizophrenics, including studies of visual scanning differences between schizophrenics and normal individuals (9, 10). Abnormalities in reaction time (11) and histamine metabolism (12) are not being as actively examined now as they have been in the past, but they remain fairly secure associated abnormalities of the disorder.

The statistical, psychological, physiologic, and biochemical tools for understanding schizophrenia may be in our hands, but we will know this only in retrospect. It is clear from the papers in this section, however, that these tools are becoming extremely powerful—a final example is the incredibly increased capabilities of such research methods as mass spectrometry. In the meantime, in order to be honest, we must say to our patients and our readers, "We don't know yet—but we *are* working on it."

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Current Diagnostic Concepts in Schizophrenia

BY WILLIAM T. CARPENTER, JR., M.D.

The diagnosis of psychiatric disorders currently relies primarily on clinical observation and evaluation of manifest psychopathology. Symptom-based diagnosis of schizophrenia defines a highly variable patient population and thus makes replication of research results difficult. However, this diagnostic approach is useful because it has established communicative value, encourages clinicians to rely on the diagnostic data most readily available (i.e., the patient's behavior), and relates directly to treatment plans. The author describes a 12-point symptom-based diagnostic system for schizophrenia that enhances the reliability and (presumably) validity of the diagnostic process. He then illustrates methods that can be used to select homogeneous schizophrenic populations for specialized research purposes.

FOCUSING ON current diagnostic concepts of schizophrenia is both simple and complex. It is simple because nosologic concepts of the functional psychoses have undergone relatively modest alterations over the past few decades. We now, as always, rely heavily on manifest psychopathology (catalogued as signs and symptoms) for diagnosis. The diagnostic process is built on clinical observation—the foundation of scientific medicine.

The weakness of present techniques for identifying schizophrenic patients is several-fold and relates to validity, reliability, and the meaning of manifest psycho-

Dr. Carpenter is Director, Schizophrenia Research Programs, Department of Psychiatry, Albert Einstein College of Medicine, Bronx Municipal Hospital, Eastchester Road and Pelham Parkway, Bronx, N.Y. 10461.

pathology. In these areas matters become complex. To date we have had few validating criteria for the diagnosis of schizophrenia (1–3), and those validity criteria presently applicable would fail to identify many patients diagnosed schizophrenic, even when stringent criteria are used for the diagnosis (1, 2, 4). Poor diagnostic reliability has also often been demonstrated (5–11).

The most critical limitation in using manifest psychopathology to identify schizophrenic patients, however, is that symptoms are not the disease itself. We know that some characteristic symptoms of schizophrenia may be highly discriminating, but they are not pathognomonic. Furthermore, aspects of schizophrenia have been observed in nonschizophrenic persons with organic psychosyndromes, including those induced by drugs. That symptoms are not the disease itself is, of course, a truism. It is important to note only because the etiology and pathogenesis of schizophrenia are largely unknown, and ancillary diagnostic tools such as laboratory tests are not yet available. Since nosology and treatment of schizophrenia relate so directly to the patient's manifest behavior, and since the search for etiological clues often focuses on variables that alter (by either inducing or reducing) symptoms, it is worth keeping in mind that a patient's accusing hallucinations may relate to schizophrenia the way a tubercular patient's cough relates to his disease. Schizophrenia is an imperfectly defined syndrome that encompasses patients with different etiologies, divergent pathogeneses, various treatment requirements, and heterogeneous outcomes.

Despite these problems and others (e.g., the negative attributes of labeling a person schizophrenic [12–17]), I believe there is ample evidence that the inclusion of schizophrenia in our nosology carries far more useful information than misinformation. The other papers in this section, for example, document important associations between a schizophrenic diagnosis and communication styles in families, status in society, family history of schizophrenia, some biologic variables, and pharmacologic responsivity.

SIGNS AND SYMPTOMS AS DIAGNOSTIC CRITERIA

Recent methodologic advances have encouraged a fresh look at the symptom criteria for schizophrenia. These advances can be simply stated. 1) Structured clinical interview techniques can now be used to reliably and systematically assess manifest psychopathology in large numbers of patients (18–20). 2) Computer and mathematical techniques enhance the identification of critical diagnostic variables. Analysis of variance, discriminant function analysis, factor analysis, cluster analysis, and other programs can be applied to large bodies of data to evaluate old and seek new diagnostic information. 3) Factors such as differential psychopharmacology, family history of major psychoses, and platelet monoamine oxidase activity have emerged as potential validating criteria for the diagnosis of schizophrenia.

Rather than review the current literature, I will illustrate the application of these methodologic advances in one study (21) that was conducted under the auspices of the International Pilot Study of Schizophrenia (IPSS). The choice of this study is justified by several attributes of the IPSS—large sample size, systematic and reliable data collection, and use of patients and psychiatrists with diverse backgrounds.

The IPSS is a transcultural psychiatric investigation of 1,202 patients in nine countries: China, Colombia, Czechoslovakia, Denmark, India, Nigeria, the Union of Soviet Socialist Republics, the United Kingdom, and the United States. It was designed as a pilot study to lay scientific groundwork for future international epidemiologic studies of schizophrenia and other psychiatric disorders. Details of the methodology employed in this investigation are provided elsewhere (20).

The material to be reviewed relates to a specific question: what signs and symptoms can best distinguish schizophrenic patients from other severely disturbed psychiatric patients? For practical clinical and research purposes, operational criteria for diagnosing schizophrenia are essential. Schneider's diagnostic model is a suitable paradigm (22, 23). Following this model, we approached our data atheoretically (with no etiological assumption). We wished to find highly discriminating symptoms of schizophrenia that occurred frequently enough to have practical application and that could be reliably assessed by observing clinicians. Such symptoms, taken together with other relevant diagnostic data (e.g., history), could provide a basis for reliable and replicable diagnosis.

The Present State Exam (PSE) was used by investigators in each of nine centers to rate signs and symptoms in recently admitted patients with evidence of psychosis. Patients with organic psychosyndromes and those who were chronically institutionalized were excluded. This study design selected 1,202 patients, 811 of whom were diagnosed as schizophrenic by the interviewing psychiatrist, based on manifest psychopathology during the past month (Present State Exam), history and social descriptive data obtained using other research instruments (20), and any other information available to the psychiatrist. Eighty-three of the 1,202 patients (6%) were dropped because they could not be clearly assigned to either the schizophrenic or nonschizophrenic group (e.g., those with paranoid states). The remaining 1,119 patients were randomized into two cohorts, each containing about 405 schizophrenic and 155 nonschizophrenic patients. Cohort B was omitted from the initial data analysis and held in reserve to replicate or refute findings in cohort A. The 230 symptoms and 130 signs of the PSE were arranged as 443 overlapping variables, including single items and psychopathologic dimensions formed by combining related signs and symptoms.

These methods illustrate one recent advance in study techniques—systematic and reliable collection of behavioral data for substantial numbers of selected patients. A second advanced technique used in the IPSS study was the application of mathematical techniques with computer assistance. We have reported elsewhere the results of evaluating several established and experimental diagnostic systems (24–26). I will review only the system that most effectively separated diagnosed schizophrenic patients from nonschizophrenic patients in cohort A and was replicated in cohort B.

An analysis of variance (ANOVA) program was applied to the 443 variables, yielding the discriminating power of each variable taken alone. Using the ANOVA results as a guide, the less discriminating variables in groups of obviously overlapping variables were eliminated. This resulted in 69 significant discriminating variables ($p<.01$ for each). Data from the two groups of patients (schizophrenic and nonschizophrenic) were then subjected to a stepwise discriminate function analysis based on these 69 variables. The first step in the discriminate function run showed diagnostic assignments based on the single most discriminating symptom from the ANOVA (restricted affect). The second symptom added was chosen for its power to increase discrimination between the schizophrenic and nonschizophrenic groups, given knowledge of the first symptom. Thus, symptoms were added stepwise by taking into account the symptom information already included. Appendix 1 lists the 12 most discriminating symptoms, ranked in order of their appearance on the stepwise discriminate function program, as well as PSE items associated with each symptom and their reliability.

This 12-point system is designed to be applied as a unit. The 9 signs and symptoms more prevalent in schizophrenia are scored 1 point each if present, while the 3 symptoms—waking early, depressed facies, and elation—more prevalent in the nonschizophrenic group are scored 1 point each when absent. Thus, the most stringent criteria for a diagnosis of schizophrenia would be a total score of 12 (i.e., 9 schizophrenic symptoms present and 3 nonschizophrenic symptoms absent).

The effectiveness of this system is shown by table 1. A meaningful split between schizophrenic and nonschizophrenic diagnostic assignments results when one determines that at least 5 points are present. A substantial number of diagnosed schizophrenic patients would be judged schizophrenic and relatively few of the patients assigned nonschizophrenic diagnoses would be relegated to the schizophrenic group by these criteria. The percentages in the top (schizophrenic) row can guide the clinician as to the applicability of this system to a schizophrenic population. For example, he might expect 80% of acute and subacute schizophrenic patients to have at least 5 of these symptoms. In the second (nonschizophrenic) row, the percentages indicate the degree of error one might anticipate when making diagnostic assignments with this system. For example, if a patient with 5 or more of these symptoms is assigned automatically to a schizophrenic category, one would expect about 13% of diagnosed nonschizophrenic patients to be assigned to schizophrenic groups.

TABLE 1
Results of a 12-Point System Used to Differentiate Schizophrenic and Nonschizophrenic Patients

Percent of Patients	Number of Points		
	5 or More	6 or More	7 or More
Cohort A			
Schizophrenic patients (N=407)	80	66	44
Nonschizophrenic patients (N=152)	13	4	1
Cohort B			
Schizophrenic patients (N=404)	81	63	39
Nonschizophrenic patients (N=156)	22	6	1

This 12-point system was derived from cohort A and thus worked well with those patients. The results in cohort B (table 1) represented a replication study, since these patients did not contribute to the derivation of the system. A chi-square comparison of the results in cohorts A and B revealed no difference ($p<.4$), and examination of table 1 indicates similar effectiveness of the 12-point system in both cohorts.

This 12-point symptom-based diagnostic system can be used at different levels of stringency, for each of which the number of false-positive inclusions can be estimated. The system is based on reliable data, its effectiveness has been replicated, and it is sufficiently brief to be readily taught and applied.[1]

BEYOND SIGNS AND SYMPTOMS

The 12-point system for identifying schizophrenic patients presented above has considerable potential for increasing diagnostic reliability. Even so, there is little reason to expect a homogeneous patient group to emerge from its application, regardless of how stringent the symptom criteria, because we have little reason to think that schizophrenia, as presently defined, is a single illness. The evidence for this can be readily surmised from reports dealing with genetic and biologic factors in schizophrenia (1, 2, 4, 28–30). There are

[1]Methodologic details and a more complete discussion of this system are given elsewhere (21). Also discussed elsewhere are the questions why 12 symptoms are better than 11 and as good as 13 or 14, and how this system compares with other systems. An evaluation of extant subtypes of schizophrenia and a proposal for new subgroups are presented in an article now in press (27).

thus a number of theoretical and practical limitations to the use of symptom criteria for schizophrenic diagnois. Symptoms are not etiologic indicators (i.e., if substance A induces symptom remission, schizophrenia cannot be said to be caused by the insufficiency of substance A), and symptoms do not define a homogeneous population regarding genetics, function, outcome, and treatment.

Strauss (31, 32) has proposed a multi-axial diagnostic model that would include social functioning and course of illness along with symptoms as diagnostic criteria. Each of these three axes would be conceptualized along a continuum. There are a number of other axes that can enter a nosologic scheme, depending on the aims of classification. Some examples of these diagnostic dimensions are: 1) signs and symptoms, 2) course and outcome, 3) social functioning, 4) work functioning, 5) psychiatric history, 6) family history of mental disease, 7) pharmacologic responsivity, 8) biochemical/psychophysiologic variables, and 9) communication patterns in families.

For example, if it is desirable for a diagnosis to predict outcome, then prognostic variables must be added to symptom criteria, since symptoms alone (with the probable exception of defect or negative symptoms [e.g., flat affect]) do not define a homogeneous outcome (33, 34). If diagnostic consistency over time is desired, then psychiatric history variables could be used to exclude patients with previous nonschizophrenic diagnoses. If the aim of diagnosis is to identify schizophrenic patients for a study of psychological factors, it might be desirable to use stringent symptom criteria in combination with evidence for a communication defect in the family and an absence of schizophrenia in biologic relatives.

The immediate practical value of a multidimensional diagnostic approach can be illustrated by its application to investigations of the role of biological factors in schizophrenia. Many investigators are currently studying biological variables in schizophrenia, an area of study greatly reinforced by findings in Danish adoptive studies and by increased knowledge of the mechanism of neuroleptic drug action. Until recently, failure to replicate biologic findings was almost routine (35), and inability to select comparable patients for study in various centers no doubt played a role. Recent biologic work, such as that by Dr. Meltzer in this section, is more satisfying but still far from adequate (30). A major problem resides in the failure to adequately define biologically homogeneous subgroups of schizophrenia, let alone describe patients sufficiently to permit others to select similar patients. This variation in patients can be reduced by applying current knowledge of schizophrenia. An investigator could begin by considering for study patients clinically diagnosed as schizophrenic. This large and heterogeneous population could then be narrowed by applying the 12-point symptom criteria system and including only patients with, for example, 6 or more points. Next, patients with histories suggesting a nonschizophrenic mental illness would be excluded, reducing the number of false positives. Criteria for poor social functioning prior to the current episode of illness can be specified and applied (34, 36) and would result in a patient cohort relatively homogeneous in symptom and social functioning status. This population could be further reduced by requiring a family history of schizophrenia, thus increasing the genetic loading. The final result would be a small but far less variable group of patients suited for biological investigations.

The criteria described above are clinical variables, but laboratory variables may also soon be useful in sorting out subgroups of schizophrenia. For example, sharply reduced platelet monoamine oxidase (MAO) activity may be associated with a genetic vulnerability for chronic schizophrenia (3), but MAO activity appears to be normal in good-prognosis acute schizophrenic patients (4). Holzman has similarly hypothesized that impaired smooth pursuit in eye tracking may separate potentially chronic schizophrenic patients from those with a good prognosis (37).

CONCLUSIONS

Nosology provides a foundation for scientific psychiatry—systems of classification are essential for the acquisition of information and generation of hypotheses. Kraepelin and Bleuler made landmark contributions in defining the diagnostic class now called schizophrenia, but progress in nosology since 1911 has been relatively modest. We continue to identify patients as schizophrenic based primarily on signs and symptoms, but even when well-defined and stringent criteria are used we have a heterogeneous patient population in terms of genetics, biochemistry, psychology, environment, treatment, course, and outcome. This variability results partly from the absence of readily available diagnostic validation (e.g., laboratory tests) and partly because the schizophrenic syndrome is probably comprised of several illnesses not yet teased apart and identified.

Despite this confusing and at times discouraging picture, there is abundant rationale for using sign and symptom criteria in defining schizophrenia. It is traditional and thus has established communicative value. Diagnosing with signs and symptoms uses the backbone of modern medicine—clinical observation; it thus encourages conceptualizing mental illness in terms of the information at hand, i.e., the patient's manifest behavior. Furthermore, symptom-based diagnosis relates directly to the treatment process, since symptoms rather than pathogenesis appear to be the most frequent targets of treatment.

I have reviewed one system that can enhance the identification of schizophrenic patients when used in the clinical diagnostic process. This system, even when its most stringent criteria are applied, will not define a homogeneous clinical entity. Other clinical or laboratory dimensions may, however, be applied to

the diagnostic process to select increasingly homogeneous populations for specialized purposes.

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APPENDIX 1

A 12-Point Diagnostic System

The following items from the Present State Examination (19, 20) correspond to the signs or symptoms of a 12-point diagnostic system for identifying schizophrenic patients. (r=reliability; (–) indicates that absence of the criterion favors a diagnosis of schizophrenia.)

1. *Restricted affect:* Blank, expressionless face (r=.62); Very little or no emotion shown when delusion or normal material is discussed which would usually bring out emotion (r=.63)
2. *Poor insight:* Overall rating of insight (r=.85)
3. *Thoughts aloud:* Do you feel your thoughts are being broadcast, transmitted, so that everyone knows what you are thinking? (r=.95); Do you ever seem to hear your thoughts spoken aloud? (Almost as if someone standing nearby could hear them?) (r=.74)

4. *Waking early* (–): Have you been waking earlier in the morning and remaining awake? (rated positive if one to three hours earlier than usual) (r=.83)
5. *Poor rapport:* Did the interviewer find it possible to establish good rapport with patient? (r=.86); other difficulties in rapport (r=.75)
6. *Depressed facies* (–): Facial expression sad, depressed (r=.73)
7. *Elation* (–): Elated, joyous mood (r=.67)
8. *Widespread delusions:* How widespread are patient's delusions? How many areas in patient's life are interpreted delusionally (r=.74)
9. *Incoherent speech:* Free and spontaneous flow of incoherent speech (r=.74)
10. *Unreliable information:* Was the information obtained in this interview credible or not? (r=.73)
11. *Bizarre delusions:* Are the delusions comprehensible? (r=.69)
12. *Nihilistic delusions:* Do you feel that your body is decaying, rotting? (insufficient variability to determine reliability); do you feel that some part of your body is missing, for example, head, brain, or arms? (r=.70); do you ever have the feeling that you do not exist at all, that you are dead, dissolved? (r=.71)

The Interaction of Social Class and Other Factors in the Etiology of Schizophrenia

BY MELVIN L. KOHN, PH.D.

Epidemiological evidence clearly indicates an especially high rate of schizophrenia at the lowest social class levels of urban populations. The author suggests that this relationship between class and schizophrenia exists because the conditions of life experienced by people of lower social class position foster conceptions of social reality that are so limited and rigid as to impair their ability to deal resourcefully with the problematic and the stressful. Such impairment does not in itself result in schizophrenia; however, in conjunction with genetic vulnerability and great stress, it could be disabling.

IN THE LIMITED space available, I shall attempt to summarize the evidence on the aspect of the social environment that has most consistently been shown to be related to schizophrenia—social class. Since this evidence has been thoroughly reviewed in the literature (1–3), I shall not review it here, but instead assess its strengths and weaknesses. I will focus on the questions of how social class enters into the etiological process and how we can develop causal models that take account of the contributions of social class, genetics, stress, and other variables whose pertinence to schizophrenia has not yet been established.

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Dr. Kohn is Chief, Laboratory of Socioenvironmental Studies, National Institute of Mental Health, Bldg. 10, Rm. 3D54, 9000 Rockville Pike, Bethesda, Md. 20014.

This paper is based in substantial part on a more extensive discussion (1) which provides the documentation that is lacking in this brief overview of the issues.

CLASS AND SCHIZOPHRENIA

I will use social class to mean, essentially, position in the hierarchical ordering of society, usually indexed by occupational position or by some combination of occupation and education. There have been more than 50 studies of the relationship between social class and rates of schizophrenia. Almost without exception, these studies have shown that schizophrenia occurs most frequently at the lowest social class levels of urban society. The evidence comes from research conducted in Canada, Denmark, Finland, Great Britain, Norway, Sweden, Taiwan, and the United States—an unusually large number of countries and cultures for establishing the generality of any relationship in social science. Moreover, the exceptions to this established relationship are few and incomplete: they occur in small cities and rural areas or in special subpopulations of larger cities, and none contradicts the larger generalization of an especially high rate of schizophrenia at the lowest social class levels of urban populations. What is not clear is *why* class is related to schizophrenia.

Some argue that the statistical relationship between social class and rates of schizophrenia is only artifactual, the result of methodological error. The princi-

pal issues are the adequacy of indices, the completeness of search procedures, and the relative merits and defects of incidence and prevalence as appropriate statistical measures. Although these issues are not entirely resolved, the very fact that numerous studies using many different research methods have all come to the same conclusion argues strongly that the class-schizophrenia relationship is not just a statistical artifact.

Others accept the statistical relationship but assert that schizophrenics are found disproportionately in lower social classes because of the impairment they have suffered or because of some characterological defect of nonsocial origin. The social status of schizophrenics, from this point of view, tells us something about how they fare in society but little or nothing about what produces schizophrenia.

The evidence on this issue is far from unequivocal, but it seems to me that the bulk of it argues against this hypothesis providing a *sufficient* explanation of the class-schizophrenia relationship. In all probability, families from the lower social classes produce a disproportionate number of schizophrenics, although not by so large a margin as one would conclude from studies that rely on schizophrenics' own occupational attainments.

Still other interpretations emphasize the processes by which people come to be perceived as mentally disordered and what consequently happens to them. One point of view holds that psychiatric and other authorities are especially prone to stigmatize and hospitalize people from the lower social classes. Other explanations focus on the processes by which families, employers, police, and others come to label some deviant behaviors as mentally disordered, thereby setting in motion complex changes in social expectation and self-concept that may result in hospitalization. In this framework, social position matters not because of its role in producing the initially deviant behavior but because it affects other people's perceptions of and reactions to that behavior. The class-schizophrenia relationship thus is seen to document the discriminatory readiness of many people to see signs of mental disorder in lower-class behavior.

There is little solid evidence to support this interpretation; some evidence indicates that this approach is based on assumptions that are inconsistent with what is known. A principal rebuttal stems from the evidence that people strongly resist seeing deviant behavior as mentally disordered, attempting to interpret even grotesque behavior as somehow normal and situationally explainable.

Many interpretations of the epidemiologic data accord primary causal significance to processes that are only tangentially or incidentally related to social class position itself. These interpretations emphasize such processes as social isolation, social integration, discrepancies between aspirations and achievements, and minority position in the community, to name but a few. All of these interpretations are plausible, and most are consistent with existing data. However, there is no reason to favor any of them over the most straightforward interpretation possible, i.e., that social class is related to schizophrenia primarily because the conditions of life built in to lower social class position are conducive to this disorder.

There are several reasons why this might be true: people in lower social classes live in more stressful conditions, they have little money or power to employ in coping with the consequences of stress, there are fewer institutional resources available to them for escaping stressful situations or mitigating the consequences of stress, and lower-class conditions of life limit individuals' internal resources for dealing with stressful or problematic situations. These are not incompatible interpretations; they are all plausible and, as yet, untested. These hypotheses become particularly interesting when examined in conjunction with genetics and stress.

GENETICS OF SCHIZOPHRENIA

Since the evidence for the etiologic role of genetics in schizophrenia is reviewed elsewhere in this section, I will note only that I believe recent studies of monozygotic twins and adopted children demonstrate beyond any reasonable doubt that some genetic mechanism must be involved in the etiology of schizophrenia. One might argue, in fact, that genetics explains the relationship between social class and schizophrenia. If there is a heritable component in schizophrenia, there must have been higher than usual rates of the disorder among the parents and grandparents of schizophrenics. Since schizophrenia is debilitating, there would also have been downward social mobility in these earlier generations. Thus schizophrenics could come disproportionately from families in lower social classes not because the conditions of life they experience have pernicious effects, but rather because there is a concentration of genetically susceptible people in the lower social classes.

That such processes occur is indisputable, but a detailed analysis of rates of intergenerational occupational mobility of schizophrenics makes it seem highly unlikely that class differences in the incidence of schizophrenia result entirely—or even largely—from genetically induced intergenerational social mobility. Thus genetics cannot totally explain the class-schizophrenia relationship.

STRESS AND SCHIZOPHRENIA

The evidence for the role of stress in the etiology of schizophrenia is much less clear-cut than that for social class or genetics, but it is sufficient to establish a prima facie case. Although I am aware of no definitive study in this area, there are pertinent studies that indicate an association between stress and the occurrence of schizophrenia. One important study (4) in-

dicated that the relationship between stress and schizophrenia does not simply reflect the high levels of stress prevalent in the lower social classes. This investigation of schizophrenics and matched controls in the lowest social class of San Juan, Puerto Rico, showed that during the year before the onset of symptoms, the schizophrenics experienced notably greater stress than the controls. Even when judged by the harsh standards of life of the San Juan slums, the stresses that preceded the onset of schizophrenia were unusually severe.

As with genetics, we must ask whether stress explains the relationship of class to schizophrenia. Unfortunately, the only study that has provided data for all levels of social class assessed mental disorder in general rather than schizophrenia in particular (5). This study is nevertheless germane because it showed that at any given level of stress, people of lower social class position are more likely to become mentally disturbed than are people of higher social class position. In fact, the more sources of stress there are, the greater the class difference in the proportion of people who manifest psychotic symptoms. The implication is that the relationship of class to mental disorder (and, if we may extrapolate, to schizophrenia) is not attributable to the amount of stress that people endure. There must also be important class differences in how effectively people deal with stress.

THE INTERACTION OF GENETICS, STRESS, AND SOCIAL CLASS

On the basis of the discussion thus far, we can summarize three essential facts about the current state of our knowledge of the epidemiology of schizophrenia:

1. There is abundant evidence that social class is related to schizophrenia but considerable uncertainty as to why.
2. There is abundant evidence that genetics is involved in the etiology of schizophrenia but much uncertainty as to how.
3. There is enough evidence to make it seem likely that stress is also involved, although we are not yet certain of this.

An implicit but certainly important observation is that none of these three factors can possibly account for schizophrenia except in combination with one or both of the other two and perhaps other, as yet unknown, factors.

The evidence that none of these factors alone can account for schizophrenia is clear and results from the research in each individual area. The incidence of schizophrenia in the lowest social class, although higher than that in other social classes, is low by any absolute standard, certainly less than 10% no matter what measurements are used. Similarly, the concordance rate for monozygotic twins of schizophrenics, although higher than that for dizygotic twins and for siblings, is much less than 100% and probably less than 50% in the more carefully executed studies. Also, the incidence of schizophrenia among people subjected to even the most heavy bombardment of stress in their daily lives is probably not very high by absolute standards, although in this case the evidence is too limited to estimate absolute magnitude.

These facts argue that the explanation of schizophrenia lies in the interaction—the joint occurrence—of genetic predisposition, stress, and the conditions of life associated with social class. I think it would be profitable for us to begin to develop theoretical models that attempt to take account of such interactions.

A PROPOSED INTERACTIVE MODEL

I have designed a formulation that I chose because I think it is plausible, potentially testable, and offers a good illustration of the type of interpretive model that we should begin to develop.

I suggest that the constricted conditions of life experienced by people of lower social class position foster conceptions of social reality that are so limited and so rigid as to impair their ability to deal resourcefully with the problematic and the stressful. Although such impairment is unfortunate, it would not in and of itself result in schizophrenia. However, in conjunction with a genetic vulnerability and the experience of great stress, such impairment *could* be disabling and result in schizophrenia.

This formulation is admittedly speculative. However, it is a direct extrapolation from what is known about the relationship between social class and conceptions of reality (6). The lower an individual's social class position, the more likely he is to value conformity to external authority and to believe that such conformity is all that his own capacities and the exigencies of the world allow. In particular, the lower a person's social class position, the more likely his orientational system is to be characterized by a rigidly conservative view of man and his social institutions, by fearfulness and distrust, and by a fatalistic belief that he is at the mercy of forces beyond his control and often beyond his understanding.

The existence of these class differences in beliefs and values is hardly accidental, nor is it cultural in the sense employed by "culture of poverty" theorists, who believe that lower-class orientations are handed down from generation to generation independently of current social conditions. In actuality, social class embodies such basic differences in conditions of life that subjective reality is necessarily different for people differentially situated in the social hierarchy. Lower-class conditions of life allow little freedom of action and give individuals little reason to feel they control their fate. To be in a low social class is to be insufficiently educated and to work at a job of little substantive complexity under close supervision, with little possibility of varying the routine flow of work. These are precisely the conditions that narrow one's concep-

tion of social reality and reduce one's sense of personal efficacy.

Thus there is ample evidence that class differences in conditions of life produce differences in conceptions of social reality. But do these differences in orientation contribute to the class-schizophrenia relationship? There are three reasons for thinking that they might.

1. Instead of searching aimlessly among the innumerable correlates of social class for one that might help explain its relationship to schizophrenia, I think it is strategic to look to something that is fundamental to the social psychology of class: that members of different social classes, by virtue of enjoying (or suffering) different conditions of life, come to see the world differently—to develop different conceptions of social reality, different conceptions of the desirable, and different aspirations, hopes, and fears. Class differences in orientation are an important bridge between social conditions and psychological functioning.

2. My brief review of the literature on the epidemiology of schizophrenia points to the desirability of considering any factor that might bear on class differences in people's effectiveness in dealing with stressful or problematic situations. It seems to me that the orientational system characteristic of the lower social classes is less adequate for dealing with such situations than that of the higher social classes. Admittedly, the characteristic lower-class orientational system, molded as it is by actual conditions, may often be useful, in that it provides a defensive strategy for people who truly are vulnerable to forces they cannot control. However, there are times when a defensive posture invites attack and the assumption that one is at the mercy of forces beyond one's control—although justified—leaves one all the more at the mercy of these forces. An orientational system predicated on conforming to the dictates of authority results in a perception of social reality that is too simplistic and fearful to allow the individual to take advantage of options that might present themselves. It is too inflexible to permit effective coping with precisely those problematic and stressful circumstances that most require subtlety, flexibility, and a perceptive understanding of larger social complexities.

3. Orientations—conceptions of reality—are a fundamental aspect of schizophrenia: fearful, inflexible reactions to threat are integral to the schizophrenic experience. One reason for the disproportionately high incidence of schizophrenia at lower social class levels may be that schizophrenic disorders build on conceptions of reality firmly grounded in the experiences of these social classes.

My proposed formulation thus attempts to bring genetics, stress, and the conditions of life related to social class position into one coherent interpretation of schizophrenia. The thrust of the argument is that the conditions of life experienced by people of lower social class position tend to impair their ability to deal resourcefully with problematic and stressful situations. This impairment, occurring in conjunction with a genetic vulnerability to schizophrenia and the experience of great stress, could be disabling. Since both genetic vulnerability and stress appear to occur disproportionately at lower social class levels, people in these segments of society may be in triple jeopardy.

Whether or not this particular formulation will prove to be the key to the puzzle of schizophrenia, it does illustrate a type of interpretive model more adequate to the complexities of the current and rapidly accumulating evidence than the earlier models. These models tried, unsuccessfully, to attribute schizophrenia to a single causal variable—genetics, family relationships, social class, stress, etc. An interactive model of the type I have proposed can deal with the evidence that no one factor explains schizophrenia and that some combination of relevant conditions must be present to cause schizophrenia.

Testing any theoretical model based on the hypothesis of interactive relationships among causal variables requires a different mode of research from that ordinarily used in studying schizophrenia. This mode should include the study of genetic, psychological, and social factors in the same research designs. Although this will not be easy, it is counterproductive to continue to examine social class, stress, and genetics in separate studies as if they were additive or as if any one of them could in itself explain schizophrenia.

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The Family and Schizophrenia

BY DAVID REISS, M.D.

The author discusses the role of the family in the etiology of schizophrenia and suggests how this research might be integrated with our growing knowledge of the biology of schizophrenia. Abnormalities of family interaction–particularly communication deviances–are frequently associated with schizophrenia and may play a causal, rather than epiphenomenal, role in its pathogenesis. Communication processes in family life affect the child's development of attentional and perceptual capacities, which also have strong biologic roots. The author suggests that the study of normal and abnormal perceptual development might be a meeting ground for researchers in the areas of the biological and familial theories of schizophrenia.

THE LAST DECADE has been full of exciting advances in the study of schizophrenia, and this special section represents a sampling of these new findings. Although the data are new, this group of papers displays the familiar Cartesian split: two papers represent the Cartesian "mind," now called "psychosocial variables," and most of the other papers represent the "body," now given such labels as "genetic mechanisms," "creatine phosphokinase," and "synaptic transmission." This dualism reflects the structure of our field, which in turn reflects the continuing dualism of our conception of mental illness and human development. Each of us must develop proof of the validity of our variables and their specific role in the etiology of schizophrenia. We have applied to this work a set of increasingly rigorous scientific standards that seem only to have increased the austerity and solitude of our pursuits. Why should we not now enjoy some of the fruits of our work by joining our efforts in an attempt to formulate a unified concept of the evolution of schizophrenia?

In this brief review of the role of the family in the etiology of schizophrenia, I will present first my opinion of how far our field, working in relative isolation from other areas of investigation (chiefly biologic), has advanced in proving the etiologic role of the patient's family and then speculate on how this work on the family might relate to the biologic investigations.

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Dr. Reiss is Professor, Departments of Psychiatry and Behavioral Sciences and of Medicine, George Washington University School of Medicine, 2300 I St., N.W., Washington, D.C. 20037.

RECENT RESEARCH ON FAMILY VARIABLES IN SCHIZOPHRENIA

The role of the family in the etiology of schizophrenia can be assessed by the same general scientific principles as the role of biologic variables. First, the hypothesized variables must be clearly defined and measured by reliable and objective methods. Second, the causal role of the variable must be assessed by demonstrating that it 1) is specifically linked with schizophrenia as opposed to other conditions and states, 2) has an impact on the individual before the onset of schizophrenia, and 3) is not confounded with a covarying or concomitant variable that is the "true" etiologic variable.

I will discuss the relevant work that has been done in each of these areas and the research strategies that have been developed to carry out this work.

Identification and Measurement

The major impetus for family research in the area of schizophrenia was the early work of Theodore Lidz at Yale University, Gregory Bateson at the Mental Research Institute, Palo Alto, Calif., and Lyman Wynne at the National Institute of Mental Health. Mishler and Waxler (1) have critically reviewed these ground-breaking concepts.

Lidz' work stimulated interest in two aspects of relationships between the parents of the schizophrenic—the quality of their emotional ties and the relative power of the parents. Lidz found intense discord and distrust in the marital relations of parents of schizophrenics, particularly females. He also suggested that in the case of male schizophrenics, the father is abnormally submissive and the mother is abnormally dominant. In recent years a variety of techniques has been developed to measure affective quality and power balance in marital relations.

Bateson and Wynne, on the other hand, stimulated interest in communication between parent and child. Bateson noted profound logical inconsistencies in the communications of parents of schizophrenics, and Wynne observed blurring, fragmentation, and disqualification in communication. In response to these early observations and ideas, researchers have developed a variety of measures of clarity of speech, the capacity of family members to understand one another,

and the quantity of information and flexibility in verbal exchanges.

Many of these variables meet scientific standards reasonably well. They are all assessed by direct, quantitative measurement of family interaction, often in well-controlled laboratory settings. A few of these measurements meet quite exacting psychometric standards: they possess concurrent, discriminatory, and predictive validity, as well as adequate interrater reliability and stability over time. Unfortunately, there have also been weaknesses in these variables. Particularly in their application to actual studies, they do not match the sophistication or complexity of the theories from which they are derived. This is particularly true for those measuring conflict, dominance, and affect. A second weakness is that the variables depend too heavily on rater judgments: an independent judge observes a family interacting, listens to a tape, or codes a typescript and then scores the family. These judgments, however reliable, leave too much room for bias and subjectivity. Riskin and Faunce (2) have reviewed problems of validity and reliability in family interaction research methods.

In the past few years there have been some exciting new trends in identifying and measuring family variables that have not yet been systematically applied to schizophrenia. However, I believe they are fully applicable. I will briefly mention two of them. The first trend resulted from the application of ethology and structural linguistics to family life, which has stimulated an interest in the structure of rules each family seems to develop for itself over time. These rules govern the family's relationships with other families and the wider social community. Both clinical and laboratory evidence indicates that families of schizophrenics have peculiar or idiosyncratic relationships with the wider social community. Our laboratory has reported one method for assessing these relationships: repeated measurements of families interacting with one another in a multiple family group (3). A second trend is the increasing recognition that behavior of family members during family interaction is tightly coordinated on a microscopic level. For example, Condon and Ogston (4) observed that during a dinner table conversation the movement of every individual was carefully regulated by the phonemic structure in the speech of the family member who was talking. These and many similar observations suggest a vast unseen continent of family interaction processes completely invisible to the unaided observer and measurable only with precise instruments in laboratory situations. It seems entirely possible that the most devastating and enduring influences of the family may be manifest on this microscopic level of family interaction.

Controlled Study of Family Variables as Etiologic Factors

Specific, concurrent links between family and schizophrenia. Despite substantial advances in the identification and measurement of family variables, their specific links to schizophrenia are modest at best (5, 6). The results in the areas of power, affect, and conflict have been inconsistent. For example, some studies have shown more evidence of abnormal affect in normal families than in families of schizophrenics; others have reported just the reverse. Studies of parental submissiveness have generally been inconclusive. However, studies of communication clarity have yielded consistently reproducible results. More than 20 separate studies in at least 10 different laboratories have demonstrated that communications between parents of schizophrenics and one or another of their children are blurred, poorly understood, inconsistent, poorly focused, wandering, disruptive, and illogical. The difference between families of schizophrenics and those of normal individuals is often large. More important, this abnormality of family communication seems specifically linked to families of schizophrenics and occurs much less frequently, if at all, in families that include individuals with other serious psychiatric disorders such as impulsive character disturbances. These findings seem reproducible in a variety of conditions; for example, several findings from middle class families can be replicated among lower class families, and findings with well siblings repeat those with the identified patient present.

The time of impact of family variables. There are two fundamentally different explanations for the finding that family abnormalities are regularly associated with schizophrenia. The first is that the family abnormalities precede the schizophrenic disorder (and presumably are one of its causes). The second is that the onset of schizophrenia may come first; in that case, abnormal communication might represent the family's response to the disorganizing impact of the schizophrenic member. To my knowledge there have been three approaches to assessing these alternatives: experimental models, follow-back and follow-up studies, and longitudinal studies.

In the experimental model, investigators have tried to shorten drastically the time course of the evolution of schizophrenia. They have introduced experimentally an abnormality in either parental or sibling interaction and observed the impact on the normal children and parents, respectively. For example, Haley (7) exposed normal children to audiotapes of parents of schizophrenic children, and Waxler (8), in a problem-solving experiment, included two groups of pseudo-families (parents and child drawn from different families), one with parents of schizophrenics and a normal child and the other with parents of normal children and a schizophrenic child. In our laboratory, we used secobarbital to induce a temporary thought disturbance in an offspring and observed the impact on family interaction (9).

In the follow-back studies, school or clinic records of schizophrenics from periods before they were overtly ill are compared with similar records from control subjects. Little data on families are available from these studies. In the follow-up study, the 20- to 30-year

old records of contrasting groups of children are located and compared. A search is made for these adults and the follow-up data in terms of psychopathology are correlated with the childhood records (10–12).

In the longitudinal studies (there are two in progress focusing particularly on the family) a high-risk sample of children is identified and followed for some years (6). Family and other variables are measured at regular intervals. These variables are correlated with later pathologic outcome.

The findings from the longitudinal studies are just becoming available. Findings from the laboratory model and follow-up and follow-back studies have been published in the last few years. The combined evidence suggests that a number of the family abnormalities are unlikely to be a consequence of (i.e., occur after) the onset of the schizophrenia. The basic pattern of evidence suggests that family abnormalities exist before the onset of the illness. Not all of these studies examined communication in families. Haley (7) and Reiss (9) studied some of the specific communication variables that seem so promising at present, whereas Ricks (10) and Waring (11) reported the establishment of more global family abnormalities, such as symbiotic relationships and withdrawal of the family from the community in the years before the onset of schizophrenia.

Separation from confounding variables. Even if we can ascertain that a family abnormality (e.g., a communications deficit) precedes the onset of schizophrenia, we cannot say that it is either a necessary or sufficient cause of schizophrenia. It is entirely possible that abnormalities in parental behavior reflect an underlying, preclinical genetic disturbance in which case schizophrenia in the offspring would be the result of genetic transmission. Disturbed communication would in this model be considered epiphenomenal, not causal. In other words, genetic and family variables may be confounded because they covary. Through the years, there have been many attempts to disentangle these two variables. Most scholars in the field agree, however, that this has only been possible in the last 8 years since the publication of the Oregon and Danish adoption studies (13–16.) Most family researchers who have carefully reviewed the data from the earlier studies regard them as strong support for the role of a genetic mechanism. We did not, however, believe they provided strong evidence for or against the necessary or sufficient role of family variables. A more recent report from the Danish adoption studies, specifically investigating family variables, demonstrates a small but distinct role for family variables. Rosenthal and associates (17) found significant correlations between disordered family relationships and pathologic outcome in adoptees whose biologic parents were not schizophrenic. This suggests that family abnormalities may produce a pathologic outcome in the absence of pathogenic genes but may have no appreciable effect when the genes are present. However, this finding is limited by the design of the study, which required the investigator to recreate the early family life of adoptees from their comments and recollections as adults.

Major Research Strategies

Each of the three separate groups of investigators has a very special role to play in the elucidation of the family's role and its relationship to other variables (including the biologic one). The *cross-sectional group* use laboratory and other precision methods for measuring interaction processes in families with a schizophrenic member. They usually conduct measurements during a single time period. Their two distinct tasks are identifying variables that are likely correlates of schizophrenia, which requires the development of careful and comprehensive theory as well as precise and reliable methods, and systematically testing the specific relationship between these variables and schizophrenia. They note distinctions between various subtypes of schizophrenia and contrast family patterns associated with schizophrenia with those associated with other psychiatric syndromes, other nonpsychiatric disabilities, and with those in families whose members are physically and mentally healthy. Their work has yielded and should continue to yield variables specifically linked with schizophrenia or one of its several subtypes.

The investigators who conduct *adoption studies* use two basic approaches: either the parents are directly observed (e.g., Wender [16] compared parents biologically related to a child who becomes schizophrenic, parents raising an adopted child who becomes schizophrenic, and parents raising adopted children who remain healthy), or the adopted children are studied. Rosenthal (17) and Heston and Denney (13) compared children who had different biologic and rearing experiences; for example, the mental health of a group of adoptees whose biologic parents were schizophrenic was compared with a group whose biologic parents were healthy. Recently, a third comparison group of children has been added—adoptees whose biologic parents were healthy but whose adopting parents were schizophrenic (18). In the study of adopted children, the parents are rarely (if ever) available for direct study. Thus the investigator must depend on the adopted offspring (usually an adult at the time of the study) to reconstruct his/her childhood from memory.

Those investigators who conduct *longitudinal studies* follow the course of children and their parents from a period of time before the child became schizophrenic. In order to improve the chances of finding schizophrenics in their sample, these investigators select children at risk (i.e., either their parents are schizophrenic or the children display some suspicious abnormality). These researchers are in a unique situation in that they can determine the time of onset of schizophrenia and observe whether it precedes or follows the family abnormalities. They have the special role of determining how successfully an investigator, by using data available after the disorder has appeared, can reconstruct

the family life of a schizophrenic before the disorder was developed.

There is an important system of exchanges between the three groups of investigators. The consistent correlates found in the cross-sectional studies can be used in the longitudinal and the adoption studies. Because these variables require direct observation of parents, they cannot be extended to the studies of adopted children. However, the longitudinal studies can determine which of the consistent correlates hold up over time and which can be reconstructed by other means (e.g., retrospective accounts by adults of their childhood). These purified variables become most salient for studies of adopted children.

POSSIBLE RELATIONSHIPS BETWEEN FAMILY AND BIOLOGIC VARIABLES

We need some assurance that the purified variables that may emerge from a conglomerate of cross-sectional, longitudinal, and adoption investigations relate in some way to a general conception of schizophrenia that can integrate both biologic and social findings. I shall focus on the communication abnormalities, which have proved to be consistent family correlates of schizophrenia. According to present odds, if you like to wager, these are the family variables most likely to emerge from the multi-design studies as a consistent correlate of schizophrenia, as preceding its onset and as a necessary etiologic agent for at least a subset of the schizophrenias. In relating these findings to biologic studies, I will make one major assumption—that biologic and family abnormalities will be found in the same population of schizophrenics (i.e., that we do not have a situation in which biologic variables, without influence from family variables, produce one subset and family abnormalities, unrelated to biologic factors, produce another quite distinct subset).[1]

Parental communication patterns serve in a very fundamental way to shape and mold the basic perceptual functions of young children. Clear parental communication patterns tend to highlight and amplify the child's stimulus world; they vividly convey the breadth and variety of the parent's experience of his own existential universe (19–21). As research by Mishler and Waxler (22) strongly suggests, flexible communication patterns permit the child to insert his own percepts and observations into the family interaction when he is ready. Ambiguous and inconsistent parental communication patterns blur the stimulus world for the growing child. He is unable to grasp his parents' experience of the world and his own experience is often disqualified and undercut (23). Inflexible communication patterns between his parents may prevent him from presenting his own observations to others or requesting help. We can understand, then, how family interaction patterns—particularly the communication variables so specifically linked with schizophrenia—can have a profound effect on the growing child's capacities and styles for processing stimuli: his attentional capacities and his styles or habits of regulating stimulus intensity and of integrating, differentiating, and giving meaning to stimuli. I emphasize these elementary capacities and styles of stimulus processing because recent evidence has indicated that there may be very specific deficits in these processes that underlie many, if not most, of the cardinal symptoms of schizophrenia (24, 25). Furthermore, these psychological processes have strong biologic roots: they seem to have a genetic component (26), they can be studied with biologic tools such as the average evoked response (27, 28), and they can be altered by psychotropic drugs (29). Thus these elementary psychological processes of stimulus processing are important intervening variables. As we purify family variables to determine their etiologic significance in schizophrenia, we should pay special attention to their influence on these elementary perceptual processes. Biologic investigators may benefit from the same strategy; they can examine the relationship between measurements of enzymes, synaptic physiology, and neuropharmacology on the one hand with assessments of elementary perceptual processes on the other. Wyatt and I have discussed such a strategy in detail elsewhere (30). The fundamental point is that the Cartesian split is poor scientific strategy. "Mind" and "body" researchers need to join forces and find variables both can love. Schizophrenia may be conceptualized as a special form of human development shaped by deficits or distortions of specific and elementary psychologic variables that (according to present evidence) are vulnerable to both social and biologic influence. The perceptual variables that I and others have emphasized may not be the final meeting ground for psychosocial and biologic investigators, but they do permit a strategy worth pursuing.

[1]Rosenthal's recent research (17) does not, in my view, seriously challenge this assumption because of the methodologic limitations of his study.

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Estimating the Genetic Contribution to Schizophrenia

BY STEVEN W. MATTHYSSE, PH.D., AND KENNETH K. KIDD, PH.D.

The authors discuss the limitations of the concept of heritability in schizophrenia and, using reported incidence figures, assess two alternatives—the multifactorial and single major locus (SML) models. Both models predict genetic heterogeneity in schizophrenia. For some parameter values, the SML model indicates that homozygotes are rare but at very high risk. According to the multifactorial model, 9.1% of the schizophrenic population has a genetic risk of 99% or more. The authors conclude that neither model adequately accounts for the data but that predictions from both can be used to design sampling procedures which will increase the probability of selecting for study individuals whose illness is highly genetically determined.

STRONG EVIDENCE for genetic factors in the etiology of schizophrenia has been provided by adoption studies (1), but the mode of transmission and the extent of genetic determination are still unknown. Since not all identical twins of schizophrenics are also schizophre-

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Dr. Matthysse is Assistant Professor of Psychobiology, Department of Psychiatry, Massachusetts General Hospital, Boston, Mass. 02114. Dr. Kidd is Assistant Professor, Department of Human Genetics, Yale University School of Medicine, New Haven, Conn.

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nic, nongenetic events must have some influence; however, it is not known whether these events are pre- or postnatal, psychosocial or physical.

The concept of "heritability" has often been used to estimate the magnitude of the genetic contribution, but this term has semantic and mathematical ambiguities. Although the dictionary definition of heritable as "capable of being inherited" is unobjectionable, the geneticist uses a different and more precise definition. First, the total variation of a trait within a population (measured as the variance) is divided into genetic and environmental components and a component representing their interaction. The genetic component is then further divided into "additive" and "dominance" effects. The distinction between the latter components is best understood by recalling that each individual has two alleles (genes) at each locus, one from each parent. If one form of the gene is pathogenic and the other is not, an individual can be genetically normal at the locus, heterozygous (single dose of the pathogenic allele), or homozygous (double dose). If the effect of a single dose is exactly half that of a double dose, the allele is said to be additive. Otherwise, there will be dominance variation as well as additive variation. Thus heritability in the genetic definition (symbolized by h^2) is the proportion of a trait's variation in the population that is due to additive genetic variation among individuals.

LIMITATIONS OF THE CONCEPT OF HERITABILITY

In its precise form, the heritability concept has several limitations.

First, the heritability of a trait depends on the range of relevant environmental variation in the population being studied. If the population is subjected to a wider range of environments, heritability will decrease; conversely, if the relevant aspects of the environment are held constant, heritability will increase. Furthermore, it frequently happens that the range of environments in the sample studied is not representative of the general population, so the heritability estimate cannot be safely extended to the population as a whole. In fact, drug therapy and psychotherapy can be considered as novel environments so that, logically speaking, one would have to conclude that the heritability of schizophrenia changed when these modalities were introduced.

A second limitation is that heritability requires quantitative definition of a trait. When there are several different ways of defining a trait (as is always the case in psychiatry), there will be several different heritability estimates. Gottesman and Shields (2) first applied the multifactorial model of Falconer (3) to schizophrenia and obtained a heritability estimate of about 80%. The attribute to which this heritability applies is not actually a measured trait; it is a hypothetical underlying, continuous, and normally distributed variable called "liability." One can also calculate heritability for schizophrenia defined as an all-or-none trait (i.e., arbitrary discrete scores for normal and affected individuals). With this definition of the attribute, heritability would be placed at 20%–40% for a single-gene model. It is also possible to construct a single-gene model with an underlying continuous liability that is not normally distributed (4, 5); heritability of the liability scores is in the range of 0%–15%. Thus, three different definitions of the trait result in three different heritabilities.

The third problem is that in order to calculate heritability, it is necessary to assume that genetic and environmental components contribute additively to the trait, that they are not correlated with each other (6, 7), and that the rearing environment is not dependent on whether the parents have the trait. When there is "cultural transmission," as in learning a language, the last assumption certainly breaks down.

In summary, heritability does not provide information on etiology, is not able to predict response to novel environments, and cannot be unambiguously estimated from data. Therefore, it is our conclusion that heritability of schizophrenia is not a meaningful concept or statistic, even though the disease has a clear genetic component.

THE SINGLE MAJOR LOCUS AND MULTIFACTORIAL MODELS

A more satisfactory approach to estimating the genetic contribution to schizophrenia begins by adopting a specific model of transmission. After fitting the parameters of the model to the data as well as is possible, the relative contribution of genetic factors is estimated within the context of the model. The major limitation of this approach is that we do not yet have enough information to decide among the alternative models. Nevertheless, one can select two extreme genetic models and estimate the genetic contribution in terms of each of them.

The single major locus (SML) and multifactorial models are among the simplest that incorporate both genetic and environmental factors; they are both frequently encountered in the study of human genetics. These models represent the genetic extremes of "monogenic" and "polygenic" inheritance. It is known that nongenetic factors can mimic genetic and even Mendelian inheritance (8). Such influences as children's learning a trait from their parents or from imitation of their siblings (e.g., language and dietary preferences), are called "cultural inheritance." Neither model specifically incorporates cultural transmission. Although the multifactorial model would allow culturally transmitted factors, they would not be separable from the genetic factors. Thus environmental influences are assumed to be randomly distributed for both models. This assumption may not be too restrictive, since there is no compelling evidence for cultural transmission in schizophrenia (9).

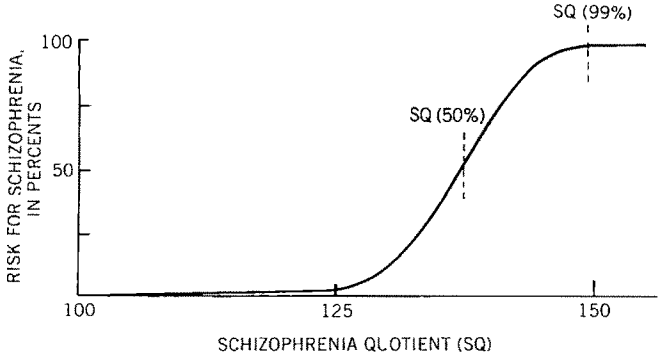
The SML model has four unknown parameters that must be estimated from the data (10): 1) q, the fre-

quency of the pathogenic allele (the form of the gene that increases the risk for schizophrenia) in the general population; 2) f_0, the probability that an individual who is genetically normal nevertheless becomes schizophrenic at some time in his life (these individuals are called "phenocopies" because they have the phenotype without the genotype); 3) f_1, the probability that an individual with one pathogenic allele at the "schizophrenia locus" and one normal allele (a heterozygote) develops the disorder; and 4) f_2, the probability that an individual with two pathogenic alleles at this locus (a homozygote) becomes schizophrenic. The parameters f_1 and f_2 are sometimes called the penetrances of the allele in the heterozygous and homozygous state. When $f_1 = f_0$, the allele only has an influence when it is present in double dose, and the trait is said to be recessive. When $f_1 = f_2$, the heterozygote has just as high a risk as the homozygote, and the trait is said to be dominant. Intermediate cases in which $f_0 < f_1 < f_2$ are also possible; in such situations the terms "dominant" and "recessive" are not applicable and can be misleading.

The multifactorial model represents an opposite extreme. Genetic vulnerability is assumed to be caused by the action of a large number of independent genes; no one genetic factor predominates, but each contributes a small amount to the risk. Intelligence is thought to be a trait of this kind, and the analogy with IQ can be very helpful. The IQ score is derived by scaling test results so that the scores will have a normal (i.e., bell-shaped) distribution in the general population, with a mean of 100 and standard deviation of 15. We can postulate the same kind of scale for vulnerability to schizophrenia (although we do not have a test to measure it) and call it the schizophrenia quotient (SQ). SQ will also be scaled to have a normal distribution in the general population, with a mean of 100 and standard deviation of 15. For each value of SQ, there is a certain lifetime risk for schizophrenia. SQs well below 100 are associated with a risk near 0, while SQs well above 100 are associated with a risk near 100%. Continuing the analogy with IQ, the risk is like the probability that an individual with a certain IQ will graduate from college. The normal distribution curve for SQ in the population is not an assumption—like IQ, the variable is scaled in such a way that it has to be normal—but the shape of the risk function is an assumption. In the multifactorial model the postulated risk function is a cumulative normal (or sigmoid) curve (11) as illustrated in figure 1. The sigmoid shape for the risk function is plausible, but there is no special empirical justification for it; it is really chosen for mathematical convenience. There are two unknown parameters in the cumulative normal risk function: its position on the SQ axis and its steepness. These can be determined in several ways, for example, as is indicated in figure 1, by the values of SQ associated with 50% risk and with 99% risk.[1]

It is important to make a distinction between vulnerability to schizophrenia (SQ) and "schizoid personality." A person who is genetically vulnerable to schizophrenia may never develop either schizophrenia or schizoid personality if the environment is favorable. Conversely, some schizoid individuals may maintain their dubious adjustment for a lifetime without becoming schizophrenic. Indeed, Pollin (12) was impressed that the identical twins of schizophrenics who do not themselves have the illness are not rated as more schizoid than individuals not related to a schizophrenic.

FIGURE 1
Risk Function for the Multifactorial Model



ESTIMATION OF PARAMETERS

Having defined the two extreme genetic models, let us try to fit their parameters (q, f_0, f_1, f_2 in the monogenic model; SQ(50%) and SQ(99%) in the polygenic model) to the data on schizophrenia. The data we have available are primarily the frequencies with which schizophrenia occurs among the relatives of a diagnosed schizophrenic. Published reports of these frequencies are unfortunately not fully consistent (4), and the estimates cannot be combined in any rigorous way because the studies were carried out under different conditions using different criteria for schizophrenia. For concordance rates in identical twins (IMZ), we have used an average based on five recent studies (those of Gottesman, Kringlen, Fischer, Tienari, and Pollin) reviewed by Gottesman and Shields (13).[2] The figure for incidence in offspring of dual matings (i.e., two schizophrenic parents), symbolized as I(S × S), is from a personal communication with L. Erlenmeyer-Kimling. The percentages for incidence in offspring (IPO) and incidence in siblings(ISS) are consensus figures from Slater's review of the literature (15); the general population incidence (GI) is a weighted average of studies tabulated by Slater. Our estimates are as fol-

[1]The liability scale of Falconer (3) mentioned in the first section is the sum of SQ and a score representing environmental factors. Falconer's risk function is 0 if this combined score is below a threshold value and 100% if it is above the threshold. Nevertheless, the two models give identical predictions.

[2]All of the monozygotic twin concordance figures are probandwise rather than casewise or pairwise; for a good discussion of the different ways of computing twin concordance rates, see Gottesman and Shields (13) and Smith (14).

lows: IMZ = 47.0%, I(S × S) = 39.0%, ISS = 8.7%, IPO = 12.0%, and GI = 0.87%. In our calculations, we have averaged ISS and IPO, giving an incidence of 10.4%, because the monogenic and polygenic models both require that the risk for offspring not exceed that for siblings. The observed difference is small.

In both the SML and multifactorial models, any three of the incidences presented above are sufficient to determine the other two. In principle, one might choose any combination of three as data for estimating the parameters of the model. Statistical goodness of fit techniques are not applicable because the standard errors of the data are not known. Since ISS, IPO, and GI are based on by far the largest numbers of cases, they will be used as input data for adjusting the parameters of the models.[3]

ANALYSES: SINGLE MAJOR LOCUS MODEL

When ISS=IPO (i.e., siblings and offspring of schizophrenics are equally at risk), it can be shown that $f_1 = \frac{1}{2}(f_0 + f_2)$.[4] In other words, the risk to the heterozygote lies halfway between the risk to the genetically normal individual and the risk to the homozygote. This case is, strictly speaking, an absence of dominance, but it is often called "intermediate dominance" because the gene is neither fully recessive nor fully dominant. Furthermore, when ISS=IPO, the SML model predicts that the incidence in offspring of dual matings will equal the incidence in identical twins, and both can be computed from the formula,

$$\text{IMZ} = \text{I(S} \times \text{S)} = 2(\text{IPO}) - \text{GI}.$$

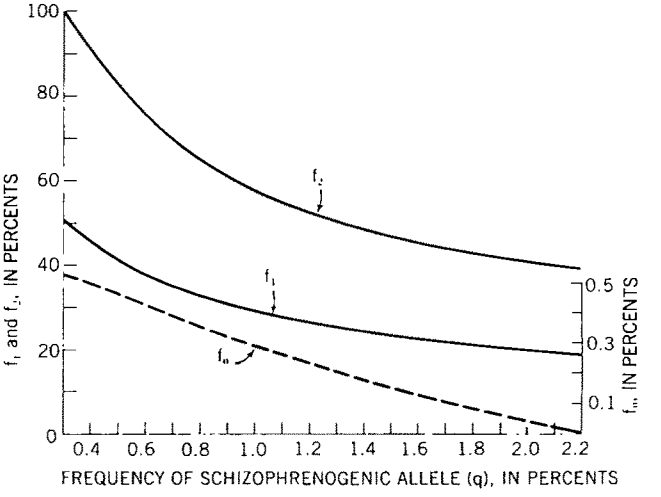
Using the incidence estimates presented earlier, the calculated value for IMZ and I(S × S) is 19.9%. This prediction is too low, unless the observed figures for twins and offspring of dual matings have been very much influenced by special environmental effects.

A wide range of possible parameter sets (q, f_0, f_1, f_2) fit the input data (GI = 0.87%, ISS = IPO = 10.4%) (see figure 2). As mentioned above, all of them predict the same IMZ and I(S × S) (19.9%), so this dilemma cannot be escaped by appropriately choosing the parameter set. The population frequency of the pathogenic allele, q, can vary between 0.3% and 2.2%. As q varies, the 3 f values change accordingly. For the lowest permissible gene frequency, the f values are 0.5%, 50.3%, and 100.0% for genetically normal individuals, heterozygotes, and homozygotes, respectively. For the highest permissible gene frequency, the values are

[3]Other treatments of the data are possible (4) and can lead to numerically different results, although the major qualitative conclusions remain unaltered (5). Because of the heterogeneity of the data, it is difficult to decide which approach is better in a statistical sense, although the values used here are believed to be better estimates for family incidences.

[4]Proofs have been omitted here but will appear in a forthcoming article.

FIGURE 2
Risk Functions for the Single Major Locus Model



0.0%, 19.4%, and 38.9%. All these versions of the model fit the data equally well (i.e., they fit the data for GI, IPO, and ISS exactly, and underestimate IMZ to the same extent). For the lowest gene frequency (0.3%), 100% of the homozygotes are predicted to become schizophrenic, but very few schizophrenics are expected to be homozygotes. Most will be heterozygotes or genetically normal. This calculation will be presented in detail because it gives some insight into the workings of the SML model.

According to the Hardy-Weinberg theorem (for an excellent explanation of this theorem, see reference 16), the expected frequencies of each genetic type in the population as a whole are:

genetically normal	$(1 - q)^2 = 99.3\%$
heterozygotes	$2q(1 - q) = 0.7\%$
homozygotes	$q^2 = 0.001\%$

In order to calculate the breakdown into genetic types for schizophrenics, we employ Bayes' theorem, which relates the probability of event B given event A, P(B|A), to the probability of event A given event B, P(A|B):

$$\text{P(B|A)} = \text{P(A|B)}\frac{\text{P(B)}}{\text{P(A)}}.$$

The theorem is proved by considering the two equivalent expressions for the joint probability of A and B:

$$\text{P(A)P(B|A)} = \text{P(A and B)} = \text{P(B and A)} = \text{P(B)P(A|B)}.$$

Let A stand for being schizophrenic and B for being homozygous for the pathogenic allele. Then, using Bayes' theorem,

$$P(\text{homozygous}|\text{schizophrenic}) = P(\text{schizophrenic}|\text{homozygous}) \times \frac{P(\text{homozygous})}{P(\text{schizophrenic})}.$$

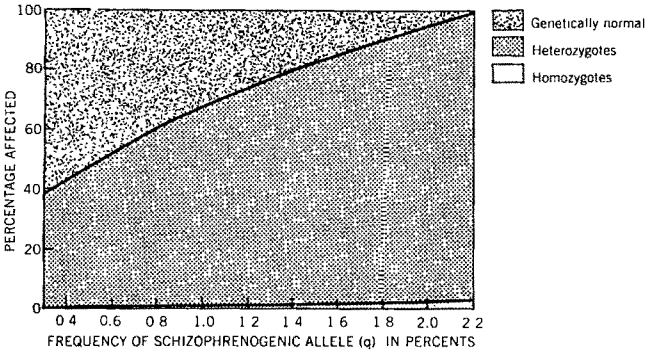
P(schizophrenic|homozygous) = 100% for this gene frequency; P(homozygous) = .001%, and P(schizophrenic) = .87%. Substituting these values into the equation, P(homozygous|schizophrenic) = 100% × $\frac{0.001}{0.87}$ = 0.1%. Let p_0, p_1, and p_2 stand for the probabilities that a schizophrenic is genetically normal, heterozygous, or homozygous, respectively, for the deleterious allele. We have shown that p_2 = 0.1%, and by similar calculation, p_0 = 61.2% and p_1 = 38.7%. We can summarize the p and f values as follows:

genetically normal	$p_0 = 61.2\%$	$f_0 = 0.5\%$
heterozygous	$p_1 = 38.7\%$	$f_1 = 50.3\%$
homozygous	$p_2 = 0.1\%$	$f_2 = 100.0\%$

Since identical twins are of the same genetic type, it is easily seen that the predicted IMZ = $p_0f_0 + p_1f_1 + p_2f_2$ = 19.9%, which is the figure obtained from the earlier calculation.

In figure 3, the values of p_0, p_1, and p_2 are plotted for each gene frequency. For SML models, the proportion of schizophrenics who are homozygous lies between 0.13% (1 in 769) and 2.24% (1 in 45). The probability that identical twins of homozygous schizophrenics are also affected is given by f_2, which ranges from 100% to 38.9%. These concordance rates are estimates of the degree of genetic determination in individuals in whom genes play their full role, namely, those who are homozygous. We see that, according to some versions of the SML model, genes play an extremely important role when they are present in double dose; but such individuals are very rare, even among schizophrenics. According to other versions of the SML model—which fit the data equally well—the genetic contribution is less striking, even in the homozygote. We can conclude only that the data do not rule out a very high degree of genetic determination in homozygotes.

FIGURE 3
Genetic Composition of the Schizophrenic Population



SAMPLING FOR HOMOZYGOSITY

According to all applicable SML models, homozygous individuals are infrequent, even among schizophrenics (not more than 1 in 45). It is conceivable that these rare individuals are precisely the ones who should be studied biologically. The heterozygote may have an enzyme abnormality that is confined to the brain, whereas in the homozygote it is generalized to peripheral tissues as well. Another possibility is that some abnormal biochemical process is limited to the developmental phase in heterozygotes but persists into adulthood in the homozygote. The usual random sample of patients would be expected to contain mostly heterozygotes and nongenetic schizophrenics (phenocopies). If only homozygotes manifest a particular biological abnormality, one could expect to find at most a few deviant values, but there would be no statistically significant difference between the patient group and controls. For example, in a study of blood levels of the enzyme catechol-*O*-methyltransferase in schizophrenics and controls, no statistically significant difference was found, but the highest values were all among the schizophrenics (17). Other interpretations of this result are, of course, possible, but it suggests the value of sampling procedures that would increase the proportion of homozygotes in the sample.

At first glance, such sampling seems difficult to accomplish. Homozygotes are not necessarily more severely affected than heterozygotes; however, on the chance that they might be, it would be reasonable to work with a severely disturbed group. Certain sampling procedures would be expected to increase the proportion of homozygotes, according to the SML model. One could select schizophrenics with adopted-away offspring who also became schizophrenic. Details of the calculation will not be given here, but with this selection procedure, p_2 (the probability of homozygosity) ranges from 0.63% (for q = 0.3%) to 4.27% (for q = 2.2%). This figure is two to five times greater than that for schizophrenics in general, but it is still too low to be of much use. A more effective method is selecting offspring of dual matings (S × S). The dramatic increase in homozygosity in the sample that would result from this method is evident if one considers diseases like Tay-Sachs or phenylketonuria, where there is a probability of 1 in 4 affected (homozygous) offspring when two phenotypically normal heterozygotes mate.

The probability that offspring of dual matings are homozygous can be easily computed using figure 4. The numbers 0,1,2 refer to the number of pathogenic alleles in each parent; p_0, p_1, and p_2 refer to the probability of each genetic type among schizophrenics, as explained earlier. The probability that offspring of dual matings will be homozygous is calculated by multiplying each entry in the figure (probability of the combination of genetic types, given that both parents are schizophrenic) by the corresponding entry in parentheses (probability offspring are homozygous, given the com-

bination of genetic types). The result is,

$$p_2(S \times S) = \tfrac{1}{4}p_1^2 + p_1p_2 + p_2^2,$$

and it is plotted in figure 5 as a function of gene frequency. The value of $p_2(S \times S)$ ranges from 3.8% to 26.1%, 12 to 29 times the value of p_2 for schizophrenics in general. It is possible to go one step further in the sampling procedure. Since many of the offspring of dual matings are raised in foster homes, it is feasible to select dual-mating offspring who become schizophrenic even though they are raised apart from their parents. Applying Bayes' theorem to this group, we have,

$$P(\text{homozygous}|\text{schizophrenic}) = P(\text{schizophrenic}|\text{homozygous}) \times \frac{P(\text{homozygous})}{P(\text{schizophrenic})}.$$

If we call the probability of homozygosity in adopted-away offspring of dual matings who are schizophrenic $p_2(S \times S, S)$, the formula is,

$$p_2(S \times S, S) = \frac{f_2 p_2(S \times S)}{I(S \times S)}.$$

which is also plotted in figure 5. It ranges between 19.0% and 51.0%, an increase in homozygosity over the general schizophrenic population of from 23 to 146 times.

The expected frequency of homozygosity in adopted dual-mating offspring who are schizophrenic is sufficiently high that statistically significant biologic deviations might be found in this special sample, even if they are manifested only in homozygotes. One caution should be noted: the power of the dual-mating sample selection method will be substantially less if there are several genetic subtypes of schizophrenia. For example, if there are three equally common types, the probability of homozygosity for a particular type will fall by a factor of 9.

FIGURE 4
Probability of Homozygosity in Offspring of Dual Matings

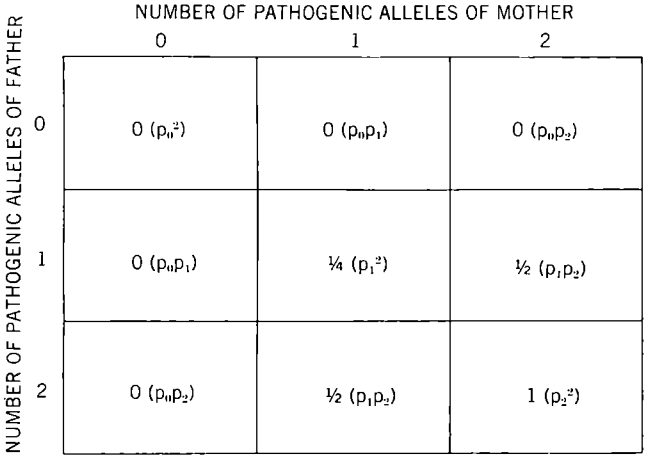
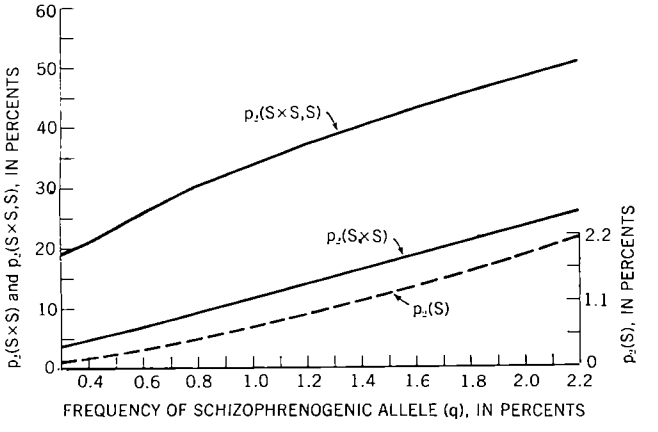


FIGURE 5
Expected Proportion of Homozygotes in Dual Mating Samples



ANALYSES: MULTIFACTORIAL MODEL

In the multifactorial model as we have defined it, there are two unknown parameters that must be adjusted to fit the data: SQ(50% risk) and SQ(99% risk). In order to fit the selected estimates of general incidence (.87%) and incidence in siblings and offspring (10.4%), the SQ values are SQ(50% risk) = 137 and SQ(99% risk) = 148. The analogy with IQ is still more or less applicable, if instead of the risk for schizophrenia we think of the "risk" for graduating from college. An individual with an IQ of 137 would have a 50% probability of graduating; with an IQ of 148, the probability would be 99%. Of course the risk function for college graduation in terms of IQ would have a different shape.[5] The fit of the multifactorial model to the incidence estimates for identical twins and offspring of dual matings is somewhat better than for the SML model, but the errors are in the opposite direction. The predicted IMZ is approximately 61% (actual IMZ = 47%) and the predicted $I(S \times S)$ is approximately 54% (actual = 39%). Since the common environment shared by identical twins and the unfavorable rearing conditions of offspring of dual matings presumably elevate the observed frequencies in those categories, the difference between observed and predicted incidences may be less than our calculations suggest for the SML model and more for the multifactorial model. It is not possible, therefore, to decide between them on the basis of these data.

If an SQ of 148 has a 99% risk, we may ask how frequent those SQs are in the general population. Since SQ has been scaled to be normally distributed with a mean of 100 and a standard deviation of 15, this question can be answered directly by consulting tables of the normal curve: 0.08% of the general population

[5]According to the risk function we have selected, SQ(1%) is 126, so individuals with SQs below 126 are at less than 1% risk for becoming schizophrenic. This includes 96% of the population.

will have an SQ greater than or equal to 148. We can also calculate the proportion of schizophrenics with SQs greater than or equal to 148. To do this, we again apply Bayes' theorem:

$$P(SQ \geq 148 \mid \text{schizophrenic}) = P(\text{schizophrenic} \mid SQ \geq 148) \times \frac{P(SQ \geq 148)}{P(\text{schizophrenic})} =$$

$$\geq 99\% \times \frac{.08}{.87} = 9.1\%.$$

Stated another way, 9.1% of the schizophrenic population (1 out of 11) is predicted to have a genetic vulnerability (SQ) so high that their risk would be 99% or greater were they to start life over again in a new environment. If an individual in this category had an identical twin, the twin's risk would be the same—99% or higher. According to the multifactorial model, therefore, in a substantial proportion of schizophrenics (1 out of 11) the illness is almost completely genetically determined. Sampling methods similar to those described for the SML model can be used to increase the proportion of these individuals in the sample.

CONCLUSIONS

Several conclusions can be drawn from these analyses. First, it seems that neither of these simple models can adequately account for the data. If incidence in the general population and in offspring and siblings of schizophrenics are used as input data, the predicted incidence in identical twins and in offspring of dual matings is too low for the SML model and too high for the multifactorial model. It may be that a model with two interacting genes can account for all five incidences, since the interaction could tend to increase IMZ and I(S × S) relative to the other frequencies.

According to both models, genetic factors may be more important than the observed identical twin concordance rate of 47% might suggest. In the SML model, several sets of parameters (gene frequencies and penetrances) can explain the data equally well—or poorly—and according to some of the acceptable parameter choices, homozygotes are rare but have a very high genetic risk. The multifactorial model predicts that a significant subgroup of the schizophrenic population—1 out of 11—has an extremely high genetic risk, 99% or more.

Finally, it is worth noting that both models predict genetic heterogeneity among schizophrenics. In the SML model, some schizophrenics are homozygous, some are heterozygous, and some are genetically normal. In the multifactorial model, some schizophrenics have a high SQ, and some have a low SQ. Biologic attributes related to the same gene(s) might not, therefore, be found in all schizophrenics. We have suggested some practical ways of increasing the probability of selecting individuals whose illnesses are highly genetically determined, a strategy that might improve the likelihood of discovering biologic differences.

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Serum Creatine Phosphokinase in Schizophrenia

BY HERBERT Y. MELTZER, M.D.

The author notes that increased serum creatine phosphokinase (CPK) activity is found in the majority of hospitalized acutely disturbed schizophrenics and patients with affective psychoses. It is probable that some of these increases do not result from nonspecific factors such as activity, trauma, or stress, which do cause increases in some cases. Those patients who show increased CPK activity have more florid psychopathology and tend to have higher CPK levels in nonacute periods than those without increases. First-degree relatives of psychotic patients with elevated serum CPK have high-normal or slightly increased serum CPK. There is also evidence of other neuromuscular dysfunction in psychotic patients. The author concludes that the investigation of CPK may have considerable heuristic value for the study of schizophrenia and other psychoses.

SERUM OR PLASMA and the formed blood elements have proved to be convenient and safe sources of biologic material for investigators studying biochemical aspects of the schizophrenias and affective psychoses. This article will review one such approach to the biology of schizophrenia—the investigation of serum creatine phosphokinase (CPK) activity. I will focus on seven areas: 1) the origin of the serum enzyme, 2) nonspecific causes for increased CPK activity, 3) clinical characteristics of patients with and without increases, 4) serum CPK activity in nonpsychotic periods in psychotic patients with and without increases, 5) serum CPK activity in first-degree relatives of psychotic patients, 6) comparison with serum CPK increases in patients with acute brain diseases, and 7) other evidence of neuromuscular dysfunction in psychoses.

Increased serum CPK activity has been found in some schizophrenic patients experiencing an acute psychotic phase (1–10). Increases are also present in patients with acute affective psychoses (2, 5, 11). In a recent study of 187 acutely psychotic patients—163 with schizophrenia and 24 with affective psychoses—it was found that 47.1% of the patients (N=88) had increased serum CPK activity in samples obtained within 48 hours of admission (5). Serum CPK levels were then determined each weekday throughout hospitalization, and nearly two-thirds of the patients who showed increases at admission also had one or more increases later in hospitalization. Fifty-four patients who had not shown increased CPK activity at admission did so later during their hospitalization. This indicates that serum CPK determinations must be made throughout hospitalization if all patients with increased CPK activity are to be identified. The increases range from just above the 95% upper limit of normal for each race-sex group up to 50 times the upper limit; the median increase is 2 to 3 times the 95% upper limit. The increase lasts 1 to 3 days in half of the cases but is much longer in others (4, 5).

ORIGIN OF SERUM CPK ACTIVITY

There are now 3 known isozymes of CPK: a brain type (BB), a skeletal muscle type (MM), and a cardiac muscle type (MB). Each type is a dimer made up of some combination of B and M subunits. Brain and skeletal muscle have almost exclusively BB and MM type CPK, respectively; cardiac muscle has a mixture of MB, BB, and MM (12, 13). Other organs have only small amounts of CPK and generally do not contribute to any increase in serum CPK activity. The isozymes of CPK can be identified by chromatographic, electrophoretic, or radioimmunologic methods. The CPK in the serum of schizophrenics has been identified as the skeletal muscle type by chromatographic and electrophoretic methods; no brain type was found (3, 4). This suggests that the origin of the CPK is skeletal muscle, a hypothesis that is further supported by findings of increased serum aldolase (2, 3) and pyruvate kinase (according to the unpublished data of Gordon R. Pschedit and myself) in psychotic patients with increased serum CPK activity. Like CPK, elevated aldolase and pyruvate kinase levels in serum are associated with skeletal muscle disease.

There is some controversy as to whether cerebrospinal fluid CPK activity is increased in acute schizophre-

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Dr. Meltzer is Professor, Department of Psychiatry, Pritzker School of Medicine, University of Chicago, 950 East 59th St., Chicago, Ill. 60637, and Director of Adult Services, Illinois State Psychiatric Institute.

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nia. No increases were found in several studies (4, 14, 15), but there is one report of increased cerebrospinal fluid CPK levels in acute schizophrenics (16) and another of increases in patients with LSD psychoses (17). Further study of this problem with sensitive methods for determination of CPK activity or protein and careful attention to potential artifacts is needed.

Although the evidence I have reviewed may seem to indicate that the origin of the serum CPK in psychotic patients is skeletal muscle, uncertainty has been introduced by a report that brain type CPK is unstable in serum, especially in vivo, with rapid conversion to the cardiac and skeletal muscle types (18). We (Hyong Won Cho, Jong I. Joung, David J. Goode, and myself) have partially confirmed this in work that showed human brain type CPK to be unstable in vitro; after incubation for 42 hours it migrated like the cardiac type and a species intermediate between cardiac and skeletal muscle type developed. We could not identify any skeletal muscle type even after prolonged incubation in vitro; however, it is possible that a species that migrates like skeletal muscle type CPK could be produced in vivo from brain CPK.

FACTORS NOT RELATED TO PSYCHOSIS AS POSSIBLE CAUSES OF ELEVATED SERUM CPK ACTIVITY IN PSYCHOTIC PATIENTS

Intramuscular injections may elevate serum CPK activity. Although less than 50% of patients given intramuscular chlorpromazine (Thorazine) have increased serum CPK levels following injection (19), all patients who have received such injections should be excluded from studies of serum CPK. In order to avoid the necessity of eliminating the majority of psychotic patients whose serum CPK would be of research interest, intramuscular injections should be avoided. Oral psychotropic medication does not influence serum CPK levels in man, and if parenteral medication is needed, subcutaneous prolixin enanthate or intravenous haloperidol might be used. Alcoholism (20) and muscle trauma may also increase serum CPK activity and must be excluded by history, laboratory, and physical examination. Restraining psychotic patients for purposes of management frequently causes sufficient trauma to produce large increases in CPK activity. Dietary factors, including starvation, have not been found to increase serum CPK activity (21).

Stress does not usually increase serum CPK activity in people without psychotic disorders: nonpsychotic psychiatric patients experiencing severe anxiety, agitation, or depressive symptoms do not have increased serum CPK levels (4, 5, 11), and preoperative patients generally have normal serum CPK activity (22). However, we have found moderate increases in serum CPK levels in some psychotic patients before hospital discharge and in several volunteers before psychotomimetic drug administration that may be related to stress (4, 23).

Physical activity is a well-known cause of increased serum CPK activity (24). However, very strenuous activity (e.g., crew racing or marathon running) is generally required to produce increases, except in a small percentage of so-called enzyme-labile subjects who have large increases in serum CPK activity with relatively slight motor activity (24). A variety of indirect evidence summarized elsewhere (25) suggests that increased motor activity is not a sufficient explanation of the increased serum CPK levels in most psychotic patients. We have recently found that exhausting isometric exercise does not produce increases in serum CPK levels in psychiatric patients that exceed those present during other phases of hospitalization (26). Although Foster and Kupfer (8) reported correlations between serum CPK levels during certain periods of the day and non-dominant-arm motor activity monitored by telemetry, and we have found that nurses' ratings of motor activity and CPK activity are significantly correlated (5), this does not prove causality. In some patients, both motor activity and serum CPK activity could be the consequence of a basic neurochemical abnormality in psychosis, e.g. increased dopaminergic activity. Sleep disturbance in psychotic patients, particularly non-REM sleep time, is negatively correlated with serum CPK levels (27) and may be another example of a central nervous system function that correlates with serum CPK levels without requiring a causal relationship. In normal volunteers, sleep deprivation for 48 hours can produce modest increases in serum CPK activity (28).

CLINICAL CHARACTERISTICS OF PSYCHOTIC PATIENTS WITH AND WITHOUT INCREASED SERUM CPK ACTIVITY

My colleagues (Glenn Lucht and Pamela Holy) and I have studied the clinical characteristics of psychotic patients with and without increased serum CPK activity at admission or at any time during hospitalization to identify the factors that correlate with and perhaps contribute to the increases (5). Such analyses are critical in biologic investigations of this type because of the high likelihood of heterogeneity in the schizophrenic syndrome and because the biologic characteristic under study may be a state-dependent variable rather than a stable trait. This would mean that the clinical state of the patient at the time(s) of study would exert an important influence on the presence of the biochemical abnormality.

I will summarize only our major findings here. The time of onset of psychotic symptoms was significantly related to the presence or absence of increased serum CPK activity. Increased serum CPK activity at admission was significantly more frequent in patients who had had gross psychotic symptoms for less than 1 week (54 of 92 patients, or 58.7%) than in patients who

had had symptoms for more than 1 week (34 of 95 patients, or 35.8%) (χ^2=8.945, p<.005). This is undoubtedly because serum CPK levels are only transiently increased. General characteristics of schizophrenic patients (e.g., acute or chronic, paranoid or nonparanoid) were not related to increases except that chronic nonparanoid schizophrenic patients had significantly fewer increases at admission (3 of 15 patients) than the other three groups (76 of 148 patients) (χ^2=4.178, p<.05). Schizophrenic patients with and without increased serum CPK at admission did not differ on Stephens-Astrup prognosis criteria.

We also found that psychotic patients with increased serum CPK at admission had higher peak ratings on 5 of 8 nonredundant items of a 14-item daily behavior rating scale (derived from Hargreaves [29]) administered during the first week of hospitalization than psychotic patients without CPK increases. The significantly different items included ratings of psychotic behavior, paranoia, lack of effective contact with staff and patients, hyperactivity, and anger. There were no significant differences in ratings of depression, underactivity, and anxiety. A discriminant function analysis of all 14 items of the scale indicated that peak admission ratings significantly discriminated between patients with and without increased serum CPK activity (according to the unpublished data of Glenn Lucht, Pamela Holy, and myself). Psychotic patients with increased serum CPK levels had higher ratings on the psychiatric illness (a measure of global psychopathology) and anxious manner items. Both analyses statistically controlled for differences in the time of onset of psychotic symptoms between the two groups.

Similar analyses using mean behavior ratings throughout hospitalization to compare patients who had increased serum CPK activity at any time during hospitalization with those who had no increases revealed that the former group had significantly greater psychotic behavior ratings (5). A discriminant function analysis of the 14-item scale showed that the best discriminants were the psychiatric illness item for patients with increases and the paranoia item for those without increases (according to the unpublished data of Glenn Lucht, Pamela Holy, and myself). These analyses also controlled for differences in the time of onset.

In addition to these differences in psychopathology, schizophrenic patients with increased serum CPK levels at any time received higher doses of phenothiazine and had significantly longer hospitalizations than those who had no increases (5). The greater length of hospitalization was exclusively due to the patients who had late increases in serum CPK activity, suggesting that a late increase could be some indication of exacerbation of the psychosis. We are currently exploring this possibility.

It appears that those patients with increased serum CPK activity at admission or later in hospitalization tend to have a more florid psychosis that is more resistant to treatment during that episode. The psychotic patients without serum CPK increases tend to be acutely ill patients with brief, treatment-responsive illnesses or chronic patients who are admitted after only relatively slight intensification of their psychoses.

SERUM CPK ACTIVITY IN NONPSYCHOTIC PERIODS AS A FACTOR IN SERUM CPK ELEVATIONS

I assessed the mean serum CPK levels for each patient, based on all samples throughout hospitalization, excluding those which exceeded the 95% upper limit of normal and those taken 5 days or less after a muscle biopsy or 3 days after an intramuscular injection (5). The mean serum CPK levels in psychotic patients with increased serum CPK levels at any time were significantly greater than in psychotic patients who never had an increase. Similar results were obtained using predischarge serum CPK levels without any exclusions, which suggests a biochemical diathesis toward increased serum CPK levels in those patients who have increased serum CPK activity when psychotic. These results also suggest that such individuals may have increased permeability of skeletal muscle (or brain) or slower inactivation or clearance of CPK from serum.

SERUM CPK LEVELS IN FIRST-DEGREE RELATIVES OF PSYCHOTIC PATIENTS

Moline and I (4, 5, 11) have previously reported that first-degree relatives of both schizophrenic patients and patients with affective psychoses have serum CPK levels that are near to or slightly exceed the 95% upper limit of normal. To date, we have obtained one or more serum CPK samples from 314 first-degree relatives of 134 psychotic patients: 32.2% (N=101) have had at least one elevation of serum CPK levels based on the 95% norm for their respective race-sex groups (5). The percentages of increases were approximately the same in parents, siblings, and children of these psychotic patients. The incidence of increased serum CPK activity in 253 first-degree relatives of patients who themselves had increased serum CPK activity (37.2%, N=94) was significantly greater than the incidence in 61 first-degree relatives of patients who did not have serum CPK elevations (11.5%, N=7) (χ^2=13.699, p<.001). The serum CPK levels from families as a unit have also been considered; it was determined that 54.4% (N=56) of the 103 families of patients with an increase had at least one family member with increased serum CPK, while the figure was only 16.1% (N=5) for 31 families of patients without an increase (χ^2=12.551, p<.001) (according to the unpublished data of Robert Moretti and myself). This finding is of interest because it suggests that there could be a genetically determined predisposition toward increased serum CPK levels. Our findings in first-degree relatives take on added significance in that

we have found that serum CPK levels are under significant genetic control in both normal and schizophrenic monozygotic twins, with heritability indices of 0.6–0.8 (30, 31).

SERUM CPK ACTIVITY IN ACUTE BRAIN DISEASES

Increased serum CPK activity has been reported in patients with hemorrhagic and thrombotic cerebrovascular disease, necrotic gliomas, cerebral anoxia, status epilepticus, brain trauma (e.g., gunshot wounds), subarachnoid hemorrhages, drug-induced coma, encephalitis, and meningitis (32, 33). Other characteristics of the serum CPK increases in acute brain syndromes and acute psychoses are similar and differ distinctly from those in chronic neurogenic muscle disease or a myopathy such as polymyositis (5). These characteristics include 1) time from onset of symptoms to the first rise in serum CPK activity, 2) time from the onset of the CPK increase to the peak increase in CPK activity, 3) duration of increased CPK activity, 4) magnitude of increased serum CPK activity, and 5) percentage of patients who have increased serum CPK activity.

Although there are basic similarities in the time course and magnitude of the serum CPK increases in psychotic patients and patients with well-defined acute brain diseases, this does not mean that these increases have the same etiology. It is possible that disruption of tonic upper motor neuron influences on lower motor neurons, loss of trophic substances transported to muscle via blood or nerve, elaboration of myotoxic substances by the brain itself, changes in muscle blood flow, muscle electrolyte composition, oxygen supply, or muscle contraction patterns and intensity might occur in the acute psychoses and acute brain syndromes and have an effect on muscle enzyme efflux.

Other possible causes of enzyme release might also come into play during an acute psychosis or an acute organic brain disease. Entirely different factors or variations of a similar process might account for the increased muscle enzyme release in this wide range of disorders. For example, abnormalities of catecholamines and indoleamines are being investigated in the schizophrenic psychoses (34, 35), affective psychoses (36, 37), acute cerebrovascular diseases (38), and Duchenne-type muscular dystrophy (39). Serotonin can increase serum CPK levels in rats (40) and produce skeletal muscle pathology under various conditions (41, 42). I have observed that the hallucinogenic indole harmaline can increase serum CPK levels in rabbits. Brain lesions in cats can increase some serum enzymes (43).

It should be apparent from this brief discussion that 1) there are mechanisms by which central processes might influence muscle enzyme release; 2) serum CPK increases in the schizophrenic and affective psychoses indicate at least one shared pathophysiologic process (increased muscle enzyme efflux) but not necessarily a common etiology of that process; and 3) the acute psychotic states share this pathophysiologic process with acute organic brain diseases.

SERUM CPK ACTIVITY AND NEUROMUSCULAR DYSFUNCTION IN SCHIZOPHRENIA

On the basis of the evidence of a link between psychosis and the excessive release of CPK from skeletal muscle, we initiated a study of the skeletal muscle fiber morphology in first-degree relatives, psychotic patients, and normal volunteers. Briefly, we found that many psychotic patients had muscle fiber abnormalities, usually confined to a small number of fibers, which is more consistent with a denervation process than a myopathy (44–46), although the distinction between these two classes of muscle disease is blurred (47). Less frequent but similar changes were found in the biopsies of first-degree relatives of psychotic patients (46). These findings led to an examination of the subterminal motor nerves of psychotic patients. We found that 30 of 68 psychotic patients (44%) and 5 of 8 of their first-degree relatives (68%) had excessive branching of subterminal motor nerves and excessive multiple innervation of muscle fibers (46, 48, 49). There were also large numbers of sprouts (branches of subterminal motor nerves) without terminal arborizations, and the nerve endings themselves tended to be more fragmented and dispersed than those of the controls (according to the unpublished data of John W. Crayton and myself).

A significant association between elevated serum CPK levels and the presence of an abnormal muscle biopsy has not been shown. The incidence of abnormal muscle biopsies in psychotic patients with and without increased serum CPK levels was not significantly different, and 90% of the 187 patients had either increased serum CPK activity or an abnormal biopsy (46).

Preliminary neurophysiologic investigations have also been carried out by Crayton and associates (50), who studied serum CPK activity, skeletal muscle fiber pathology, and subterminal motor nerve branching in 50 psychotic patients. The Hoffman (H) reflex recovery curve, which provides an index of α-motor nerve excitability, was significantly related to the diagnosis of psychosis and serum CPK activity. The H reflex is a monosynaptic response evoked by submaximal stimulation of a mixed motor and sensory nerve (51). The afferent discharge travels in the lower threshold sensory fibers. The H response, recorded from the appropriate muscle, occurs as a result of discharge of the afferent fibers of the two-neuron arc or monosynapse in the spinal cord. When two test stimuli are applied to the mixed nerve at various intervals, the second H response is modified by residual effects of the first stimulus on the α-motor neuron. The ratio of the evoked muscle response following the second stimulus to the response following the first stimulus (H_2/H_1) can be

plotted against the delay between stimuli to generate an H reflex recovery curve. Twenty-one of 35 schizophrenic patients (60%) and 4 of 9 patients with affective psychoses (44%) had abnormal recovery curves, compared with 2 of 20 normal subjects in a control group (10%). The abnormalities were of two types: 1) delayed onset of initial recovery and a markedly reduced or absent peak of secondary facilitation when the second stimulus was given 100–300 milliseconds after the first; and 2) an accentuation of the trough following secondary facilitation at 300–500 milliseconds. Both findings suggest decreased excitability of the α-motor neuron. Each of 9 patients with serum CPK levels that exceeded 300 IU/liter had abnormal recovery curves. It is of interest that increased central nervous system dopaminergic activity can produce decreased α-motor neuron excitability (52), and increased brain dopaminergic activity has been proposed as a factor in both schizophrenia and mania (34, 37). This is but one of the many possible bridges between neuromuscular dysfunction and aminergic theories of the major psychoses.

CONCLUSIONS

Measurement of serum CPK activity in psychiatric patients may have some value for the differential diagnosis of psychotic from nonpsychotic psychiatric patients. However, there are several reasons why such determinations may have primarily heuristic rather than clinical value: 1) the brief duration of any increases, 2) those with less florid symptomatology have increases significantly less frequently than the more severely ill psychotic, and 3) some nonpsychotic as well as psychotic patients may have increases for reasons such as injections, trauma, or alcoholism. Interest in CPK has already led to studies that have demonstrated a variety of pathologic changes in the neuromuscular system of psychotics, including morphologic changes in muscle fibers and subterminal motor nerves (44–46) and physiologic abnormalities in nerve conduction velocity (according to the unpublished data of David J. Goode and myself) and spinal cord reflex mechanisms (50). Other techniques for investigating the spinal cord, motor nerves, and muscle fibers will certainly be employed in the study of psychotic patients and may yield further information about the extent of involvement of the neuromuscular apparatus in psychosis. These studies will contribute to the evidence for an organic component of the major psychoses; they may also provide significant insight into the causes of the major psychoses. It seems unreasonable to think that the causes of the neuromuscular dysfunction will be found to be unrelated to the causes of the psychoses themselves. It is conceivable that determining the causes of the neuromuscular dysfunction can be useful in testing current theories of the etiology of the schizophrenic and affective psychoses or generating new hypotheses.

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The Dopamine Hypothesis of Schizophrenia: Focus on the Dopamine Receptor

BY SOLOMON H. SNYDER, M.D.

Alleviation of schizophrenic symptoms by phenothiazines and butyrophenones is associated with blockade of dopamine receptors, while exacerbation of symptoms by amphetamines appears to result from enhanced synaptic activity of dopamine and/or norepinephrine. The author suggests that biochemical labeling of the dopamine receptor with ^{3}H-dopamine and ^{3}H-haloperidol may clarify mechanisms of drug effects on the dopamine receptor.

THE DOPAMINE HYPOTHESIS of schizophrenia is by definition supported by no direct evidence. No one has found anything conclusively abnormal about dopa-

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Dr. Snyder is Professor, Department of Pharmacology and Experimental Therapeutics, and Professor, Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine, 725 North Wolfe St., Baltimore, Md. 21205.

mine (DA) in body fluids or brains of schizophrenics, but the indirect evidence is impressive. The symptoms of schizophrenia can be selectively exacerbated or relieved by the use of drugs. When pharmacologists decipher the neurochemical mechanisms of these drugs, all evidence points to DA.

DOPAMINE

In most areas of the brain, DA, one of the two principal catecholamines in the brain, is merely the precursor of norepinephrine (NE). It is transformed into NE by the enzyme dopamine hydroxylase; however, some parts of the brain lack dopamine hydroxylase, and DA is the presumed neurotransmitter (1–3) in these areas.

The best known DA neuronal pathway has cell bodies in the substantia nigra of the brain stem with nerve terminals in the corpus striatum, the caudate nucleus, and the putamen. This pathway degenerates in patients with idiopathic Parkinson's disease. The resultant DA depletion is causally related to the symptoms of the disease because replacing the missing DA by treatment with its amino acid precursor L-dopa dramatically alleviates symptoms. Thus in Parkinson's disease a dopamine hypothesis of causation has been translated into proof. A DA pathway important to endocrinology has cell bodies in the arcuate nucleus of the hypothalamus and nerve terminals synapsing on the portal vessels in the median eminence that convey releasing factors from the hypothalamus to the pituitary gland. DA in these neurons regulates releasing factor activity and subsequent endocrine effects.

Other DA pathways may be more related to the symptoms of schizophrenia. Pathways with cell bodies close to the substantia nigra project to the nucleus accumbens and olfactory tubercle, both components of the limbic system of the brain that regulates emotional behavior. Recently described DA pathways with cell bodies in the same areas project to parts of the frontal, cingulate, and entorhinal cerebral cortex, phylogenetically older parts of the cerebral cortex functionally related to the limbic system.

AMPHETAMINE ACTIONS

One way to link a drug to a particular illness is to show that the drug reproduces symptoms of the disease. LSD psychosis was once thought to be a "model schizophrenia"; however, one cannot accurately equate the effects of such psychedelic drugs as LSD with schizophrenic symptoms. Psychedelic drugs produce perceptual changes in the visual sphere, whereas schizophrenic hallucinations are usually auditory. Under the influence of psychedelic drugs, people do not demonstrate classic schizophrenic disorders in affect or thought. Moreover, clinicians rarely mistakenly diagnose individuals under the influence of psychedelic drugs as schizophrenics. By contrast, there are numerous reports of amphetamine psychosis being misdiagnosed as acute paranoid schizophrenia before the history of drug ingestion is obtained. Some psychiatrists studying individuals with amphetamine psychoses have felt that they manifested schizophrenic disorders of affect and thought (4, 5), but others disagree (6). Although the specifics of amphetamine psychoses may not exactly match those of schizophrenia, the mere fact that experienced psychiatrists can mistake the two diagnoses indicates that amphetamine psychosis is probably the best current drug model of schizophrenia.

Amphetamine psychosis is not simply a precipitation of a latent schizophrenia or a sleep deprivation psychosis, since psychosis has been elicited by amphetamine in individuals screened for the absence of any history of schizoid disorders; in some cases the psychosis has occurred within 24 hours (6).

Like amphetamine, cocaine is a widely abused CNS stimulant that can, in high doses, provoke a psychosis that is clinically indistinguishable from acute paranoid schizophrenia. Indeed, most of the symptoms of cocaine psychosis are essentially identical to those of amphetamine psychosis. How does cocaine act? An abundance of pharmacologic evidence indicates that cocaine's behavioral effects result from its facilitation of catecholamine synaptic activities by blocking the reuptake inactivation of catecholamines.

Very large doses (100–500 mg) of amphetamine are required to elicit amphetamine psychosis, but amounts prescribed therapeutically for dieting or stimulant effects rapidly and reproducibly exacerbate schizophrenic symptoms (7). Amphetamine and related drugs such as methylphenidate (Ritalin) do not superimpose a drug psychosis on the schizophrenia but instead worsen the patient's own symptoms. This effect is selective—it is not elicited in depressed or manic patients. There is no other drug known that so specifically and dramatically exacerbates schizophrenic symptoms.

How do amphetamines act? Behavioral effects of amphetamines are exerted via the catecholamines NE or DA. Amphetamines enhance the actions of catecholamines by directly releasing them into the synaptic cleft or by preventing their inactivation via reuptake into the nerve terminal that released them. Amphetamines do not affect other neurotransmitter or biochemical systems in the brain so specifically and potently. Although it is difficult to make a clear-cut distinction, the actions of amphetamines in exacerbating schizophrenic symptoms and eliciting amphetamine psychosis probably involve brain DA more than NE. While the neurochemical effects of amphetamines and cocaine are similar, there are differences; cocaine apparently lacks the amphetamines' capacity to directly release catecholamines.

If amphetamines exacerbate schizophrenic symptoms by increasing synaptic DA, then other pharmacologic maneuvers that produce the same biochemical

end product should also worsen schizophrenic symptoms. The simplest technique to determine this would be the administration of L-dopa, the precursor of DA. In the few studies in which L-dopa has been administered to schizophrenics, it worsened behavioral abnormalities in much the same way that amphetamines do (8, 9).

PHENOTHIAZINES

Clinical psychopharmacologists generally agree that phenothiazines exert a specific antischizophrenic action and act primarily on the fundamental symptoms of schizophrenia (10). Their effects are not merely sedative, since they activate withdrawn patients and calm hyperactive individuals. Many other psychotropic drugs, including potent sedatives and antianxiety agents, have failed to provide selective benefits in schizophrenia comparable to the impressive actions of phenothiazines and related agents. Moreover, while phenothiazines are used in the treatment of mania and many other conditions, the relief they afford in schizophrenia appears to be far more specific than their effects in other emotional disturbances.

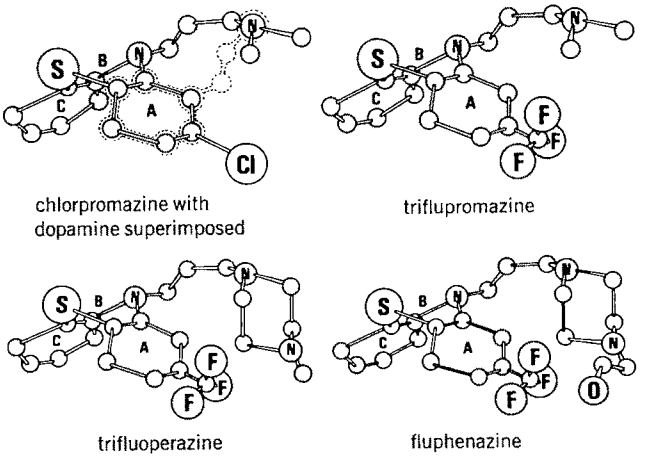
Understanding the mechanisms of action of phenothiazines might shed light on brain mechanisms in schizophrenia. Unlike the amphetamines, the phenothiazines do not possess obvious chemical resemblance to particular neurotransmitters (figure 1). However, these drugs probably act by blocking postsynaptic receptor sites for the neurotransmitter actions of DA. This theory was first advanced by Carlsson (12) on the basis of limited indirect biochemical data. Carlsson hypothesized that if phenothiazines block DA receptors, animal behavior which resembled that induced by the DA depletion after treatment with such drugs as reserpine would result. Postsynaptic neurons would respond to the receptor blockade by sounding an alarm calling for more dopamine. By a feedback system DA cells would then fire more frequently and release more DA, providing an excess of DA metabolites or breakdown products. Carlsson observed an increase in DA metabolites after phenothiazine administration and showed that these biochemical effects of phenothiazines were correlated with the clinical potency of the drugs. This is a crucial task in relating drug "effect" to "mechanism of clinical action."

Since phenothiazines are highly reactive chemicals, they produce many biochemical actions, most of which do not correlate with clinical effects. Thus the phenothiazine promethazine (Phenergan) is a potent antihistamine, but it is totally ineffective in treating schizophrenia, although it exerts most phenothiazine-induced biochemical actions just as effectively as chlorpromazine. Carlsson showed that the increase in DA metabolites produced by chlorpromazine and other phenothiazines was in proportion to their milligram potency in treating schizophrenia, but promethazine was totally ineffective.

Subsequently, other pharmacologists found that the increased formation of DA metabolites elicited by phenothiazines derives from an increased synthesis and, presumably, release of DA (13, 14). Only recently did Aghajanian and Bunney (15) actually show that the DA neurons fire more frequently after phenothiazine administration and that phenothiazines actually block the neurophysiologic effects produced by the injection of minute amounts of DA onto postsynaptic cells possessing DA receptors. Thus, at a neurophysiologic level, one can now demonstrate the synaptic actions of DA, which appears to function as an inhibitory neurotransmitter, and the capacity of phenothiazines to block these effects in proportion to their clinical efficacy.

Because neurophysiologic studies are quite difficult to perform, only a limited number of drugs can be screened. It has been possible to monitor DA receptor activity biochemically via two techniques—an indirect and a more recent direct approach. Cyclic adenosine monophosphate (cAMP) is thought to be a second messenger mediating the actions of many hormones and neurotransmitters. Kebabian and associates (16) showed that a cAMP accumulating system, or adenylate cyclase, in areas of the brain rich in DA nerve terminals responds selectively to DA and is affected much less by other catecholamines (table 1). This DA-sensitive adenylate cyclase is thus linked in some way to the DA receptor and provides an indirect reflection of dopamine receptor activity that can be readily monitored in vitro. As seen in table 1, phenothiazines inhibit the effects of DA on the adenylate cyclase in proportion to their clinical potency (18, 20). Thus trifluoperazine (Stelazine) and fluphenazine (Prolixin, Permitil), two very potent phenothiazines in clinical practice, are considerably more active on the DA-sensitive adenylate cyclase than chlorpromazine, which is clinically less potent.

FIGURE 1
Phenothiazines With the Side Chain "Tilted" Toward the A Ring*



*From Feinberg and Snyder (11).

In order to directly measure the DA receptor biochemically, we labeled neurotransmitter receptors in the brain by binding radioactive forms of the neurotransmitter or its antagonists to synaptic membranes in brain homogenates (21–23). Using such procedures, we recently succeeded in biochemically identifying the DA receptor in the brain (17). DA receptor binding displays all the characteristics one would expect of the DA receptor. Of the various catecholamines, DA has by far the greatest affinity—it is almost 20 times more potent than NE and several thousand times more potent than isopoterenol, the catecholamine with greatest affinity for β-adrenergic receptors. The relative potencies of phenothiazines competing for DA receptor binding closely parallels their clinical potency and their effects upon the DA-sensitive adenylate cyclase. Optical and geometrical isomers of phenothiazine-related drugs are uniquely valuable in the study of receptor specificity. If the physical properties of the two isomers, especially the optical isomers, are essentially the same, any difference in pharmacologic potency must be related to the structure of the receptor sites. The isomeric specificity of the DA receptor binding sites for the isomers of butaclamol and flupenthixol parallels the influences of these isomers on the DA-sensitive adenylate cyclase and their pharmacologic potency in intact animals (table 1).

Interestingly, one group of drugs does not fit in particularly well in either the DA-sensitive adenylate cyclase or DA receptor binding. Butyrophenones such as haloperidol (Haldol) are considerably more potent on a milligram basis than phenothiazines in vivo but are not particularly potent in their influences on the DA receptor in vitro. However, in pharmacologic paradigms in intact animals the potencies of butyrophenones in apparently blocking DA receptors correlates well with their clinical actions. The discrepant actions of butyrophenones on dopamine receptors have been resolved by labeling the receptor with ^{3}H-haloperidol (24–26). Haloperidol and dopamine appear to bind to discrete antagonist and agonist states of the receptor. The clinical potencies of butyrophenones and phenothiazines are predicted impressively by their affinities for haloperidol binding sites (24).

How might phenothiazines be physically accommodated by the DA receptor? In what appears to be their optimum shape or conformation, the side chain of phenothiazines tilts toward the A ring rather than being extended symmetrically between the A and C rings (figure 1). Since the A and C rings are essentially identical except for the presence of a substituent at the 2-position on the A ring, it is likely that this substituent directs the side chain toward the A ring. In the conformation with the side chain approximating the A ring, the amino nitrogen of the side chain and the A ring correspond impressively to the amine and benzene rings of DA and indeed can be superimposed (figure 1) (11, 27, 28). One might predict that chemical factors stabilizing this DA-like conformation of phenothiazines would facilitate DA receptor blockade and enhance clinical potency. The most obvious chemical feature of importance for this conformation is the presence of the substituent at the 2-position on the A ring. A phenothiazine drug lacking such a substituent would possess symmetrical A and C rings so that the side chain would be fully extended in the midline and would not well approximate the DA conformation. In accordance with this prediction, of all the widely used phenothiazines in clinical practice, the only two which are systematically inferior are promazine (Sparine) and mepazine (Dartal) (10). The sole systematic chemical difference between these two drugs and the other phenothiazines is the absence of a substituent on the A ring.

What determines which of the phenothiazines that are clinically efficacious will be the most potent on a milligram basis? One might presume a priori that differences in metabolism and ability to penetrate into the brain account for such large differences in potency that 5 mg of fluphenazine are as potent as 400 mg of chlorpromazine. However, pharmacodynamic studies indicate that such factors cannot account for the differences in clinical potency. Moreover, when assayed directly against DA receptors in binding studies or with the DA-sensitive adenylate cyclase, differences in clinical potency are predicted by affinity for the DA receptor. One might therefore conclude that the more potent phenothiazines must be capable of assuming the DA-like conformation more frequently than less potent phenothiazines. If the A ring substituent and the amine side chain are mutually attracted by chemical forces, the chemical variations that increase these attractive

TABLE 1
Phenothiazine and Thioxanthene Drug Effects on Specific Dopamine Receptor Binding and a Dopamine-Sensitive Adenylate Cyclase

Drug*	Relative Potency in Competing for Dopamine Receptor Binding**	Relative Potency in Inhibiting Dopamine-Sensitive Adenylate Cyclase of Rat Corpus Striatum***
Haloperidol	50	50
(+)-Butaclamol	1200	728
α-Flupenthixol	625	4545
Fluphenazine	465	1087
Trifluoperazine	181	250
Triflupromazine	214	—
Perphenazine	125	—
Chlorpromazine	100	100
Promazine	27	17
(−)-Butaclamol	8	<1

*Drugs are listed in approximate descending order of milligram potency in treating schizophrenia and eliciting extrapyramidal side effects.

**Potency in competing for binding to the dopamine receptor is defined as the reciprocal of the nanomolar concentration to occupy 50% of receptor binding sites × 1.5 × 10^5 in calf striatal membranes using the assay of Burt and associates (17). Thus (+)-butaclamol inhibits binding 50% at 1.25 × 10^{-7} M concentration. Larger values indicate greater potency.

***Data are derived from the studies of Miller and associates (18) and Lippman and associates (19). Clement-Cormier and associates (20) obtained similar results.

forces should stabilize the DA conformation of phenothiazines, producing more potent drugs. Phenothiazines with a triflumethyl substituent in the A ring are more potent than those with a chlorine substituent. Molecular modeling (figure 1) and computer calculations (11) indicate that the triflumethyl group approaches more closely to the amine side chain, providing for greater attractive forces than a chlorine substituent; therefore, drugs with triflumethyl groups should be more potent in binding to the DA receptor. Similarly, phenothiazines with simple piperazine side chains, such as trifluoperazine (Stelazine), are more potent clinically than those with alkylamino side chains. Again simple observations of space-filling molecular models indicate how the piperazine side chain affords more points of attraction to the A ring substituent than the alkylamino side chain. Drugs with hydroxyethylpiperazine side chains, such as fluphenazine (Prolixin), are systematically more potent than those with simple piperazine rings, such as trifluoperazine. Again, drugs with hydroxyethylpiperazine groups provide still more attractive possibilities than piperazine. Thus, simply by designing phenothiazine drugs that more accurately mimic the DA conformation, one can obtain progressively more potent agents.

Phenothiazines may exert presynaptic effects on DA neurons as well, since Seeman and Lee (29) have recently shown that they inhibit DA release in proportion to their clinical potency. The correlation with actual milligram potency is better for this effect than for postsynaptic receptor blockade.

CONCLUSIONS

The blockade of DA receptors correlates with the antischizophrenic activity of phenothiazines, and more potent agents seem almost sculpted to plug into the DA receptor, although they were synthesized by chemists who never heard of DA. The case for the hypothesis that phenothiazines exert their clinical actions by blocking DA receptors is rather impressive. While blocking DA receptors relieves schizophrenic symptoms, amphetamines, which flood the receptors with more DA, exacerbate schizophrenic symptoms. However, none of this proves that DA is the causative "germ" of schizophrenia. The biologic abnormality in a major portion of the schizophrenic population implied by compelling genetic evidence could be far removed from DA. No one has yet shown a specific aberration in DA in the brains of schizophrenics, but Wise and Stein (30) found that dopamine hydroxylase activity is lower in the brains of schizophrenics than in control subjects. A deficiency of this enzyme would result in a build-up of DA, consistent with the effects of drugs on schizophrenic behavior. In one study attempting to confirm the abnormality of dopamine hydroxylase in the brains of deceased schizophrenics, some low dopamine hydroxylase levels were found, but these seemed to depend on how long the brain had been stored after death before assay (31).

Arguments derived from drug influences on schizophrenic behavior are flawed. Phenothiazines do not "cure" all schizophrenics. Many schizophrenics in remission after phenothiazine treatment still have aberrant behavioral patterns. Of course, it can be argued that one can hardly expect a drug, even one that acts at the specific locus of abnormality in the brain, to completely reverse events that have been going on for a lifetime. Certainly, while appropriate antibiotics arrest advanced cases of tuberculosis, the patient is still left with sequelae of the disease. Such analyses are complicated by the likelihood that schizophrenia represents a conglomerate of several "diseases."

The use of amphetamine psychosis as a model for schizophrenia has been questioned on the grounds that the former disorder is usually paranoid, rarely resembling other forms of schizophrenia, and does not display abnormalities of thought and affect that are classic for schizophrenia. Some investigators have observed thought and affect disorders in individuals with amphetamine psychosis. It may be difficult to pick up thought and affect abnormalities in intelligent patients with well-organized paranoid systems of ideation. Also, even if amphetamines cause changes in the brain similar to those which occur in true schizophrenics, major differences are apparent since the drug psychosis occurs in a nonschizophrenic individual who knows that the episode is likely to be short-lived. By contrast, a schizophrenic has experienced an abnormal mental state for most of his life and has no hopes of any major relief. The selective exacerbation of schizophrenic symptoms by low doses of amphetamines is probably stronger evidence for the psychotogenic capabilities of these drugs.

Despite these reservations, there is little doubt that research prompted by the relationship of drugs, DA, and schizophrenia has greatly advanced our understanding of psychotropic drug action. It is hoped that such research will be relevant to schizophrenia.

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The Psychedelic Model of Schizophrenia: The Case of *N,N*-Dimethyltryptamine

BY J. CHRISTIAN GILLIN, M.D., JONATHAN KAPLAN, M.D., RICHARD STILLMAN, M.D., AND RICHARD JED WYATT, M.D.

The authors review the research on N,N-*dimethyltryptamine (DMT) as a possible "schizotoxin." DMT produces psychedelic effects when administered to normal subjects, the means are present to synthesize it in man, it has occasionally been found in man, and tolerance to its behavioral effects is incomplete. However, DMT concentrations have not been proven to differ significantly in schizophrenics and normal controls. Also, in vivo synthesis of DMT has not been convincingly demonstrated, and the psychological changes it produces do not closely mimic the symptoms of schizophrenia. The authors conclude that more data are necessary before the validity of this theory can be determined.*

MANY THEORIES of schizophrenia are based on the assumption that the illness is caused by a chemical substance, a "schizotoxin," that would mimic the clinical symptomatology of schizophrenia if it were isolated and administered to normal individuals. An example of this research strategy is the transmethylation hypothesis, first proposed by Osmond and Smythies in 1952 (1). These researchers noted the similarity in chemical structure of mescaline and norepinephrine, an observation that spurred the search for psychedelic methylated metabolites of endogenous compounds in schizophrenic patients that might account for hallucinations, delusions, and other clinical symptoms.

Interest in the transmethylation hypothesis was further stimulated by the work of Pollin and associates (2), who reported in 1960 that methionine, a natural methyl donor, administered in combination with a monoamine oxidase (MAO) inhibitor, exacerbated clinical symptoms in 3 of 9 schizophrenic subjects. Since that time, methionine has been administered to schizophrenics in 10 studies reviewed by Cohen and associates (3). One-third of the patients in these studies exhibited increased clinical symptoms without evidence of organic brain dysfunction. These results suggest that methionine increases the synthesis of an abnormally methylated substance, as yet unidentified, that was responsible for the observed clinical exacerbation.

Several substances have been proposed as candidates for the schizotoxin, including 3,4-dimethoxyphenylethylamine (pink spot), bufotenine, 5-methoxy-*N,N*-dimethyltryptamine, and *N,N*-dimethyltryptamine (DMT) (see figure 1). The first two substances have not been shown to produce psychotic psychological changes when administered to normal individuals (4, 5); in the case of bufotenine, this may be because it does not cross the blood-brain barrier. Little is known about 5-methoxy-DMT, although it has been reported to produce psychedelic effects in normal individuals (6), and urinary concentrations of 5-methoxy-DMT have been noted to increase with clinical exacerbation in schizophrenic patients (7). Figure 1 presents the postulated routes of synthesis of DMT and 5-methoxy-DMT.

In this paper, we shall review current evidence on the role of DMT as a causative agent in schizophrenia. Fortunately there are criteria (modeled after Koch's postulation) with which to evaluate a hypothesized schizotoxin. The following criteria were initially proposed by Hollister (8) and have been slightly modified here:

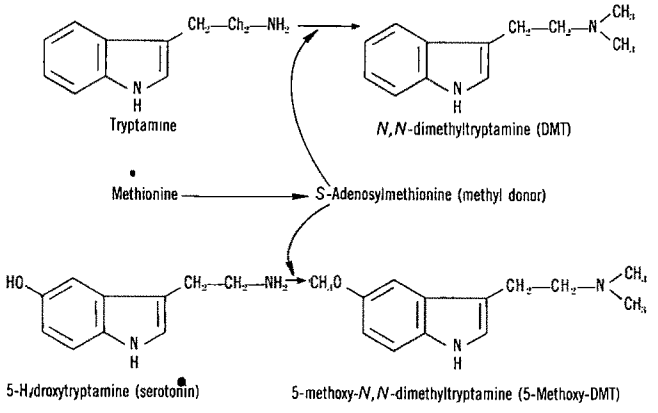
1. The substance must mimic important clinical aspects of schizophrenia.
2. The substance must be found in man.
3. The precursor must be found in man.
4. The agent must be synthesized in man. (It remains a logical possibility that it is of dietary origin.)
5. The agent must be differentially synthesized or metabolized in schizophrenia.
6. Tolerance to the agent should not develop. (This assumes that schizophrenia is not caused by a substance that is present only briefly and sets irreversible changes in progress.)
7. Neuroleptic drugs must be capable of inhibiting the synthesis, increasing the metabolism, or antagonizing the behavioral effects of the agent.

The body of our paper will consist of an examination of each of these criteria as they apply to DMT.

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Drs. Gillin and Stillman are Research Psychiatrists, and Dr. Wyatt is Acting Chief, Laboratory of Clinical Psychopharmacology, National Institute of Mental Health, St. Elizabeths Hospital, Washington, D.C. 20032. Dr. Kaplan is Staff Psychiatrist, Menlo Park Veterans Administration Hospital, Menlo Park, Calif.

FIGURE 1
Postulated Routes of Synthesis of DMT and 5-Methoxy-DMT



DOES DMT PRODUCE SIGNIFICANT SCHIZOPHRENIC-LIKE SYMPTOMS?

In 1956 Szara (9) found that the effects of DMT on 20 normal volunteer subjects were similar to those of LSD and mescaline: visual illusions and hallucinations, distortions of spatial perception and body image, speech disturbances, and euphoria. A striking finding was that the effects of DMT began within 5 minutes and ended within 1 hour after injection. Similar results have since been reported by Rosenberg and associates (10) and Turner and Merlis (5).

In order to reexamine the psychological effects of DMT and to correlate them with pharmacokinetic aspects, we administered .7 mg/kg of DMT intramuscularly to 15 male volunteers. Each subject was an experienced user of LSD, mescaline, or other psychedelic substances who expressed an intention to continue using these agents in the future. All subjects were interviewed by two psychiatrists and were given a complete medical history and examination prior to testing in order to ensure the absence of psychiatric and physical impairment.

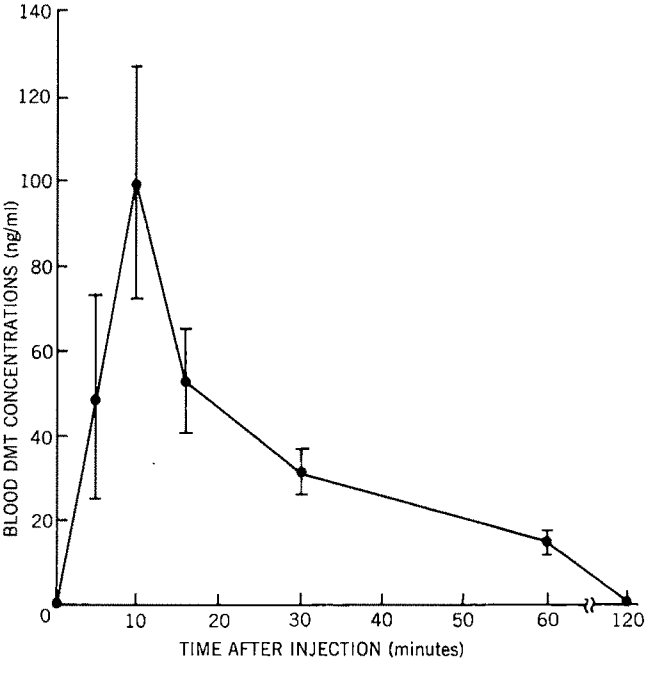
Like previous investigators, we found that DMT was a hallucinogen with rapid action and a short duration of effect. Psychological changes were evident within 5 minutes of injection, peaked at about 10 to 15 minutes, and ended within 45 to 120 minutes. The major psychological effects are shown in table 1. The subjects became so uncommunicative and withdrawn during the drug experience that we were forced to inquire about the subjective effects with simple "yes-no" questions. Although all subjects reported visual distortions and illusions, these were color or spatial distortions rather than formed visual hallucinations. Only 1 subject reported an auditory hallucination, a "buzzing bee" in his ear. We did not observe formal loosening of associations, although several subjects seemed to have thought blocking. Two subjects had paranoid symptoms that lasted less than an hour.

These psychological changes were accompanied by

TABLE 1
Subjective Effects of DMT Experienced by 15 Normal Volunteer Subjects, in Percents

Subjective Effect	Percent
Visual hallucinations	100
Hallucinations with eyes closed	100
Movement of surroundings	93
Difficulty talking	93
Difficulty describing feelings	93
Relaxation	93
Difficulty concentrating	93
Colors seem brighter	87
Excitation	87
Thinking faster	87
Dry mouth	87
Tenseness	80
People look different	75
Depersonalization	60
Nausea	60
People have orange-red hue	53
Hallucinating "real things"	27
Paranoia	20
Auditory hallucinations	7

FIGURE 2
Mean DMT Concentrations in Whole Blood Following Injection of DMT in 15 Normal Volunteers



mydriasis, tachycardia, and increased blood pressure. Blood levels of DMT (see figure 2), assayed by a gas chromatographic-mass spectrometric (GC-MS) isotopic dilution technique, closely paralleled the psychological and autonomic changes (11). Peak concentrations of DMT, which averaged approximately 100 ng/ml,

were reached about 10 to 15 minutes after injection; the concentration then fell rapidly to baseline, undetectable levels within about 45 to 120 minutes after administration.

IS DMT FOUND IN MAN?

Several investigators have reported the presence of DMT in blood and urine of psychiatric patients (see table 2). Positive reports were more common with the earlier, less specific methods of analysis than with the later GC-MS techniques. Although DMT has occasionally been identified by GC-MS techniques in some subjects, the concentration has been extremely low. Also, the presence of DMT has not been reported more frequently in patients with particular diagnoses or in psychiatric patients in general compared with normal controls. The origin and significance of the identified DMT is unknown. Although it might reflect endogenous synthesis, it could also result from diet, bacterial products, laboratory error, or other sources.

TABLE 2
Recent Research on DMT in the Blood and Urine of Schizophrenics

Authors (in chronological order)	Method*	Sample	Subjects	Number Tested	Number Positive
Heller and associates (12)	TLC, GLC	Whole blood	Acute unmedicated schizophrenics	5	5
		Urine		2	2
		Whole blood	Chronic schizophrenics	9	0
			Psychotic depressives	1	0
			Normal subjects	2	0
Rosengarten and associates (13)	TLC	Urine	Schizophrenics	15	3
Narasimhachari and associates (14)	TLC, GLC	Serum	Acute schizophrenics	22	13
			Neurotics	5	1
			Chronic schizophrenics	4	0
			Normal subjects	10	2**
			Mother of manic-depressive	1	1
Narasimhachari and associates (15)	TLC, GLC	Urine	Chronic unmedicated schizophrenics	216***	9
			Normal subjects	56	0
Kanabus and associates (16)	PC	Urine	Nonschizophrenics	16	1
			Schizophrenics	26	4
Wyatt and associates (17)	GC-MS	Plasma	Acute schizophrenics	10	0
			Chronic schizophrenics	9	0
			Psychotic depressives	10	1**
			Normal subjects	11	1**
Narasimhachari and associates (18, 19)	TLC, GLC, GC-MS	Urine	Chronic schizophrenics	6	3
			Normal subjects	4	0
Axelsson and Nordgren (20)	Gel C TEC	Plasma	Acute and chronic schizophrenics	9	0
			Neurotics	4	0
Bidder and associates (21)	GC-MS	Whole blood or plasma	Psychiatric patients	37	2
		Urine		39	7
Lipinski and associates (22)	GC-MS	Whole blood or plasma	Chronic schizophrenics	6	0
			Schizophreniform psychotics	11	2
			Patients with hepatic coma	11	0
			Normal subjects	11	0
Mandel (23)	GC-MS	Whole blood, plasma, serum	Acute schizophrenics	35	6
			Chronic schizophrenics	46	4
			Nonschizophrenics	66	3
			Normal subjects	84	4
Oon and associates (24)	TLC, MS	Urine	Schizophrenics	42	20
			Affective psychotics	30	4
			Other psychotics	18	7
			Neurotics	32	6
			Normal subjects	20	1
Carpenter and associates (25)	GC-MS	Urine	Acute schizophrenics	12	4
			Normal controls	9	4

* TLC=thin layer chromatography, PC=paper chromatography, GLC=gas-liquid chromatography, GC-MS=gas chromatography-mass spectrometry, Gel C=gel chromatography.
** Doubtful.
*** Number of samples for 6 patients.

IS THE PRECURSOR OF DMT PRESENT IN MAN? CAN DMT BE SYNTHESIZED IN MAN?

As figure 1 indicates, DMT is thought to be synthesized from tryptamine in a reaction catalyzed by an *N*-methyltransferase (NMT). Tryptamine is reportedly present in human lung and elsewhere (26). NMT activity has been reported in human brain (27, 28) and blood (29), but its highest activity and specificity is in human lung (30). Mandel and associates (30), using GC-MS techniques, demonstrated in vitro conversion of *N*-methyltryptamine to DMT in human lung. Wyatt and associates (31) and Murphy and Wyatt (32) have found that MAO activity in platelets is low in some schizophrenics compared with control subjects, and that this defect may be genetically determined. Low MAO activity may lead to elevated concentrations of tryptamine, which would favor the synthesis of DMT.

In vivo human synthesis of DMT has not been convincingly demonstrated to date. We studed 1 male chronic schizophrenic patient before and during administration of 45–60 mg/day of the MAO inhibitor phenelzine. Although the patient was more hallucinatory and autistic on phenelzine than he had been during the placebo period, DMT concentrations in venous blood did not change from their very low placebo values (approximately .07 ng/ml). We also studied another male schizophrenic patient before and during treatment with 20 g/day of L-tryptophan, the biosynthetic precursor of tryptamine. Again, there was no change in blood DMT concentration. The patient's clinical condition was not altered by L-tryptophan.

IS DMT SYNTHESIZED OR METABOLIZED DIFFERENTLY IN SCHIZOPHRENICS THAN IN CONTROLS?

Synthesis of DMT in man has yet to be conclusively demonstrated, and little knowledge currently exists on the metabolism of DMT in schizophrenics compared with normal control subjects. Szara (9) suggested two major routes of metabolism of DMT: 1) dealkylation and oxidative deamination, leading to indole-3-acetic acid and 2) 6-hydroxylation followed either by glucuronide formation of 6-hydroxy-DMT or by dealkylation and oxidative deamination leading to 6-hydroxyindoleacetic acid (33). To our knowledge, there have been no studies comparing the concentrations of these metabolites in schizophrenics and controls.

Our recent study of the effects of DMT on volunteer subjects (11) suggested that the drug is rapidly metabolized. Assuming that DMT is distributed equally throughout the blood, we could account for only about 2% of the administered dose at the time of the peak blood concentration. Moreover, less than .01% of the administered dose was found in urine within 24 hours, and most of that amount was excreted within the first 6 hours.

Thus the failure of various investigators to find elevated concentrations of DMT in venous blood or urine of schizophrenics may be explained by the rapid metabolism of this compound. If DMT is actually formed by lung NMT, then schizophrenia is a lung disease, and DMT may be transported directly from lung to brain via the arterial system. Once DMT is exposed to liver, kidney, or muscle, it may be so rapidly metabolized that measurable concentrations cannot be detected in venous blood.

DOES TOLERANCE TO DMT DEVELOP?

Since schizophrenia is a clinical syndrome that lasts for weeks to months in its acute forms and for years to decades in its chronic forms, a biochemical theory must be able to explain long-term symptoms. The issue of tolerance has been a major problem in the schizotoxin theory of schizophrenia. Tolerance to LSD, mescaline, and psilocybin develops rapidly in man and animals, for some if not all behavioral effects.

In our initial efforts, we found that tolerance did not develop to unconditioned behavioral and EEG effects of DMT in cats administered DMT twice daily for 15 days or every 2 hours for 24 hours (34). Also, lack of behavioral tolerance has been reported in squirrel monkeys given DMT once daily for 38 days (35).

More recently, we studied the issue of tolerance in 4 normal male volunteers who received 0.7 mg/kg of DMT intramuscularly twice daily for 5 days. Repeated administration did not consistently alter the peak blood concentration of DMT; autonomic changes in pupil size, pulse, or heart rate; the number of psychological items changed in a psychological scale; or the frequency of errors in a test requiring the subject to cross out a specific number in a list of random numbers. Three of the 4 subjects reported diminished subjective "highs" on a scale of 0 to 10 after two to four injections of DMT, but their subjective responses were variable from trial to trial and did not indicate a general loss of responsiveness to DMT. Rather, these subjects exhibited a variable or aperiodic partial tolerance to DMT. This pattern is reminiscent of Koella and associates' report of a cyclic change in ambulation produced by LSD in goats (36). Further studies, including longer or more frequent trials with DMT, are necessary to fully evaluate this phenomenon.

This type of variable tolerance has also been reported recently by Kovacic and Domino (37), who studied the suppressive effects of DMT on the operant behavior of appetitively conditioned rats who were given DMT every 2 hours for periods of up to 21 days.

Böszörményi and Szara (38) reported that schizophrenics are less sensitive to the effects of DMT than controls. If schizophrenics do show diminished responsiveness to DMT, this may result from increased metabolism or variable tolerance resulting from long-term endogenous synthesis of DMT.

DO NEUROLEPTICS INHIBIT THE SYNTHESIS, INCREASE THE METABOLISM, OR ANTAGONIZE THE EFFECTS OF DMT?

Although the evidence is currently incomplete, there are data suggesting that antipsychotic medications may inhibit the activity of NMT. Axelrod (39) has suggested that chlorpromazine might have this effect. Moreover, a dialyzable inhibitor of NMT has been reported in bovine pineal gland (40), which is an interesting finding because Bigelow (41) obtained favorable clinical results in some schizophrenic patients with an aqueous extract of bovine pineal gland. Some dimethylated metabolites of chlorpromazine have also been reported to be competitive inhibitors of NMT (42). Thus it is conceivable but unproven that antipsychotic drugs inhibit the synthesis of a methylated schizotoxin. Little is known, however, about the influence of antipsychotic medications on the metabolism or behavioral effects of DMT.

DISCUSSION

Although the DMT model of schizophrenia remains attractive, most of the positive evidence is indirect, supporting the plausibility rather than the reality of the model. DMT does produce striking psychological changes in normal subjects, the enzymes and precursors are present to synthesize it in vitro in human tissue, it has been found occasionally in man, and tolerance to its behavioral effects is incomplete. Moreover, effective antipsychotic treatments may conceivably interfere with the synthesis of DMT.

On the other hand, significant differences in DMT concentrations in schizophrenics versus controls have not been proven, in vivo synthesis of DMT in man has not been demonstrated, and the psychological changes induced in man by DMT do not closely mimic the clinical symptoms of schizophrenia. When psychological changes have been produced in normal subjects, this has been with DMT blood concentrations 50 to 100 times greater than those in the few schizophrenic patients in whom DMT has been identified.

Arguments can undoubtedly be produced to refute each of these objections. The psychological effect of DMT in normal individuals probably should not be expected to mimic schizophrenia. After all, the volunteer knows what is happening to him when the experimenter administers DMT, but DMT is (hypothetically) synthesized endogenously in schizophrenic patients. The data on DMT in blood and urine may be of dubious relevance in view of its rapid clearance from blood and its failure to appear in appreciable amounts in urine.

Like any good scientific theory, the DMT model of schizophrenia will ultimately live or die by the data that it heuristically generates. We hope that, within the foreseeable future, forthcoming data will give this theory either a new lease on life or a decent burial.

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Recent Developments in the Drug Treatment of Schizophrenia

BY JOHN M. DAVIS, M.D.

The author reviews six topics relevant to the drug treatment of schizophrenia. The quantitative effectiveness of promazine is of interest with respect to the structural models of the phenothiazines and the dopamine theory of schizophrenia. The quantitative effectiveness of antipsychotic drugs is also important in evaluating new agents and therefore relevant to a discussion of two newly released neuroleptics, molindone and loxapine. The author's discussion of high-dose treatment for typical acute schizophrenics or treatment-resistant patients reviews the available data and calls attention to the fact that these areas of pharmacologic research have not received sufficient attention.

SOME RECENT DEVELOPMENTS in the drug treatment of schizophrenia are relevant to six problems of practical importance: 1) the effectiveness of the antipsychotic agents as compared with placebo in the treatment of schizophrenia; 2) indications for use of antipsychotic drugs; 3) the usefulness of massive doses in improving clinical response in typical acute schizophrenia; 4) drug treatment of the phenothiazine-resistant patient; 5) phenothiazine effectiveness and molecular models; and 6) the effectiveness of two new neuroleptic agents, molindone and loxapine. Since the use of the intramuscular depot drugs such as fluphenazine decanoate (a useful strategy for schizophrenic patients who tend to forget to take their pills and an important addition to clinical therapeutics) has already been reviewed recently in the *American Journal of Psychiatry* (1), I will limit the content of this paper to avoid redundancy.

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Dr. Davis is Director of Research, Illinois State Psychiatric Institute, 1601 West Taylor St., Chicago, Ill. 60612, and Professor of Psychiatry, University of Chicago, Pritzker School of Medicine, Chicago, Ill.

QUANTITATIVE MEASUREMENT OF NEUROLEPTIC EFFICACY

In a statistical analysis of the efficacy of any medication, it is important to distinguish the quantitative magnitude of an effect from the statistical probability that it may occur by chance alone. For example, one could find a very high correlation of marginal statistical significance because of a small sample size or an extremely low correlation that is highly significant because it is based on a large sample. The real issue is the magnitude of the neuroleptic effect, i.e., the degree of difference in improvement or deterioration between neuroleptic drug and placebo groups, regardless of sample size.

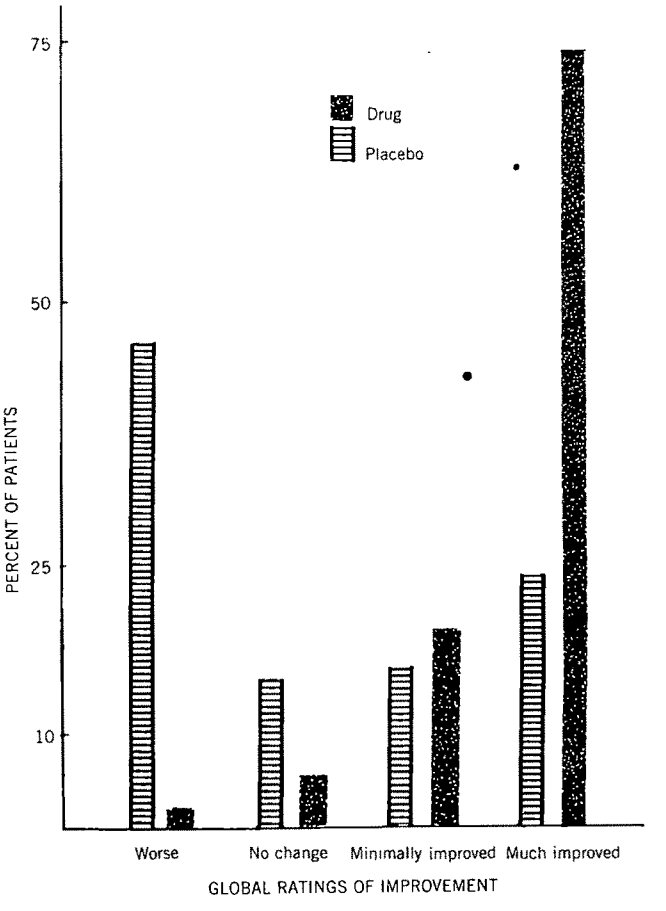
In the National Institute of Mental Health (NIMH) collaborative study on the treatment of acute schizophrenics (2, 3), many patients in the placebo group deteriorated to such a degree that they had to be removed from the study (2, p. 248 [table 2]). In any drug study one important index of drug-placebo differences is the number of patients whose conditions deteriorate to such a degree that they must be removed from the study or who improve to such a degree that they can be discharged. The NIMH collaborative study also evaluated the patients who completed the trial as to whether they were much improved, minimally improved, unchanged, or worse (2, p. 252 [figure 2]).

Figure 1 presents these data, combining the number of patients who deteriorated with the global evaluation of deterioration or improvement on drug or placebo. The magnitude of the drug effect can be seen in this figure without recourse to mathematical statements. One can see that three-fourths of the patients benefitted substantially from antipsychotics during the six-week trial and virtually no patients deteriorated, whereas nearly 50% of the placebo group deteriorated.

Quantifying these impressions presents a somewhat more subtle problem. The total drug-placebo difference can be estimated by assigning the arbitrary numbers 0 and 1 to drug and placebo and the numbers 2, 1, 0, and –1 to the different degrees of improvement. One can then calculate from this kind of data a rank order correlation coefficient. (Because of the ties, the product-moment formula is the appropriate statistic to use.) It should be noted that since the data underlying the arbitrarily chosen numbers are not necessarily at equally distant intervals, no precise estimate of the percent of variance explained by the correlation can be calculated.[1]

In any case, the correlation between therapeutic improvement and whether or not drug or placebo was given would be estimated at about .60. Using parametric statistics, the squared product-moment correlation

FIGURE 1
Doctor's Global Rating of Improvement in Patients After Treatment with Phenothiazines or Placebo



coefficient ($.60^2 = .36$) indicates a percentage of the variance explained by the correlation. Unfortunately, with nonparametric statistics the concept that one's findings explain a particular percent of the variance is somewhat problematic, so we cannot offer this as a quantitative estimate of percent of variance explained in any precise sense other than perhaps as an estimate of the general order of magnitude effect. Parenthetically, it might be added that thioridazine, when used to treat anxiety, explains (through a similar calculation procedure) about 5% of the variance (or a correlation of around .20), a much smaller effect than its antipsychotic effect in schizophrenia.

A somewhat smaller drug-placebo difference was expected in studies of chronic patients, such as maintenance trials on chronic inpatient schizophrenics or on chronic relapsing outpatient schizophrenics. To estimate the order of magnitude of this side effect for the more chronic population, we examined four particularly good NIMH-sponsored double-blind placebo-controlled trials of antipsychotic medication in chronic schizophrenics (2 inpatient [4–6] and 2 outpatient [7–10] studies), and also two double-blind placebo-controlled British trials (11, 12) of chronic neuroleptic medication. Since the distance between the mean drug

[1]It is relevant to add here that even if a quantitative rating scale were used (e.g., BPRS total score) there is no assurance that the different scores or covariance–adjusted final scores would meet such mathematical assumptions as to allow precise estimation of how much variance is explained by the drug-placebo difference.

effect and the mean placebo effect on a scale of improvement increased over time, data must be adjusted to a common study length. The number of patients who deteriorated versus those who maintained their improvement was compared in all of these studies, adjusting to a common study length of 8 weeks. In each case the data from the study were fitted by the least squares method using the equation $R=R_0 \times e^{-kt}$ (R=the number of patients not relapsed; R_0=number of patients that begin in the trial; and t=time). The number of patients in the placebo and drug groups who relapsed or did not relapse at 8 weeks was included in our estimate of the magnitude of the drug-placebo difference in this population. These four numbers constitute a 2×2 table, yielding a phi coefficient (nonparametric estimate of the product-moment correlation coefficient) of .23 (4–12).

INDICATIONS FOR ANTIPSYCHOTIC DRUG TREATMENT

Figure 1 illustrates the fact that a substantial number of patients improve considerably on placebo. This raises the question of what the indications are for antipsychotic drug treatment. Should all schizophrenic patients receive neuroleptic drugs, or will some do better without drugs? This question has not received great attention in the literature. However, one large NIMH collaborative study (13) that investigated 338 patients (74 on placebo and 264 on neuroleptics) correlated a number of variables indicative of process or reactive schizophrenia with differential improvement on drug versus placebo and found that virtually none of these variables predicted response when they were considered individually. Of course, linear combinations of these variables or a higher order combination might have yielded predictors, but even so this is still significant negative evidence. More importantly, though, the lack of evidence here indicates that there is a real gap in our existing knowledge. In many hospitals most schizophrenic patients do receive neuroleptics, so the implicit assumption of many psychiatrists may be that neuroleptics are indicated for all cases of schizophrenia, a somewhat undifferentiated treatment strategy. Conversely, other practitioners proceed as though neuroleptics are to be used only as a last resort after a clear failure of psychotherapeutic methods. A more rational form of therapy would be to base the choice of treatment with antipsychotic drugs on clinical indications defined by research.

MEGADOSE TREATMENT FOR ACUTE SCHIZOPHRENIA

It is clear that one can give massive (mega) doses of such agents as trifluoperazine and fluphenazine that are substantially above the average therapeutic dose. (Doses of chlorpromazine are limited by the sedative side effects that develop, and doses of thioridazine are limited particularly by the development of pigmentary retinopathy, which can occur when gram doses are given.) Would it therefore follow from the clinical information in several studies that treatment-resistant or newly admitted schizophrenics would benefit significantly from massive doses with either more complete or quicker remissions?

Wijsenbeeck and associates (14) performed a double-blind study comparing the efficacy of 60- and 600-mg doses of trifluoperazine in newly admitted schizophrenic patients and found the two treatments to be equally effective. It seems that for the average newly admitted schizophrenic patient, megadoses do not produce substantially greater improvement than regular doses. It is important to remember that, according to an empirically derived equivalence table, 60 mg/day of trifluoperazine is equivalent to about 1 g/day of chlorpromazine and therefore is a high normal dose; 600 mg/day is about 30 times the normal antipsychotic dose (734 mg/day of chlorpromazine) (15).

Conversely, is it possible that there are ineffective doses of antipsychotic agents? Although there are few data from newly admitted patients, there are several studies of subchronic patients (i.e., those who have been ill less than 10 years). One study (16) found that doses of 150–300 mg/day of chlorpromazine were less effective than doses of 600 mg/day, and two other studies (4, 6) showed that 300-mg daily doses of chlorpromazine were less effective than 2000-mg daily doses.

Another study (5) found that a 15-mg daily dose of trifluoperazine (equivalent to 535 mg/day of chlorpromazine) was as effective as 80 mg/day of trifluoperazine (equivalent to 2.8 g/day of chlorpromazine). This probably indicates that 535 mg/day is close enough to the effective dosage range and that an exceptionally high dose will not produce more improvement for this chronic population. However, daily doses of 300 mg are probably below the effective therapeutic range for chronic patients. I have reviewed these studies on dose levels to call attention to this neglected area and to present what little evidence there is available at present on dose-response curves for the antipsychotics.

TREATMENT OF THE PHENOTHIAZINE-RESISTANT PATIENT

Estimates of the dose-response curve should be interpreted in view of our wide clinical experience, which indicates that different patients respond to widely different doses. The explanation of this differential responsiveness undoubtedly lies, at least in part, in the different rates of metabolism of the antipsychotic drugs. In previous studies (17–19), we have found over 100-fold variations of chlorpromazine blood levels between different patients on the same dose of chlorpromazine and a 30-fold variation in different patients

on the same dose of butaperazine. Fast metabolizers may destroy the drug so rapidly that they receive ineffective brain levels unless they are given very high doses, and slow metabolizers may have unusually high brain levels that may be toxic even when they receive normal doses. Some nonresponding patients may be fast metabolizers who would in fact respond if ultra high doses were used. This raises the problem of treatment strategy for the phenothiazine-resistant patient, i.e., the patient who has been treated with adequate doses of antipsychotics but did not improve.

Previous laboratory work indicates that chlorpromazine may be metabolized to a fair extent in the gut wall. To bypass this source of rapid metabolism, we have administered fluphenazine decanoate in high doses to antipsychotic-resistant patients and have achieved good clinical results in patients previously resistant to these drugs. Furthermore, in a double-blind study, Lewis and associates (20) and Curry and Adamson (21) identified such patients by studying their blood level response to a standard dose of chlorpromazine and also found better results in these fast gut metabolizers by using intramuscular fluphenazine rather than oral medication. Although further studies are needed for definitive proof, this initial clinical evidence indicates that perhaps some patients who are resistant to antipsychotic drugs might be tried on parenteral administration. Another way of dealing with fast metabolism may be the use of ultra high doses. Several groups have administered ultra doses (e.g., 1 g of fluphenazine) and reported good results (22–25). However, more evidence is needed, particularly in well-controlled double-blind studies, for definitive proof. There may be indications for megadose treatment in this selected treatment-resistant population in contrast to the random sampling of newly admitted patients where ultra doses do not appear to be helpful for the average patient.

PHENOTHIAZINE EFFICACY AND STRUCTURAL DOPAMINE MODELS

Elsewhere in this section, Dr. Snyder discusses the dopamine theory of schizophrenia. Horn and Snyder (26) noted that there is a structural similarity between the antipsychotic drugs and dopamine receptors. Therefore, it is relevant to compare the clinical efficacy of these drugs with their structural similarity to dopamine receptors. These drugs—except promazine—have equal effectiveness in the treatment of schizophrenia. Since Horn and Snyder proposed an explanation for this in terms of the structural chemistry of promazine, it is relevant to review to what quantitative degree promazine is less effective than chlorpromazine. This finding may become more intelligible in light of Horn and Snyder's observation that the chlorine substituted in the 2-position may selectively orient the chlorpromazine side chain to the A ring. Without this substitution there could be a 50:50 chance of the side chain being thus oriented. It is interesting that promazine does have antipsychotic properties. The two best controlled studies comparing the improvement produced by promazine with chlorpromazine, after subtraction of placebo effect, are those of Kurland and associates (27) and Casey and associates (28). The first study found that promazine produced 51% of the improvement produced by chlorpromazine (27, p.6); in the second study, promazine produced 47% of the improvement produced by chlorpromazine (average improvement at 18 and 24 weeks) (28, p.214). This interesting quantitative comparison, in which promazine appears to be approximately 50% as effective as chlorpromazine in its antipsychotic action, must be viewed from the perspective that the measuring instruments used may not yield additive data.

Much of the relationship between basic biologic science and clinical science rests on correlating the biochemical pharmacology of the drugs with their clinical efficacy. For this reason, it may be instructive in laboratory experimentation to investigate control substances that are structurally similar to effective neuroleptics but do not have antipsychotic properties. Mepazine may be such a drug. It is interesting, for example, that mepazine, a close structural analogue to chlorpromazine, essentially has no extrapyramidal side effects (i.e., the incidence of extrapyramidal side effects with mepazine is equal to that of phenobarbital or placebo) (29–33).

THE EFFECTIVENESS OF TWO NEW ANTIPSYCHOTIC AGENTS

Discussion of Methodology

I will describe certain methodologic points in this discussion of two antipsychotic agents recently introduced on the market. When a new drug is being compared with a standard drug, random error may result in the new drug being found to be equivalent to the standard drug by obscuring a real difference. In addition, a new drug could be found to be equivalent to a standard drug in a very treatment-resistant population—neither drug would show a significant effect. This raises a question regarding the use of statistical tests to rule out false-positive results. Is a new drug found to be falsely equivalent to the standard drug for the above reasons?

Cohen (34) discussed the question of what statistical power is necessary in order to rule out these false-positive (type 2) errors, with the probability of also ruling out false-negative (type 1) errors. In order to do this, it is necessary to have a statement of the treatment effect that I am defining here as the statistical difference between drug and placebo. Assuming our admittedly imprecise estimate reflects the magnitude of the true treatment effect, we can estimate how many patients should be studied in a new drug versus standard drug trial in order to state at the 99% confidence level for both type 1 and type 2 errors that a new drug is in fact

different from the standard drug. At the same time we can keep our statistics sensitive enough to detect a real drug difference, holding the probability of both types of error to the .01 level. Our estimate is that in a maintenance trial 400 patients are needed for this purpose.

Clinical Aspects

Recently two new antipsychotic agents of different structural classes have been introduced on the market. Molindone (Moban) is the first antipsychotic agent that belongs to the indole category and is chemically quite separate in structure from the previous classes (phenothiazines, thioxanthenes, and butyrophenones.). Since molindone blocks amphetamine-induced stereotypic behavior in animals (an abnormal movement disorder mediated by dopamine) and also causes extrapyramidal side effects, it can be inferred that it is probably a dopamine receptor blocker.

The principal evidence that molindone is an effective antipsychotic comes from a very carefully controlled study performed by Clark and associates (35) that compared molindone, chlorpromazine, and placebo. Molindone was clearly superior to placebo on global rating scales of both physician and nurse and on the BPRS and NOSIE scales. Chlorpromazine, as expected, was also superior to placebo.

There are a total of six controlled studies (35–40) with approximately 200 patients that found molindone approximately equal to the standard drug: it was slightly superior in one study, equal in three, and slightly inferior in two. Since *none* of these differences were statistically significant and since the small, minor drug-placebo differences were approximately normally distributed around the standard drug, the empirical data indicate that molindone is approximately equally as effective as the reference antipsychotic. However, in view of the previously mentioned power considerations, too few subjects have been studied to infer that molindone is more effective than placebo, as its similarity to a standard drug may be due to random error (or use of a treatment-resistant population). Despite this problem, these results are consistent in direction with the work of Clark and associates (35), who found molindone to be superior to placebo. In addition, molindone has been used in studies with both acute and chronic schizophrenic patients and in short- and long-term use and in two further comparison studies that (although they suffered from such methodologic limitations as inappropriate statistical analysis) also suggested that molindone was approximately equal to standard drug. Qualitatively, molindone produces roughly the same antipsychotic spectrum of action as the standard neuroleptics. It has the usual antipsychotic effect on schizophrenic symptoms, reducing their intensity with a similar time course, and it also has the prophylactic properties of the other antipsychotics. Whether there are minor differences in its spectrum of action will have to be determined in further studies. Molindone generally has the same range of side effects as the phenothiazines, i.e., extrapyramidal and autonomic side effects. Molindone does not inhibit the norepinephrine uptake pump; therefore, unlike chlorpromazine, it would not interfere with the hypotensive action of guanethidine. Molindone does not cause weight gain.

The other recently introduced drug is loxapine (Loxitane). This drug represents another new chemical category of antipsychotics; in its chemical structure, it is a dibenzoxapine. Since loxapine is active in dogs in antagonizing apomorphine-induced emesis, it may also be a dopamine receptor blocker.

The best evidence for the efficacy of loxapine has been provided by the clinical investigations of Clark and associates (41, 42); in this case, these researchers performed three carefully done, well-controlled, double-blind studies. The first compared loxapine, chlorpromazine, and placebo in chronic schizophrenics; the second compared loxapine, trifluoperazine, and placebo in newly admitted schizophrenics; according to a personal communication from M.L. Clark, the third study was a dose-responsive curve of loxapine. In all three studies loxapine was clearly (statistically significantly) superior to placebo in producing improvement in schizophrenia. In addition, Van Der Velde and Kiltie (43) found loxapine superior to placebo. Selman and associates presented data at a scientific exhibition at the 128th annual meeting of APA (Anaheim, Calif., May 5–9, 1975) that also suggested that loxapine was statistically superior to placebo.

Of the 15 studies comparing loxapine to standard neuroleptics, 12 found loxapine essentially equal to standard neuroleptics (41–53, and a personal communication from H. Kiltie). However, Shopsin and associates (45) found loxapine slightly inferior to standard neuroleptics, and Steinbook and associates (46) found it superior to standard neuroleptics in some but not all measures. Filho and associates (53) found loxapine more effective than thiothixene in younger but not older patients. Charlalmpous and associates (44) found loxapine less effective than thiothixene. Thus, several studies had some evidence that loxapine was slightly superior and many found it equal to a standard drug, but if the results of Charlalmpous and associates are also considered, loxapine is almost precisely equal to the standard drug in its antipsychotic action.

In this series of 15 studies of loxapine, about 600 patients were studied under controlled conditions. Making the assumption that the drug-placebo difference is such that it would explain 5% of the variance, according to the power analysis for sample size already discussed, this sample size would be sufficiently large to detect that the drug was not inferior or superior to standard drug with the probability of .01 for both type 1 and type 2 errors. This sample size is thus probably sufficient to detect a true difference between loxapine and the standard neuroleptic.

There is a final relevant methodologic point to be made here. In combining data from many studies comparing two similar drugs (new versus standard), the new drug will be better than the standard at some

times and inferior at other times by chance alone, generally to statistically insignificant degrees. In making such data combinations, if one fails to mention several of the studies in which a new drug is slightly inferior to standard, the comparison will tilt in a direction that makes the new drug appear slightly superior to standard. In other words, eliminating one tail of the distribution biases the sample to the opposite tail. Therefore, it is very important to include the study by Charlalmpous and associates (44) in any compilation, even though precise quantitation is not possible. Thus 5 out of 6 placebo-controlled trials and the combined data of 15 loxapine versus standard neuroleptic investigations indicate that loxapine is exactly equivalent to the standard neuroleptic and superior to placebo. Loxapine has the same range of side effects as other antipsychotics (extrapyramidal, autonomic, and sedative), and is similar in this respect to trifluoperazine, the reference antipsychotic used in many of the trials.

CONCLUSIONS

Since it is possible that a new class of antipsychotic agents would help a treatment-resistant patient, a trial of these agents as well as a high dose standard drug or intramuscular depot fluphenazine are possible therapies. In addition, there is merit in psychiatrists gaining clinical experience by using new agents for new patients so that the helpful properties of the compounds can be discovered.

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EDITORIAL

An Opportunity to Stimulate Self-Evaluation and Learning

SINCE 1969 the American Psychiatric Association, well ahead of many other medical specialty organizations, has been offering its members an opportunity to observe and improve their clinical skills through the Psychiatric Knowledge and Skills Self-Assessment Program (PKSAP). Two programs have been offered to APA members—the first in 1969 and the second in 1972. The third program will be available to the membership in April of this year.

Over 6,700 psychiatrists participated in PKSAP-I and more than 9,800 in PKSAP-II. Since PKSAP-II was offered, the entire medical profession has become alerted to the interwoven issues of lifetime education, government supervision, and the responsibility professional organizations must accept vis-à-vis the constant updating of knowledge by their members. Currently 22 medical associations are offering self-assessment programs to their memberships.

From lessons learned in the first two self-assessment programs an entirely new PKSAP has been formulated. Members of the development committee were chosen to represent all geographical areas and specialty concerns within APA; the area councils and district branch presidents recommended members, of whom 100 contributed to the development of the program. Also, the program has been evaluated for its relevance, clarity, and accuracy by a representative group of psychiatrists.

PKSAP-III has been developed as a learning experience for the individual psychiatrist that will allow him to assess his competence and application of knowledge in the present state of our field. It covers the widest possible spectrum of psychiatric concerns, including personality development and functioning, child psychiatry, diagnostic techniques, social and behavioral science data relevant to psychiatry, psychopharmacology, biological and somatic treatment, psychotherapy and psychoanalysis, group therapy techniques, psychiatric liaison concerns, and psychophysiological processes.

PKSAP-III includes 200 multiple-choice questions that cover a range of psychiatric concerns and levels of competence and 6 patient management problems that lead the participant through the clinical decisions necessary for handling the cases described. One of the 6 clinical cases is presented on cassette tape. To stimulate further learning by the participant, the answers to the items will include one or more literature references. The format of PKSAP-III is an attractive one that can be utilized conveniently by the individual psychiatrist as he or she wishes and in as much time as necessary.

As has been done successfully in the past, APA will maintain absolute confidentiality in regard to individual results. However, each participant will have an opportunity to compare his or her performance with others practicing in a similar area or subspecialty (e.g., psychotherapy, administrative psychiatry, and hospital psychiatry) through selected group evaluations. In addition to the report of scores and other feedback, the participant will receive a certificate acknowledging participation in the program.

It is the hope of the PKSAP Steering Committee that each member of the Association will expand his psychiatric knowledge and ability by participating in PKSAP-III. We also hope that this new program will be utilized by district branches and other psychiatric groups in imaginative ways to engage our membership in incentives toward continuing education, a critical concern for psychiatrists. Through the program the individual psychiatrist will be able to evaluate his personal strengths and weaknesses, and district branches and area councils will be able to develop more relevant and effective continuing education programs based on the broad trends that emerge from the program. With our participation in PKSAP-III, the principles of voluntarism and independence—so important to us as psychiatrists—can be furthered at the same time that we support a valuable program of continuing education that will enhance our knowledge of new developments and maintain the high standards of our specialty.

SIDNEY L. WERKMAN, M.D.

Dr. Werkman is Chairman of the PKSAP Steering Committee and Coordinator of Undergraduate Psychiatry, University of Colorado Medical Center, Denver.

The Educational Objectives of a Psychiatric Residency Program

BY JOEL YAGER, M.D., AND ROBERT O. PASNAU, M.D.

The authors present a statement of the specific educational objectives of their psychiatric residency program. This statement is designed to help residents keep in focus the central tasks of their training and to provide the faculty with an organizing matrix for curriculum development. Through such specific delineation of their objectives, residency programs can also contribute to a clarification of the specialty.

CONFUSION ABOUNDS ABOUT the proper role of the psychiatrist and the legitimate arena of psychiatric activity (1–3). Psychiatric residents are acutely aware of this state of affairs and sometimes have great difficulty in knowing where to focus their attention (4). Several authors (5–8) have traced some of the emotional problems of psychiatric residents to the lack of clear goals in their educational programs. To counteract the tendency toward professional identity diffusion and to help residents keep the central tasks of training in focus, we have set forth specific educational objectives for each area of the residency at the University of California, Los Angeles. In this way we have operationally defined what we believe to be the core of the psychiatric specialty as a body of skills and knowledge. Our residents have found the delineation of the program's expectations to be helpful, and training directors and residents in other programs have requested that our statement be made more widely available.

Core educational objectives have been developed for the first two years of the residency. In the third year residents are encouraged to pursue their individual areas of interest. During the core period residents are expected to become competent in skills and familiar with specific areas of knowledge. These areas of ability and expertise should continue to grow through subsequent training and experience. The list is not intended to be exhaustive or exclusive but to serve as a guideline for study and professional development; although it appears to be comprehensive, it is not meant to deter interest in other aspects of psychiatry.

Dr. Yager is Assistant Professor in Residence and Director of Residency Education, UCLA Neuropsychiatric Institute—Brentwood Veterans Administration Hospital Residency Program, and Dr. Pasnau is Associate Professor in Residence and Chief, Consultation-Liaison Service, Department of Psychiatry, School of Medicine, University of California, Los Angeles, Calif. 90024.

FIRST-YEAR EDUCATIONAL OBJECTIVES

Our first-year program consists of inpatient, day hospital, and emergency psychiatry services, as well as some outpatient and aftercare experiences in adult psychiatry. All residents have completed a prior internship. Seminars on basic psychiatry and the psychiatric literature are included in the curriculum.

Skills

By the end of the residents' first year of training we expect them to be able to do the following:

1. Conduct a comprehensive psychiatric interview.
2. Make use of appropriate laboratory examinations, psychological testing, and other consultations as indicated in the workup of psychological problems.
3. Conduct a family interview.
4. Make accurate psychiatric diagnoses and thorough appraisals of the presenting problems.
5. Formulate a treatment plan and implement it through personal effort and appropriate referral (triage and disposition).
6. Use all psychopharmacological agents well.
7. Conduct supportive therapy with increasing awareness of dynamic issues.
8. Have some understanding of the assumptions and values of dynamic, insight-oriented psychotherapy and begin to apply these principles in clinical work.
9. Become aware of their countertransference problems and personal idiosyncrasies as they influence interactions with patients and begin to learn to deal with them constructively.

10. Work harmoniously within a milieu together with nurses, social workers, psychiatric technicians, and other mental health personnel in information gathering, treatment planning and implementation, and cotherapy; simultaneously learn from others and contribute to their development.
11. Conduct an inpatient group therapy meeting.
12. Administer electroconvulsive therapy when required.

Knowledge

By the end of the residents' first year of training we expect them to be able to discuss the following:
1. The theory and nature of both individual and family interview techniques.
2. The nature of psychiatric data, including phenomenology and frames of reference.
3. Indications, values, and limitations of psychological testing.
4. Methods for distinguishing between organic and functional processes.
5. The differential diagnosis of psychiatric syndromes with appropriate criteria.
6. Evaluation and management of psychiatric emergencies.
7. Patients' rights, indications for involuntary psychiatric hospitalization, and other basic legal issues.
8. Indications and contraindications for full hospitalization, day hospitalization, family intervention, crisis intervention, supportive psychotherapy, behavior therapy, intensive long-term dynamic psychotherapy, group psychotherapy, and ECT.
9. Indications and contraindications for common psychopharmacological agents, as well as dosages, side effects, and drug interactions.
10. Alternative etiological explanations for the psychoses.
11. Basic psychoanalytic concepts.
12. Basic concepts of family organization and communication.
13. Basic principles of learning theories.
14. Basic concepts of group dynamics.
15. Basic concepts of social psychiatry and epidemiology, including concerns and treatment issues with specific ethnic, minority, and disadvantaged populations.
16. A perspective on the different psychotherapeutic schools of thought.
17. Neurobiological basic science, including genetics; biogenic amines; the anatomy, physiology, and pathology of the limbic system; sleep physiology and pathology; and memory.
18. How to critically evaluate the merits and limitations of scientific literature.

SECOND-YEAR EDUCATIONAL OBJECTIVES

The second-year program consists of assignments to consultation-liaison, child psychiatry, and adult outpatient services. Some patients are followed throughout the residency, and new cases are added as indicated. A weekly clinical neurology seminar covering pertinent neurological topics, a seminar in psychophysiological medicine, and a child psychiatry seminar are included in the curriculum. In addition, during the second year the resident continues to develop skills in evaluation and in brief and long-term treatment of individuals, couples, families, and groups. Increasing attention is paid to dynamic issues in psychotherapy and the immediate environment of the doctor-patient interaction including process, countertransference, specific techniques of intervention, and nonspecific effects of the psychotherapy process.

Consultation-Liaison

Skills. By the end of the residents' rotation on consultation-liaison psychiatry we expect them to be able to do the following:
1. Assess the specific intentions of the referring physician for any given request for consultation.
2. Conduct a comprehensive interview of a physically ill patient and a thorough assessment of each case, making appropriate use of information from the referring physician, other hospital personnel, the hospital chart, and the patient's family.
3. Construct a comprehensive formulation of the patient's problems that includes attention to the life setting and specific circumstances leading to the illness and the request for consultation; the present and past significance of the illness and problems, including primary impact and secondary effects on the patient and significant others; psychosomatic and somatopsychic considerations; and the specific influences of the patient's personality on the manifestations of the illness and illness behavior.
4. Define and implement appropriate interventions, addressing the problems that initiated the request for consultation.
5. Undertake the psychotherapeutic treatment, for an extended period of time, of at least one dying patient and one patient with a psychophysiological illness.
6. Effectively communicate with the referring physician, both verbally and through concise, nontechnically worded notes, about the assessment, offering practical suggestions for management. These suggestions may include how the physician might interact with and counsel the patient and his family; ward management by the hospital staff; other environmental manipulation; the appropriate use of psychopharmacological agents in the physically ill; and further recommendations for referral, disposition, and treatment.
7. Develop a good liaison relationship with physicians, nurses, social workers, and the other staff on the service to which the resident is assigned and, using consultation models of community psychiatry, help assess and meet the needs of the staff as mental health educator, facilitator, and resource person.

Knowledge. By the end of the residents' rotation on consultation-liaison psychiatry we expect them to be able to discuss the following:

1. Models of consultation.
2. Social psychology of patient role behavior.
3. Psychological aspects of stress, coping and adaptation, including the dying process, bereavement, transition states, and life crises.
4. Psychiatric interventions in liaison settings.
5. Psychosomatic psychopharmacology.
6. Specific syndromes of special importance in liaison settings, including conversion reactions, pain, gross stress reactions, postpartum psychiatric problems, postoperative psychoses, psychoses in the intensive care unit, psychiatric evaluations for special medical procedures, compensation neurosis, and malingering.
7. Theories of psychosomatic contributions to the pathogenesis of illness.

Child Psychiatry

Skills. By the end of the residents' rotation in child psychiatry we expect them to be able to do the following:

1. Conduct a thorough evaluation of children and their families, properly assessing the nature and significance of presented problems.
2. Appropriately use psychological and physiological assessment techniques to augment the clinical evaluation.
3. Work as a team with psychologists, social workers, and other professionals to obtain a complete evaluation and to formulate appropriate interventions.
4. Appreciate the dynamic and developmental aspects of disturbing behavior in children and adolescents and the extent to which those behaviors are responsive or refractory to environmental contingencies.

Knowledge. By the end of the residents' rotation in child psychiatry we expect them to be able to discuss the following:

1. Principles of the evaluation of children and their families.
2. Major theories of child development, including psychodynamic, cognitive, and biological issues.
3. Common psychiatric problems of childhood and current concepts in the classification of childhood disorders.
4. Mental retardation, including evaluation and treatment planning.
5. Adolescence, with a focus on personality developmental issues.
6. Principles of intervention in child and adolescent psychiatry.

Adult Psychiatry

Skills. By the end of the second year we expect the residents to be able to treat individuals, couples, families, and groups as indicated, with supervision.

Knowledge. By the end of the residents' second year of training we expect them to be able to discuss the following:

1. Alternative theories of therapy, including basic assumptions, indications, and contraindications for the use of different psychotherapeutic techniques for brief or long periods of time, as well as something about available outcome evidence.
2. Characteristics of and explanations for neurotic, characterological, and borderline conditions.
3. Psychiatrically pertinent aspects of neurology.

DISCUSSION

The educational objectives we have outlined are not meant to be static. They are intended to be rethought, updated, and altered, reflecting the evolving perception of the psychiatrist's role by the faculty and residents. Specific educational objectives at other residency programs may differ from ours in substance or in emphasis. Enelow and Weinstein (9) have recently published a report on the detailed goals and objectives for psychiatric residency education. Their objectives and ours, developed independently of one another, both stress mastery of specific areas of knowledge and skill. There are many similarities—and differences—between these two sets of objectives. Such diversity, reflecting psychiatry's broad scope, is commendable. Each program can contribute to a clarification of the specialty by defining its own educational objectives as precisely as possible.

Statements of educational objectives offer advantages to applicants, residents, and faculty. We include the statement in the descriptive material sent to applicants, many of whom have commented favorably that such statements can provide applicants with additional means by which to compare and contrast training programs. The statement is distributed at the beginning of each year to new residents and those continuing in the program as part of the orientation manual and can be referred to when the resident starts a new rotation.

For the faculty, the definition of educational objectives provides an organizing matrix around which both the didactic and clinical experience in the curriculum can be constructed. Seminars and clinical experiences that compete for limited amounts of resident time can be viewed against their potential contribution to the objectives. These objectives also have proven to be useful in the development of assessment procedures for the residents and for the program as a whole.

By enumerating objectives for specific skill and knowledge areas for psychiatric residency education and by using these as a basis for discussion in the profession, residency programs may help to determine the viability and definition of our specialty in the future.

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The Effect of Marijuana on Carbohydrate Metabolism

BY M. ALAN PERMUTT, M.D., DONALD W. GOODWIN, M.D., ROBERT SCHWIN, M.D., AND SHIRLEY Y. HILL, PH.D.

The authors observed the effect of marijuana on carbohydrate metabolism in fed and fasting states in chronic marijuana users. They found no hypoglycemia in 7 patients who were given marijuana after fasting for 24–72 hours. They also found no significant difference in carbohydrate tolerance and no hypoglycemia during an oral glucose tolerance test in 10 patients who smoked placebo or marijuana on alternate days. They conclude that marijuana has no effect on carbohydrate metabolism in the fed or the fasted state in well-nourished chronic marijuana users.

BECAUSE OF THE ASSOCIATION between hunger and marijuana smoking in man, early investigators looked for a hypoglycemic effect of the drug; they found none (1–3). These studies did not specify how long before the marijuana was administered the volunteer subjects had eaten. The concentration of glucose in the blood is maintained in sufficient quantities to prevent hypoglycemia predominantly by gluconeogenesis from the liver after glycogen stores have been used up (4). After a period of starvation liver glycogen becomes depleted, and any substance that impairs gluconeogenesis, e.g., alcohol (5), can cause hypoglycemia. An effect of marijuana on gluconeogenesis might be more readily apparent in subjects who had fasted; the effect might be to produce hypoglycemia. For this reason we observed a sample of marijuana users who had fasted for 24–72 hours before administration of marijuana for subsequent hypoglycemia.

The effect of marijuana on carbohydrate metabolism after eating has also not been evaluated. If marijuana potentiates insulin secretion or action, it might produce reactive hypoglycemia 2–5 hours after a meal. We therefore studied the effects of marijuana on carbohydrate tolerance during a 5-hour oral glucose tolerance test.

METHOD

For this study 10 nonobese male volunteers ranging in age from 23 to 31 who had smoked marijuana at least once a week for one year were admitted to the Clinical Research Center of Washington University Medical School the night before the first of our tests was made. These patients fasted overnight. On the morning of day 1 of the study a fasting plasma for glucose and insulin was drawn from each patient, then either 1 g of marijuana containing 1.5% Δ-9-tetrahydrocannabinol

Drs. Permutt, Goodwin, and Hill are with the Washington University School of Medicine, St. Louis, Mo. Dr. Permutt is Assistant Professor, Department of Medicine, 660 South Euclid, St. Louis, Mo. 63110, Dr. Goodwin is Professor of Psychiatry, and Dr. Hill is Research Assistant Professor of Psychiatry. Dr. Permutt is also a Medical Investigator, Howard Hughes Medical Institute, St. Louis, Mo. Dr. Schwin was with the Department of Psychiatry, Stanford University, Palo Alto, Calif., at the time this research was done.

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The marijuana and placebo for this study were supplied by the National Institute of Mental Health, Alcohol, Drug Abuse, and Mental Health Administration, Rockville, Md.

TABLE 1
Effect of Placebo Versus Marijuana on Plasma Glucose and Plasma Insulin Levels of 10 Chronic Marijuana Users During a Five-Hour Glucose Tolerance Test

Item	Time of Glucose Tolerance Test						
	Start of Test	One-Half Hour	One Hour	Two Hours	Three Hours	Four Hours	Five Hours
	Mean±SEM	Mean±SEM	Mean±SEM	Mean±SEM	Mean±SEM	Mean±SEM	Mean±SEM
Plasma glucose (mg/100 ml)							
Placebo	92 2	156 12	146 14	120 9	90 7	79 5	86 3
Marijuana	89 2	137 9	140 8	122 6	92 6	74 2	86 3
Plasma insulin (μU/ml)							
Placebo	7 1	63 10	70 10	57 14	27 8	7 1	5 1
Marijuana	8 2	64 17	71 16	57 13	26 6	9 1	7 1

(THC) or marijuana placebo with THC removed was administered by smoke inhalation over a period of 15–30 minutes. The smoke was delivered by spirometer, which minimized the loss of smoke into the atmosphere (6). A standard 100 g oral glucose tolerance test was then performed with samples of blood removed from indwelling intravenous needles at the times indicated in table 1. On day 2 at 8 a.m. a repeat oral glucose tolerance test was performed with either placebo or marijuana, alternating marijuana-placebo sequences with placebo-marijuana sequences. Following the second glucose tolerance test, 7 patients were given nothing by mouth for 24–72 hours except water. At 8 a.m. on day 3, marijuana was again administered, and the patients continued to fast for 6–8 hours. Plasma samples for glucose and insulin were obtained at the times indicated in table 2. Plasma glucose was measured by Technicon Auto Analyser, and plasma insulin was measured by double antibody radioimmunoassay with human insulin standards (7).

RESULTS

Administration of marijuana to 6 volunteer chronic marijuana users in quantities sufficient to produce significant behavioral effects had no hypoglycemic effect after 24 hours of starvation (see table 2). One volunteer (patient 7) was starved for 72 hours before administration of marijuana; he also maintained normoglycemia 6 hours later (see table 2). Plasma insulin was appropriately low in all of the patients who had fasted, and marijuana did not stimulate insulin release.

The glucose tolerance of 10 chronic marijuana users was within normal limits following oral ingestion of glucose after smoking placebo (see table 1). Their carbohydrate tolerance was also normal after marijuana was smoked (see table 1). There was no significant difference between the peak plasma glucose level, time of the peak glucose level, low glucose level, total insulin secreted, peak insulin secreted, or time of peak insulin secretion, nor did hypoglycemia occur in any of the volunteers following ingestion of marijuana. We conclude that marijuana smoking had no effect on carbohydrate metabolism in the fed or fasted state in well-nourished chronic marijuana users.

TABLE 2
Effect of Δ-9-THC on Plasma Glucose and Plasma Insulin Levels After a 24-Hour Fast in 6 Patients and a 72-Hour Fast in Patient 7

Item	Number of Hours After Smoking Δ-9-THC					
	0	2	4	6	7	8
Patient 1						
Plasma glucose (mg/100 ml)	93	114	95	97	88	90
Plasma insulin (μU/ml)	7	5	7	6	4	9
Patient 2						
Plasma glucose (mg/100 ml)	80	95	82	76	—	—
Plasma insulin (μU/ml)	3	6	5	4	—	—
Patient 3						
Plasma glucose (mg/100 ml)	89	93	90	94	—	—
Plasma insulin (μU/ml)	6	7	5	4	—	—
Patient 4						
Plasma glucose (mg/100 ml)	102	98	91	90	—	86
Plasma insulin (μU/ml)	6	5	7	4	—	3
Patient 5						
Plasma glucose (mg/100 ml)	85	88	91	—	89	—
Plasma insulin (μU/ml)	5	6	5	—	5	—
Patient 6						
Plasma glucose (mg/100 ml)	91	92	86	92	—	86
Plasma insulin (μU/ml)	7	9	5	5	—	8
Patient 7						
Plasma glucose (mg/100 ml)	—	—	—	90	—	—
Plasma insulin (μU/ml)	—	—	—	<1	—	—

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Joint Admission as a Treatment Modality for Problem Drinkers: A Case Report

BY THOMAS J. PAOLINO, JR., M.D., AND BARBARA S. MCCRADY, PH.D.

Joint hospital admission of a married alcoholic and his/her spouse can facilitate treatment of the problem drinker. A case report illustrates the advantages of this approach in giving the staff an opportunity to observe and provide the couple with feedback about their patterns of interaction and to provide specific therapeutic interventions during the joint admission.

THE JOINT ADMISSION described in this paper is a treatment modality that maximally involves the spouse when the alcohol misuser's problems warrant full-time psychiatric hospitalization. Most, if not all, problem drinkers have painfully difficult relationships with significant people in their lives. Spouses of actively drinking alcoholics usually experience major psychological problems (1–6). While the alcoholic's behavior may adversely affect the spouse, the spouse may unknowingly behave in certain ways that either trigger drinking episodes or help to maintain problem drinking (7). Caring for the drunk alcoholic, covering for him at work, etc., may actually reinforce his drinking. Nagging may be an antecedent to drinking bouts. The misuse of alcohol may also indicate the presence of serious conflicts in the marital relationship (8). Therapists therefore have concluded that the spouse should be significantly involved in the problem drinker's treatment program (9, 10).

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Dr. Paolino is Director and Dr. McCrady is Psychologist, In-Patient Division, Butler Hospital, 333 Grotto Ave., Providence, R.I. 02906. Dr. Paolino is also Assistant Professor of Psychiatry and Dr. McCrady is also Instructor in Psychiatry, Department of Psychiatry and Human Behavior, Brown University, Providence, R.I.

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The earliest reports of joint admissions were of disturbed mothers and healthy infants (11). There have been a few reports (12, 13) of the joint admission of severely disturbed psychiatric patients and their normal families, but alcoholism was not mentioned specifically as a problem. Some investigators (8, 14) have conducted research projects in which father–son and brother–brother alcoholic pairs were admitted to research wards in order to permit observation of their interactions. Although alcoholics and their families have been jointly admitted to residential centers (15), to our knowledge there have been no published reports on joint admissions of alcoholics and their spouses to a psychiatric hospital or a medical facility for acute disorders.[1]

The Alcoholism Joint Admission Project is a clinical-research project that was established at Butler Hospital in July 1974. It provides for the admission of both the alcoholic and spouse, who share a bedroom on a community-oriented ward that includes patients of all diagnoses and all ages over 12. The spouse, officially a "guest" of the hospital, is present on the ward as much as possible but continues in his/her outside job. The spouse participates in all relevant ward activities, including daily ward community meetings, treatment team meetings, and supportive occupational and recreational therapy, and talks with ward staff and patients. The patient and spouse also participate in group therapy, which continues after discharge.

The goals of the joint admission are as follows:

1. To give the staff opportunity to observe the couple's interactions.
2. To provide comprehensive feedback to the couple about their patterns of interacting.
3. To integrate the spouse into the milieu so that the spouse has the same opportunity as the problem drinker to experience the closeness and caring of the unit.

[1]After this manuscript was prepared, Steinglass and associates (16) presented a paper on an in-hospital treatment program for alcoholic couples.

CASE REPORT

At the time of admission in February 1975 Mrs. B was a 28-year-old housewife and her husband a 29-year-old crane operator; they have a daughter who was then 4 years old. Mrs. B had had no previous psychiatric treatment before her admission because of progressive alcohol misuse, depression, marital conflict, and severe suicidal thoughts.

Mrs. B, a tall, attractive woman, described in detail a deprived, lonely childhood and adolescence, during which she rarely saw her alcoholic parents. Her father had left home when she was 5 years old, her mother had remarried, and she was raised by her grandmother, who died suddenly 10 years earlier. Mrs. B perceived her grandmother's death as a form of rejection and abandonment and began to abuse alcohol, drinking an average of 12 cans of beer or an equivalent amount of liquor each evening. From ages 18 to 22 her life was unsettled. After graduating from high school she wandered from place to place until the age of 22, when she met her husband. They married 1 year later and have never been separated.

Mrs. B was apprehensive about failing and feared rejection. She had experienced failure in numerous clerical jobs, which again precipitated excessive drinking. Fear of rejection was a constant theme throughout her life and a major antecedent to her excessive drinking.

Mr. B was outgoing, sociable, and had many friends, while Mrs. B had little social involvement with other people. Mrs. B saw this difference in social behavior as a major source of conflict and feelings of inadequacy.

Mr. B, who had 4 brothers, had no personal or family history of psychiatric illness or alcohol abuse.

On admission Mrs. B was cooperative and neatly dressed. She frequently hesitated when discussing emotionally charged material, but her speech was coherent and relevant. Her mood was one of marked sadness and apprehension but was appropriate to her thought content. There were no manifestations of thought disorder or any signs of organic disturbance. She displayed average self-understanding and a firm motivation for treatment of her alcoholism and depression.

Findings from a physical examination, medical history, and laboratory tests showed Mrs. B to be in good health. However, her scores on the Psychological Screening Inventory (17), Multiple Affect Adjective Check List (18), Quantity Frequency Index (19), and the Alcohol Impairment Index (20) were all abnormal. Mr. B's test scores were within the normal range.

During the first week of inpatient treatment Mrs. B lived in the hospital without her spouse. During that time she experienced mild alcohol withdrawal symptoms and developed a strong therapeutic alliance with the ward staff. She often spoke of her marriage, her feelings of rejection, and her compulsion to drink, but she had difficulty giving specific details about her husband's "rejections." This difficulty was clinically important, since, as will be shown later, the joint admission served to provide the ward staff with specific behavioral data clarifying this issue.

Mr. B moved into the hospital during the second week of treatment. The following are some meaningful events observed by the staff.

1. Mr. B threatened to divorce Mrs. B if she became "mentally sick" again. When Mr. B made this threat, the staff immediately interceded and encouraged the couple to talk more about the issue, with the result that there was a significant reduction in the tension surrounding this aspect of Mr. B's thinking and behavior.

2. Mrs. B was extremely jealous and constantly feared that Mr. B would leave her for another woman, when in fact he had always been faithful. Several times, Mrs. B became very distraught when she saw her husband innocently talking with female patients. On these occasions she stormed off to her room, and the staff immediately brought the couple together to discuss the problem. Mrs. B said she felt rejected and that if she were home, she would be turning to alcohol. Mr. B appreciated, perhaps for the first time, the intensity of his wife's insecurity and jealousy. Mrs. B learned how she was misinterpreting her husband's friendliness as infidelity.

3. Some nights Mr. B spent up to 5 hours watching television on the ward and "tuning out" his wife. Again, Mrs. B interpreted this as rejection, got very angry, and told the ward staff that this was another time when she would be drinking excessively if she were not in the hospital. The staff met with the couple as soon as these issues arose. Mr. B became more aware that his excessive television viewing was not only a way of avoiding interaction with his wife but also an antecedent to her alcohol abuse. Mrs. B learned that watching television was not necessarily tantamount to rejection.

4. Mrs. B felt rejected almost every time that Mr. B was not available to listen to and meet her demands. She stated that were she not in the hospital, these would also be times when she would drink to excess. The staff defined and clarified the interactions that were rejecting in contrast to the behavioral patterns of Mr. B that Mrs. B misinterpreted as rejection.

5. Most of the time Mrs. B was shy and timid toward her husband and other people, even when she was angry. The ward staff taught her to be more assertive, and eventually she was able to confront her husband and other people with her feelings without the assistance of staff members.

6. Mrs. B frequently awakened her husband in the middle of the night to "tell him how I was feeling." At these times he immediately sought the staff's help, and they promptly indicated to Mrs. B that she was being unreasonably demanding of her husband's attention and that this behavior tended to drive him away from her. Eventually, with the staff's help, the couple made some agreements to change their behavior. For example, Mr. B agreed to watch television for no more than 2 hours each night if Mrs. B would agree not to awaken him during the night.

DISCUSSION

Mrs. B improved significantly and was discharged to outpatient care after 3 weeks of the joint admission. The following clinical achievements seemed related to the joint admission.

1. The couple used the ward staff to give them continuous feedback about their relationship and how certain aspects of that relationship were directly related to problem drinking.

2. By being in the protective ward environment the couple said and felt things that might have been too frightening outside the hospital. They could express angry feelings as they occurred without worrying as much about losing control of their anger, since, obviously, the ward staff would not allow destructive behavior.

3. Mr. B was able to see that his wife, as well as oth-

er people with serious emotional and behavioral problems, could be helped to overcome these difficulties.

4. The couple were able to remain together and to support each other during the most critical part of Mrs. B's illness.

5. The joint admission made discharge less difficult and shortened the length of hospitalization, since Mr. B was less afraid and more willing to accept and deal with Mrs. B's psychiatric problems, even if they were not completely resolved.

This case history suggests that the joint admission could be a clinically effective treatment modality, replicable relatively easily at other psychiatric inpatient units. However, a more definitive statement on its effectiveness must await the results of an evaluation currently in progress.

At first we conceptualized the joint admission as a means of thoroughly observing the couple in order to better understand the dynamics of their relationship. Experience with 18 joint admissions has led us to conclude that perhaps couples could further benefit from a more structured program focusing on specific behavioral interactions that serve as antecedents to drinking or that actually reinforce drinking behavior.

Some research findings (21–23) have suggested that an operant reinforcement approach can increase the likelihood of successful treatment outcome with couples and with problem drinkers.

However, one of the difficulties frequently encountered in using behavioral approaches to the treatment of couples is the problem of "homework." Although a couple may often be acutely uncomfortable and want to achieve certain behavioral changes, initial practice of this behavioral change is usually difficult for the following reasons.

1. The couple often realize that they do not understand the instructions for homework.

2. The couple do not have the skills to initiate the new behavior.

3. There are both significant reinforcers for the old, undesired behavior and avoidance responses for the new, unknown behavior.

One possible way to facilitate practice and therefore behavioral change would be to initiate treatment within a structured inpatient ward environment that would continuously provide instructions, feedback, and contingent reinforcement for behavior. In treating a couple when one spouse is a hospitalized alcohol abuser, such contingent reinforcement, feedback, and supervised practice would be facilitated by a joint admission.

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Chronic Hashish Use and Mental Disorder

BY COSTAS STEFANIS, M.D., ARIS LIAKOS, M.D., JOHN BOULOUGOURIS, M.D., MAX FINK, M.D., AND ALFRED M. FREEDMAN, M.D.

The authors compared 47 long-term users of hashish with a control group of 40 subjects matched for age, ethnic origin, education, etc., in order to determine whether they differed significantly on psychiatric, physical health, and demographic variables. There was a significantly higher incidence of personality disorders, unemployment, and prison sentences in the group of chronic users. However, in contrast to the findings of other researchers, the hashish users did not have organic psychoses, nor did they differ from control subjects in neurologic signs or EEG and echo encephalogram patterns.

THE CONTROVERSY concerning the long-term effects of chronic cannabis use remains unresolved despite numerous publications on the subject (1–3). The long-term behavioral and mental effects of cannabis use are particularly in dispute (4–7). The need for more systematic and controlled studies has been noted (8); to meet this need, we carried out an intensive study of a population of chronic hashish users in Greece. Our study consisted of 1) population identification and comparison with a control sample, 2) experimental assessment of the effects of different strengths of cannabis preparations, and 3) assessment of withdrawal effects. In this report, we will present the results of the population study, which investigated the incidence of mental disorders and the personal and family characteristics of the chronic cannabis users.

Revised version of a paper presented at the 127th annual meeting of the American Psychiatric Association, Detroit, Mich., May 6-10, 1974.

Dr. Stefanis is Professor and Chairman and Drs. Liakos and Bouloungouris are Professors, Department of Psychiatry, University of Athens School of Medicine, Athens, Greece. When this work was done, Dr. Fink was Professor, Department of Psychiatry, New York Medical College; he is now Professor of Psychiatry, State University of New York at Stony Brook Health Sciences Center, Stony Brook, N.Y. 11794. Dr. Freedman is Professor and Chairman, Department of Psychiatry, New York Medical College. Address reprint requests to Dr. Fink.

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METHOD

Data were obtained by psychiatric examinations and social history inquiries, supplemented by physical and neurologic examinations, psychological assessments, and EEG recordings. The social history inquiry was based on structured interviews with the subjects and family members; the psychiatric assessment included a psychiatric history and a mental status examination following the structured format developed by Mayer-Gross and associates (9). Each case was discussed by the psychiatric team. Psychiatric records were obtained for subjects who had had psychiatric treatment, home visits were made, and information was gathered from local civil authorities (town hall and police). The psychiatric diagnosis was made by consensus, using *DSM-II* (10) criteria.[1]

SUBJECTS

Hashish Users

Our sample of cannabis users consisted of individuals who had used cannabis for more than 10 years and were using it at the time of investigation. Individuals who used other addictive substances, except tobacco and alcohol (social use), were excluded from the study. This included those who were reported to be drunk regularly, who were dependent on alcohol, or whose drinking interfered with their work performance. The presence of incapacitating physical illness or neurologic disease was also a basis for exclusion.

Of 60 cannabis users examined initially, 13 were excluded: 4 for heroin use, 3 for alcoholism, 1 for use of LSD, 1 for inadequate current use of cannabis, and 4 for being above the cut-off age of 58 years. The final sample included 47 male subjects. Of these, 19 were referred by Prof. C. Miras and were part of a pool of chronic cannabis users he had studied previously (11). The rest were recruited by our social services from the community.

The mean age at first use of cannabis was 17 years. Subjects had smoked cannabis an average of three times a day almost every day for 23 years, and they consumed an average of 5 g of hashish each day. An

[1]The study design made blind psychiatric assessment impractical. If we had insisted on blind and structured psychiatric procedures, we would have missed much of the information vital to the purpose of the study.

analysis of hashish described by subjects as being of average strength revealed a 4 to 5% THC content, which corresponds to a mean intake of 200 mg of THC per day.

Control Group

Control subjects were selected by matching requirements in terms of age, ethnic origin, education, place of birth, and family structure. Other criteria included 1) never having smoked cannabis, and 2) smoking tobacco (more than 10 cigarettes a day). Exclusion criteria for alcoholism, illness, etc., were identical with those for hashish users.

An initial 64 subjects were either 1) recruited by social workers from the community in the same fashion as the hashish users, or 2) recommended to the study team by the hashish users as being from similar social circumstances and known not to use hashish. Of the 64, 24 were excluded: 8 for having smoked cannabis at least once, 5 for alcoholism, 4 for being above the cutoff age, 3 for not being tobacco smokers, 2 for central nervous system diseases, 1 for having used heroin, and 1 for having a sibling who was a cannabis user. The final control group consisted of 40 male subjects, 31 derived from community sources and 9 referred by hashish users originally studied by Miras (11).

RESULTS

Personal History Data

The groups were matched for age, education, ethnic origin, place of birth, family history for mental illness, and type of upbringing. There were no differences between the groups in the incidence of neurotic traits during childhood, separation from parents before the 15th year of age, or marital status. Differences were found, however, in the following personal history data.

1. Previous psychiatric treatment: Nine hashish users (19%) had previous psychiatric treatment compared with 2 (5%) of the controls ($\chi^2=3.94$, $p<.05$).

2. Regular military service: Only 26 of the 47 cannabis users (55%) had served regularly in the Army compared with 38 of 40 control subjects (95%) ($\chi^2=17.49$, $p<.001$). Military service is compulsory in Greece, but cannabis use is cause for exemption from service; this difference may thus be related to military criteria. However, personality disorder and psychotic illness are also reasons for exemption from military service.

3. Imprisonment: Twenty-five cannabis users (53%) had been in prison for violations of cannabis laws, compared with 1 (2%) of the controls, who was sentenced for selling hashish ($\chi^2=26.49$, $p<.001$). Additionally, 29 cannabis users (61%), were sentenced for reasons other than cannabis law violations compared with 10 (26%) of the control subjects ($\chi^2=11.76$, $p<.001$). There was no relationship between the amount of hashish smoked and imprisonment for reasons other than cannabis law violations. This relationship was investigated by dividing cannabis users into heavy and moderate smokers on the basis of mean amount of cannabis smoked.

4. Work record: While the two groups did not differ in number of occupations or job changes, 20 cannabis users (42%) were unemployed at the time of the study compared with 6 control subjects (15%) ($\chi^2=7.82$, $p<.01$). Similarly, 12 cannabis users were skilled workers (25%) in comparison with 25 (62%) of the controls ($\chi^2=12.08$, $p<.001$).

Mental Disorders

In psychiatric assessments, psychopathology of some type was defined in 18 of 47 cannabis users (38%) and in 7 of 40 controls (17%) ($\chi^2=4.55$, $p<.05$). Diagnoses included personality disorder, neurosis, and paranoid schizophrenia (see table 1). No subjects exhibited organic psychoses.

The diagnosis of personality disorder was made in 12 of 47 hashish users (25%) versus 3 (7%) of the controls ($\chi^2=4.92$, $p<.05$). Five users were further characterized as manifesting a disorder of the antisocial type, while no control subjects were so diagnosed ($\chi^2=4.51$, $p<.05$). The remaining subjects were further classified among other diagnostic subcategories (paranoid, hysterical, inadequate, etc.). Three cannabis users (6%) were classed as having neuroses, as were 3 control subjects (7%). One control subject was diagnosed as clinically depressed, with no such diagnosis in the cannabis group. Three users (6%) were diagnosed as suffering from paranoid schizophrenia as compared with none in the control sample. On examination, they exhibited loosely organized paranoid delusions unrelated to police persecution for possession or smoking hashish. They neglected themselves and lacked insight. One reported visual and auditory hallucinations. Neurotic traits were prominent in their personalities before they started using cannabis. They started smoking hashish in their teens and the onset of psychosis was many years later, except in the case of 1 subject, for whom possible psychotic symptoms were reported at the age

TABLE 1
Incidence of Psychiatric Disorders

Type of Disorder	Hashish Users (N=47) N	%	Control Subjects (N=40) N	%	Chi-Square Value	Significance
Personality disorders	12	25.53	3	7.5	4.92	p<.05
Antisocial type	5	10.63	0	0.0	4.51	p<.05
Other	7	14.98	3	7.5	1.16	n.s.
Neurosis	3	6.38	3	7.5	0.04	n.s.
Depressive illness	0	0.00	1	2.5	0.00	n.s.
Schizophrenia						
Paranoid type	3	6.38	0	0.0	0.00	n.s.
Total	18	38.28	7	17.50	4.56	p<.05

of 14. Two of these 3 users had had prior psychiatric inpatient and outpatient treatment. A positive family history of mental illness was recorded for 2 of the 3.

As our hashish user and control samples included subjects recommended by Prof. Miras, a question of sample bias may be raised, since Prof. Miras has described his subjects as psychopathic (11). However, there was no difference in distribution of mental states between the portion of our sample recommended by Prof. Miras and that recruited from the community.

DISCUSSION

This study indicates a high incidence of psychopathology, predominantly the antisocial type of personality disorder, in a population of chronic hashish users. The subjects, who started smoking cannabis at a young age, were examined after an average of 23 years of hashish use. It is not possible to determine whether their personality structure was the result of their hashish use or whether their preexisting personality was conducive to the continued use of hashish in a culture where its use was proscribed.

It is possible that the absence of any diagnosis of organic psychosis in the hashish users may reflect a selection factor, since the subjects were recruited from the community. However, given the number of years these subjects had used hashish and the large amounts they smoked daily, it is clear that a toxic or organic psychosis is not a usual accompaniment of high-dose, chronic hashish use. Although pneumoencephalography was not carried out, extensive neurologic examinations and laboratory tests (EEG and echo encephalogram) were done in both the users and control subjects; the two samples were indistinguishable in the clinical and laboratory signs of organic mental syndrome (12).

These data do not confirm the reports of cerebral atrophy in cannabis users by Campbell and associates (13) or of organic dysfunction by Kolansky and Moore (14). In both of these studies, the users were younger and reported significantly less use of cannabis of much lower potency for much shorter time periods than the hashish users in the present study. If the clinical observations of these researchers were the result of the subjects' use of cannabis, we would expect to find evidence of cerebral dysfunction in hashish users and differences in the neurologic tests between the chronic hashish users and the control subjects. Because we failed to find such differences, we believe that the observations of Kolansky and Moore (14) and Campbell (13) are not the result of cannabis use.

Some authors (4, 6, 14) have suggested that chronic hashish use induces a specific type of psychosis. The 3 hashish users in our study who were diagnosed as psychotic and classified as paranoid schizophrenics did not exhibit clinical features that could distinguish their condition from schizophrenia; therefore, no conclusions concerning the association of this type of psychosis and chronic cannabis use can be made.

We observed employment differences in the hashish users, both in their employment rate at the time of examination and the quality of the jobs. Although these observations could be interpreted as a facet of an amotivational syndrome, other circumstances such as the prevalence of personality disorder, prison sentences, and the stigma related to hashish smoking may have contributed to this difference (15).

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Mania and Bromism: A Case Report and a Look to the Future

BY A. JOSEPH SAYED, M.D.

Mania has not been reported as a manifestation of bromism. This paper describes a manic patient who had an elevated serum bromide level. The author suggests that the possibility of bromide-induced mania should be considered when patients who manifest manic symptoms are evaluated. The implications of the bromide–mania relationship for a better understanding of manic-depressive illness are also discussed.

ALTHOUGH MANY ALTERATIONS in mental function due to bromism have been observed, previous reports have not mentioned the association of mania and bromism. Levin (1) listed four bromide "psychoses": simple bromide intoxication, delirium, hallucinosis, and transitory schizophrenia associated with paranoid symptoms. Previous case reports cite confusion, dementia, depression, agitation (2), disorientation, and paranoid delusions (3) as mental symptoms of patients with bromism. Hoff (4) included sleep disturbances, difficulty in comprehension, exaggerated fears, and visual hallucinations in his list of mental aberrations due to bromide ingestion.

The patient who is the subject of this report presented with typical manic behavior and had an elevated serum bromide level.

CASE REPORT

A 23-year-old man was admitted to the psychiatry service of Cincinnati General Hospital after his second visit to the emergency unit in 2 days. His first visit was prompted when he undressed in front of his mother and attempted to bathe in a wash basin in the family home; he claimed he wanted to bathe because he was dirty after picking tomatoes. He became agitated when his father attempted to restrain him and was brought to the emergency unit, but he walked out before he could be admitted. He was returned the following day. The patient expressed paranoid delusions to the staff of the emergency unit, who felt he was having a "psychotic reaction."

Dr. Sayed is a resident in the Department of Psychiatry, University of Cincinnati College of Medicine, 231 Bethesda Ave., Cincinnati, Ohio 45267.

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His parents, with whom the patient lived, gave a history of the patient's increased activity over the preceding 2 months. He had occasionally been eating five or six meals a day, often leaving home at night to purchase additional meals. He regularly drank a carton of soft drinks each evening and ate a candy bar in one mouthful. He sometimes worked two shifts consecutively—one for pay and another "for free." His parents stated that he had been going for days at a time without sleep.

For recreation the patient would bowl 40 or 50 games at a time, often resetting the pins if they were not all knocked down with the first ball. He had squandered his savings. Having recently become interested in girls, he joined a dating service that cost $500; he borrowed the money to pay for it.

The patient did not have a history of psychiatric illness or similar behavior. He denied using unauthorized drugs. There was no family history of manic behavior, although his mother was being treated with an antidepressant.

Upon examination at the time of his admission, the patient spoke with much difficulty at times. He stated that he was "retarded" and had trouble getting his words and ideas across. He was occasionally boisterous. He stated that a goal of hospitalization was to read better, "which would make me a better worker to help the factory, help the country, and help the world." He said he wanted to write the President a letter.

A neurological examination at the time of his admission was entirely normal. The patient was oriented and of clear consciousness. He denied hallucinations and his memory was not impaired. However, the patient's intelligence appeared to be markedly below normal; his reading ability was poor and his fund of general information low.

Initial laboratory data were normal except that the serum bromide level was 32.5 mg/100 ml (normal=less than 1.5 mg/100 ml). The toxicology laboratory verified this information. A serum bromide determination was made because the patient had a history of bizarre behavior, paranoid delusions, and an acneiform rash. His parents and employer were unable to supply information about where he might have obtained bromide. Urine toxicological screen was negative, and an EEG was normal.

During the first part of his hospital stay the patient would arise early and empty garbage cans and perform other chores on the ward although he had not been requested to do so. His speech remained pressured and

there were two episodes of marked irritability.

The patient was treated with 400 mg of chlorpromazine per day; a maximum daily dosage of 700 mg of chlorpromazine was required briefly. His verbal and motor behavior soon returned to normal levels. A serum specimen drawn 6 days later was negative for bromide. His chlorpromazine was decreased and then eliminated after he was discharged to outpatient treatment.

Two months after his discharge the patient was doing well and performing adequately at a different job in the same factory. His behavior at home was felt by his parents to be much improved. A blood specimen was negative for bromide. Six months later it was reported that the patient was continuing to do well but had had two episodes at home of irritability felt to be related to stress and his retardation. In retrospect, the patient suggested that he might have ingested an antacid containing bromide during his manic illness to relieve a recurrent upset stomach. He admitted to ingesting up to a whole bottle of the antacid daily.

DISCUSSION

Bromism is still a problem; it has been proposed that drugs containing bromide should be under prescription control or even banned from the market (5). The patient described here exhibited classical features of mania but did not show signs of bromide intoxication or delirium, although his serum bromide level was abnormally high. After his recovery the patient suggested a possible source of bromide. However, an alternative explanation is that because of his manic behavior, he medicated himself to reduce his various symptoms.

A review of case reports of patients with bromism revealed that some patients who were believed to be schizophrenic exhibited symptoms which were similar to those of mania. Such symptoms as violence, aggressiveness, agitation, delirium, talkativeness, and restlessness were used to describe patients with bromism (2). One patient who was "uninhibited and defective in concentration and memory" became "manic" after treatment to lower his bromide level and required ECT and chlorpromazine. Two recent reports (6, 7) emphasize the importance of properly diagnosing mania when one observes "schizophrenic" or other abnormal behavior. Perhaps patients previously reported as schizophrenic due to bromism were actually manic.

The importance of the case reported here is twofold. It is an important goal of clinical psychiatry to be aware of and diagnose any reversible causes of altered mental functioning. This case suggests that patients who exhibit manic behavior may benefit from an evaluation of bromide level. Establishing that bromism may be the cause of a manic episode would most likely remove the patient from the category of endogenous manic-depressive illness and the poor prognosis of a possibly recurrent disease. However, bromide ingestion may induce a manic episode in a predisposed individual, as the patient described may well be. Evaluation of additional manic patients is needed to clarify the possible relationship of mania and bromism.

Second, the implication that another monovalent ion is involved in mania may be important to research in manic-depressive illness. The possible roles of lithium have been the subject of much study (8). Recently rubidium has been suggested as important in depression (9). Hollister (10) reasoned that as lithium has the ability to replace sodium in mania, so might rubidium replace potassium in depression. Bromide has been recognized as being interchangeable with chloride (11). Although chloride is primarily an extracellular ion, its interchange with bromide may be important in mania, as the clinical data of this case suggest. Investigation of the role of the bromide ion in alterations of cellular electrolyte balance or neurotransmitter enzyme functions might be fruitful.

The clinical information from this case suggests that evaluation of manic patients should include a bromide level determination. Further research concerning the role of the bromide ion in cellular functions may result in better understanding of manic-depressive illness.

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Breaking Into the Prison: Conducting a Medical Research Project

BY JEFFREY S. RUBIN

Three major tasks face the researcher in a medical experiment employing prisoners as experimental subjects. Understanding the nature of the subject, crossing the territorial boundary, and obtaining informed consent are prerequisites for the successful implementation of research in the prison and rely heavily on the personal integrity of the investigator.

THE SUCCESSFUL implementation of a medical research project employing human subjects depends heavily on an ethically sound experimental design. When the researcher uses prisoner subjects, however, he is faced with three major tasks before his program can be implemented successfully. He must understand the nature of the inmate, cross the territorial boundary, and obtain informed consent. During a period when the issue of employing human subjects has come under wide scrutiny, it seems wise for researchers who are contemplating clinical research in prisons to consider the merit of *total planning* beyond experimental design.

NATURE OF THE INMATE SUBJECT

Bach-y-Rita (1) has pointed out that the prisoner as an experimental subject demands different considerations than an outpatient population due to the nature of his environment. To protect any subject from artifactual physiological and psychological effects, the researcher must first understand the subject and his needs. In the case of the prisoner, the task of learning what motivates him is difficult for a person who is not incarcerated. Banuazizi and Movahedi (2) have stated that it is difficult for nonprisoners to understand the nature of life in a prison environment.

In the maximum-security, indeterminate-sentence facility where I gained experience with inmate research subjects, the prison society is structurally similar to its counterpart outside the prison. Prisoners react to each other based on social contacts (3). There are dictums, codes, and hierarchies among the inmates to which each is expected to adhere. The inmate population is cohesive and sets itself in juxtaposition to the administrative staff and correctional officers.

Mr. Rubin is Research Administrator, Institute of Psychiatry and Human Behavior, University of Maryland School of Medicine, 645 West Redwood St., Baltimore, Md. 21201.

With this in mind, one cannot help but notice the paradoxical nature of the inmate. Here is an individual who cannot maintain himself in outside society yet functions quite efficiently in the prison environment. The researcher should not regard the prisoner subject as an antisocial individual. In the confined society of the prison the inmate is governed by similar drives and motives for his behavior as one is in free society.

While there is a structural similarity between the two environments, the difference is one of integration. By this, I am referring to the prisoner's demonstrated lack of integration into our main society due to any number of personality problems. In his confinement he is forced to create a microsociety with certain rules in order to survive. This microsociety is a closed one, and acceptance of outsiders is rare. The researcher, as an outsider to the microsociety within the prison, must first realize that it indeed exists and then cross the barrier in his search for subjects.

CROSSING THE TERRITORIAL BARRIER

At issue here is the mistrust that one group of people feels toward another group, whom they view as outsiders. As obvious and natural as this attitude may seem on the surface, a researcher can mistakenly assume that trust implicitly exists before the stage has been set. In his eagerness to obtain volunteers for his project, the researcher may inadvertently destroy the formation of a trusting relationship by an overzealously cooperative attitude.

I have found that many prisoners, before consenting to participate in a medical experiment, create their own type of experiment on the researcher to test his honesty and integrity, qualities that are valued highly in the microsociety. One such "test" I have frequently encountered is a mock intimidation by one or more inmates. The intimidation occasionally took the form of verbal attack against all prison research; in this case, the inmates were testing the investigator's personal convictions concerning his work. On other occasions the intimidation took the form of a verbal attack against the outside society; in this case, the inmates were testing the investigator's attitudes about criminals.

From this discussion one can see that these prisoners are clever individuals who realize that incensing a person through intimidation effectively precludes carefully planned approaches. I discovered that crossing the territorial boundary and gaining acceptance for my-

self and my research did not hinge on patronization or avoidance, but rather on stating personal feelings openly and honestly. The bridge is honest acceptance for who the prisoner is, unclouded by condemnation or condonation.

Once the researcher is integrated into the prison society, the last task he must face in implementing his project is obtaining informed consent.

INFORMED CONSENT

In recent years the issue of informed consent has come under public scrutiny, together with prison research in general. There has been much controversy over what constitutes informed consent as well as the degree to which a researcher can inform his subject before he invalidates his experiment.

Some clinical investigators (4) have articulated the dilemma facing researchers in trying to obtain informed consent by stating their agreement that in prison settings it may truly be impossible to obtain a totally free consent from a prisoner; however, is it not denying them their rights to refuse to allow them the opportunity to participate in research?

Arguments by both proponents and opponents of prison research tend to center on the issue of informed consent. I feel that a rigorous informed consent, mediated with a necessity to conduct a valid experiment, is an ethically sound research practice. The guidelines set forth by the Department of Health, Education, and Welfare (5, 6) are not meant to halt prison research but to ensure keen awareness of the need to protect all human subjects in medical research.

Although there are many types of experimental designs open to the clinical investigator, each involves different methodologies and necessitates different procedures. Generally, in the prison setting there is a common set of criteria that must be met in obtaining the informed consent of prisoners. In effect, the way in which the researcher meets the strict demands of informed consent mirrors how he truly views the subjects of his experiment.

The primary phase of obtaining informed consent is based on the investigator's careful and conscious desire to make his subject aware of the nature of the research and its potential hazards, both known and unknown. At the risk of perhaps frightening subjects from participating, the investigator owes the subject complete honesty regarding these issues. For a variety of reasons the prisoner may be more likely to volunteer out of covert pressure than his own willingness (1). If anything, a complete explanation of all the hazards may offset some of the pressure to participate, including the financial compensation, which is difficult for the inmate to resist.

Also imperative in obtaining consent is an enumeration of all the procedures to be performed, including a comprehensible explanation of medical procedures. I have found it a good idea to ask the inmate to restate what he understood was expected of him in order to ascertain whether he fully comprehended the given explanation.

In addition, it is advisable to inform the inmate both orally and in writing that subjects are in no way being intentionally coerced into participation—that it is strictly on a volunteer basis. The prisoner must be made to feel totally nonthreatened if he signs a consent form. Even if he is not led to believe that participation may influence his release from the prison, he may assume it. A good method for offsetting such unwarranted beliefs is stating and adhering to the principle of total confidentiality of all information gathered, including a prospective subject's refusal to participate.

I have found that a majority of prisoners believe that signing a consent form guarantees the prisoner's participation and releases the investigator from medical and legal responsibility for the care of subjects. I therefore feel it is wise to state in writing that in no way does an inmate's signature on the consent form indicate that a contract has been signed to guarantee participation or to release the investigator from any responsibility.

After the prisoner subject is made aware of this last point, the investigator can then explain the possible benefits of the research project to the prisoner, including any financial compensation. It is imperative that the researcher make a conscious effort not to make the benefits too attractive; otherwise, he may jeopardize the concept of "free" informed consent. Due to the nature of the prisoner subject, he may view as benefits of a particular research study those aspects which were not intended as a benefit, such as a chance to get "high," a break in routine, or advance knowledge of certain psychological tests.

Finally, in the consent interview with the prisoner and all through the program, the investigator should encourage questions. Frequently a prisoner will ask candid questions about experimental design that, if answered, may invalidate the results. When this happens it is best to answer the question as completely and clearly as possible and simply state that certain information cannot be disclosed to the subject until the experiment has been completed. An example of this would be a prisoner in a double-blind study asking whether he was receiving placebo or active medication.

CONCLUSIONS

A common thread emerges in accomplishing the tasks of understanding the prisoner, crossing the barriers between his society and the researcher's, and obtaining his informed consent. The personal honesty and integrity of the researcher are precursors to success. The prisoner is as much interested in the person or individuals who organize and administrate a medical research project as he is in the project itself.

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Workshop on Writing for Scientific Journals

The 1976 annual meeting of the American Psychiatric Association will feature the third annual workshop on writing for scientific journals, to be held from 9 a.m. to 5 p.m. on Sunday, May 9 (the day before the annual meeting begins). The workshop, cosponsored by APA and the American Medical Writers Association (AMWA), will offer practical information and instruction on four subjects: common writing faults, organizing psychiatric reports, getting your words on paper, and submitting a manuscript. Work assignments, which will be sent to registrants before the meeting, will form the basis for initial discussions during the workshop sessions. The course will begin with brief introductory lectures by the AMWA faculty, who include Charles G. Roland, M.D., chairman of the Department of Biomedical Communications, the Mayo Foundation, and Ms. Barbara G. Cox, medical writer, Instructional Development Section, the Mayo Foundation. A third member of the AMWA faculty will also participate in the meeting. Following the lectures there will be small-group discussions on the four basic subjects. The workshop will conclude with a panel discussion in which participants will have the opportunity to question the editors of several psychiatric journals.

Registration is limited to 40 participants. All applications for participation must be received by April 1; the registration fee is $65. For further information on registration write to Charles G. Roland, M.D., Department of Biomedical Communications, the Mayo Foundation, Rochester, Minn. 55901.

LETTERS TO THE EDITOR

Treatment of Accidental Naltrexone-Induced Withdrawal

SIR: EN-1639A (naltrexone) is a narcotic antagonist used for the treatment of heroin dependence. Naltrexone must be preceded by detoxification: patients should be opiate-free for at least a week before treatment is begun (1). Three cases of accidental ingestion of naltrexone by individuals dependent on opiates were reported by Tornabene (2). The treatment of the withdrawal syndrome consisted primarily of replacement of fluids lost by vomiting and diarrhea. One of the patients received 10 mg of morphine and low doses of methadone; these doses were ineffective.

We wish to report a similar case that was treated successfully with very large doses of hydromorphone (Dilaudid). The patient was a 28-year-old male ex-addict who had been maintained on 100 mg of methadone a day for 2 years. He had stopped using heroin but had started using alcohol and was admitted to an inpatient unit for alcohol detoxification. At 10:00 a.m. on the fourth day he was given by mistake 100 mg of naltrexone orally instead of his 100-mg dose of methadone.

At 10:10 a.m., the patient complained of chills and abdominal and muscular pain. He had generalized pilo-erection, extremely dilated pupils, and was also agitated. Subcutaneous administration of 4 mg of hydromorphone was without effect. At 10:17 a.m., he was given 4 mg of hydromorphone intravenously, after which he started vomiting and became extremely agitated. His chills and pains worsened and his pupils remained unchanged. At 10:22 a.m., he was given 10 mg of hydromorphone intravenously. His pupils showed very slight constriction, but he remained extremely agitated and ill. A further 10-mg intravenous dose of hydromorphone was given at 10:27 a.m.; his restlessness abated slightly, the vomiting stopped, and his pupils returned to normal size. Pilo-erection and chills persisted. At 11:10 a.m., the patient felt better but still complained of abdominal and muscular pain and was yawning. At 11:30 a.m., 20 mg of hydromorphone was administered intravenously. After this injection, the patient was still restless, yawning, and generally uncomfortable. Another 20 mg of hydromorphone was given, and at 11:45 a.m., the patient felt considerably better, but he was still somewhat restless and uncomfortable. He was given 10 mg of hydromorphone intravenously and 100 mg of methadone orally.

He felt well until 6:00 p.m., at which time he started complaining of chills. A total of 175 mg of methadone was given orally in divided doses over a period of 4 hours. At 11:00 p.m., he again complained of withdrawal symptoms and was given 8 mg of hydromorphone subcutaneously and 75 mg of methadone. He slept little that night, complaining of discomfort.

The next morning he felt weak and complained of headaches. He received a total of 200 mg of methadone in 3 doses on that day. On the third day, he was back on his regular dose of 100 mg of methadone without symptoms.

To summarize, an acute withdrawal syndrome was precipitated when naltrexone was given by mistake to a patient who was dependent on methadone. The treatment required a very large dose of hydromorphone—78 mg in the first 2 hours. Withdrawal signs and symptoms were used as guides for determining hydromorphone dosage.

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JAN VOLAVKA, M.D.
RICHARD B. RESNICK, M.D.
New York, N.Y.

Certification for Foreign Medical Graduates

SIR: As a member-in-training of the American Psychiatric Association, I would like to address APA members on the difficulty I am experiencing in becoming a full member of the association and seek their support for other individuals in situations like mine. I came to this country on an exchange visit. After I completed my residency in 1972, I suffered an intense conflict over the decision of staying here or going back to my country as a noncertified psychiatrist. After much turmoil and expert help, I went home in 1972. It was there that I realized that until I pass the American Board of Psychiatry and Neurology (ABPN) examination, I will merely be cheap labor both in the United States and my home country. I came back to the United States just to do that; however, the ABPN representative said that although my training is acceptable, I must have an unrestricted license in one of the states. I consider this an unnecessary requirement; I feel that successful completion of the ECFMG and licensing from my home country should suffice. Unless I complete this "initiation ceremony," I will remain a member-in-training in this country; I will be judged in need of further training in Canada and will be cheap psychiatric labor in poorer nations.

I would appreciate responses to this letter from my colleagues in the United States and from other foreign medical graduates (FMGs) in order to prepare a paper on this subject. (My definition of FMG is anyone who graduated from medical school outside the United States and who intends to stay in this country—this includes Canadians and U.S. citizens who have graduated from medical schools in other countries.) I would like to hear anything on the general topic of the situation of FMGs in American psychiatry from those

FMGs who plan to become a part of American psychiatry and American life. My goal could be better accomplished if I could compare this group with those FMGs who definitely plan to return to their home countries, but this variable is not predictable unless one has the resources to contact those who have actually followed through on this decision. I am trying to get such a comparison group from at least one country.

I will welcome responses at the address given below until July 1976.

AHMED K. NAFEES, M.P.H., M.D.
170 City Blvd., Suite 202
Orange, Calif. 92668

Parens Patriae and Psychiatry

SIR: It is remarkable to watch psychiatry attempting to plug the holes in the badly leaking vessel traditionally thought to contain the rationale for its *parens patriae* powers. The recent opinion "Is Dangerousness an Issue for Physicians in Emergency Commitment?" by Michael Alfred Peszke, M.D., and the comment by Alan A. Stone, M.D. (August 1975 issue), are examples of our apparent anxiety regarding our future as a profession without the power to force ourselves on unwilling patients.

Dr. Peszke correctly makes the point that when a psychiatrist commits a patient for "dangerousness," he is acting as an agent of the state. He then asks us to believe that if we force treatment on a patient using some other justification, we have somehow become the patient's agent. His reliance on our position as physicians to justify imposing intervention "only when there are sound and documented medical reasons" is particularly ironic because no physicians other than psychiatrists have asked for or been granted power to impose treatment.

Dr. Peszke would also have us equate lack of forced treatment with no treatment—we would be abandoning those seeking or accepting help if we no longer forced anything on those who did not seek or accept help. Dr. Stone does similar insult to the language by warning us that by relying on dangerousness as a commitment criterion, nondangerous individuals "will no longer have access to involuntary mental health care."

While properly decrying dangerousness as a valid reason for involuntary treatment, Dr. Peszke believes that "The physician can make a judgment that the severity of mental illness has impaired a patient's competence to make an informed decision as to the risks involved in not obtaining treatment." This, we are told, "returns the physician to his historical role as an agent for the patient's welfare rather than an agent of society." He gives no reason—and I see no reason—to believe that a return to an "in need of treatment" standard would do anything but make the situation worse.

Dr. Stone also suggested that we assess whether "a reasonable man in this situation [would] give up this much freedom for this much treatment." This standard is supposed to keep emergency commitment facilities from becoming jails and treatment from being preventive detention.

Could it be that more people will inevitably be harmed rather than helped by our forced intervention? A recent example is the young man who, upon losing his court battle to avoid conservatorial incarceration at the Napa State Hospital, leaped to his death from the Sacramento Medical Center. This should remind us of the quote from our Viet Nam experience: "We had to destroy the town in order to save it."

Might it not be wiser to recognize that not only dangerousness but all of the commitment criteria that we are using to justify and enforce preventive detention in facilities that are as much like jails as hospitals are lacking in scientific, clinical, and social merit? We can only become the true agents of our patients rather than of the state by renouncing all state power to force anything on anyone.

LEE COLEMAN, M.D.
Berkeley, Calif.

Dr. Peszke Replies

SIR: The fundamental issue here is whether our society will continue to accept as expedient and moral public policy the premise that severe forms of mental illness limit autonomy. To accept such a principle is certainly not necessarily to have clear-cut criteria for its implementation. I would in no way argue that the quality of care in many state institutions in this country is what a "modern" society has the right to expect.

I cannot agree with Dr. Coleman's statement that psychiatrists are the only physicians who have been granted or asked for the power to impose treatment on involuntary patients. Historically, this mandate has arisen from society and has been delegated to physicians. Only in the last 50 years has it become a psychiatric issue. In most countries, licensed physicians are mandated to commit mentally ill patients for emergency treatment and/or observation. Furthermore, most primary care physicians are continually confronted with patients who are unconscious or for other reasons unable to give informed consent. In many of these cases, the procedures that are performed are far more dramatic and heroic—and the results more untoward—than the average involuntary treatment in a mental institution.

Dr. Coleman poses the traditional question of whether more people will be harmed than helped by our forced interventions. Surely, the issue is what people and what kinds of forced intervention. We would not consider it a good practice to automatically do a tracheostomy on every patient who arrives in the emergency room following an accident, and yet such interventions are at times desirable and, in fact, lifesaving. There has to be some judgment about the harm to the patient that will result without treatment as opposed to the likelihood of benefit.

Dr. Coleman obviously misunderstood one of my arguments. I prefer the voluntary route for every patient, and I only wish that our institutions were able to provide quality care to everyone who wishes to be treated.

I do not relish the decision-making process when I am confronted with a patient who is psychotic, who refuses treatment, and whose welfare is jeopardized by his illness—particularly in cases where I know from experience that appropriate and available treatment would be helpful. I know that many psychiatrists share my reluctance in such difficult situations. I have on many occasions urged that the power to commit should be confined to specially licensed, forensically trained public health psychiatrists, working with appropriately trained attorneys. This system would provide emergency care while acting as a system of checks and balances in the clinical and administrative decision-making process.

The practice of involuntary treatment exists in most so-

cieties. It is a cross-cultural, historically legally sanctioned and philosophically acceptable approach to those people whom society considers to be at risk and to lack competence or autonomy. Society can change these ground rules. But unless we, as a profession, think that people who are seriously ill are only playing a role, then we owe it to society to show our concern, present the risks and options, and leave it to society to determine policy. It is important that we not allow society to make rules and regulations that are inimical to our professional concerns and incompatible with our expertise.

MICHAEL A. PESZKE, M.D.
Farmington, Conn.

Convulsions Following Disulfiram Treatment

SIR: We wish to report the occurrence of grand mal seizures in a patient who was taking disulfiram (Antabuse) and was abstinent. To our knowledge there has been only one previous report of such a case (1).

Our patient, a 50-year-old chronic alcoholic female, had been taking disulfiram for 6 months and had no history of neurologic seizure disorder. While she was taking 250 mg of disulfiram by mouth daily and was totally abstinent (as confirmed by her family and our observations in the hospital), she had five documented grand mal seizures. They occurred in the context of a clinical delirium that had apparently developed gradually over a 5- to 6-week period. The seizures cleared rapidly with discontinuation of the disulfiram and institution of phenobarbital and diphenylhydantoin therapy. No abnormalities were found on physical examination (except for hepatolienomegaly, thought to reflect her prior alcoholism), routine laboratory evaluations, neurologic examination, lumbar puncture, brain scan, or skull films. An initial EEG showed fast spikey background activity interpreted as being suggestive of a drug effect. A subsequent EEG, done 3 weeks after disulfiram had been discontinued, was normal and corresponded to gradual clearing of her delirium. The anticonvulsant medications were discontinued 3 weeks after admission and she has remained seizure-free, cognitively clear, and clinically well for the 6 months since her discharge.

When seizures occur in a patient taking disulfiram, they are thought to reflect an alcohol-disulfiram interaction. Since the variety of clinical situations that can lead to seizures in alcoholics is very broad, and appropriate and definitive treatment is largely dependent on an accurate diagnosis, the possibility of seizures as a direct side effect of disulfiram—even in the absence of a challenge by alcohol and especially if they occur in the context of a clinical delirium—must be considered in the differential diagnosis. Appropriate treatment is discontinuation of disulfiram and temporary institution of anticonvulsant therapy for the period of time during which the drug is being eliminated from the system.

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TREVOR R.P. PRICE, M.D.
PETER M. SILBERFARB, M.D.
Hanover, N.H.

Diabetes Insipidus and Obsessional Neurosis

SIR: The syndrome of "Acute Psychosis, Increased Water Ingestion, and Inappropriate Antidiuretic Hormone Secretion" reported by Murray A. Raskind, M.D., and associates (September 1975 issue) is of great interest. I have been following the association of psychiatric disturbance with diabetes insipidus since 1952, and in 1956 I described a syndrome of diabetes insipidus and obsessional neurosis, reporting nine cases (1). Three had been under my care (2), four had been described by other authors (3–6), and two were patients I interviewed in 1955.

The third case in my series was a woman who had been admitted to several hospitals where her obsessional features were never noticed. The patient admitted she had never told any physician about her obsessional compulsions and ruminations and that when asked specifically at one hospital, she had denied them altogether, although they had been plaguing her for years. In view of my experience that these obsessional features almost invariably go undiagnosed, I think it fair to presume that obsessional features were present in the cases presented by Dr. Raskind and associates, since they were so similar to two of my patients in many respects.

I suggest that obsessional neurosis and evidence of hypothalamic disturbance should be sought in all cases of diabetes insipidus, as I am confident this syndrome is more common than is supposed. Identification of further cases and adequate examination may contribute to our knowledge of the anatomy and physiology of appetite, thirst, and compulsion.

It may be that this syndrome could also give us a glimpse of the source or site of the generation of the "energy" underlying cathexis. Just as the syndrome of bitemporal hemianopia and diabetes insipidus suggested a single lesion in the region of the optic chiasma to Kruse in 1894 (7), which led to the investigation and further understanding of the neurohypophysis, it may be that antidiuretic hormone (ADH) or chemically similar compounds will be found to attentuate or control compulsive drives more specifically than the psychotropic drugs now available.

There is some doubt surrounding presently held concepts of the actions of ADH, since injection of aqueous vasopressin (Pitressin) reduces thirst dramatically in some patients before it could have had time to modify fluid and electrolyte balance by even 1%.

One of my patients lost her compulsions when her thirst was controlled by vasopressin. Since there is a subjective feeling of compulsion in both thirst and obsessional neurosis, there is clearly a wide area for speculation and investigation in the small area of the brain mentioned by Dr. Raskind and associates.

It was disappointing that six patients with severe obsessional neurosis without diabetes insipidus did not respond to the administration of small doses of vasopressin.

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RUSSELL BARTON, M.D.
Rochester, N.Y.

Lithium and Affective Disorders Associated with Organic Brain Impairment

SIR: The recent articles by Alan H. Rosenbaum, M.D., and Maurice J. Barry, Jr., M.D., regarding "Positive Therapeutic Response to Lithium in Hypomania Secondary to Organic Brain Syndrome" (October 1975) encouraged me to report the following case. I think this patient is unique in several ways and although lithium would generally be used with reluctance in patients with organicity, she seems to have responded to this treatment well.

The patient is a 61-year-old nun trained as a practical nurse. She was admitted in December 1973 for the fourth time to St. Louis State Hospital Complex, St. Louis, Mo., for continuing illness with main symptoms of confusion and depression.

Her psychiatric illness started in 1937, with a short episode of depression that did not require hospitalization. In 1939, the patient was hospitalized for depression with "somatic fixation, withdrawal, and inability to take care of her personal appearance." She had several short hospitalizations for essentially similar complaints in the next 2 years. In 1941, the possibility of multiple sclerosis was first considered. The patient had undergone repeated episodes of weakness of legs with incontinence and had subsequent complete disappearance of neurologic symptoms and signs.

The patient continued to suffer from frequent periods of depression until 1961. Electroconvulsive therapy (ECT) was used a number of times with apparent favorable results. In 1963 she was rehospitalized for extreme exhaustion, nystagmus, paraesthesia, and hyperreflexia on the left side with extensor plantar. Her medical management included intravenous histamine and steroids. However, the patient was noted to be quite depressed, and antidepressants were not effective. A course of ECT resulted in the development of talkative, argumentative, and belligerent behavior that was considered hypomanic and treated with chlorpromazine.

In 1964 the patient was readmitted for complaints of depression and anxiety. There was little improvement from treatment, and in 1967 long-term institutional care was finally considered. The patient was characterized as being "continuously psychotic, depressed, withdrawn, and careless of her personal hygiene and having a clouded sensorium." In 1968, she was first admitted to St. Louis State Hospital. On admission she was covered with feces from head to foot. She had a neurogenic bladder and a urinary tract infection. In 1969, she was noted to have mild dysarthria, scanning speech, positive Romberg's sign, unsteady gait, poor coordination with cogwheel rigidity, fine resting tremors, weakness of muscles, and diminished deep reflexes.

In 1970, the patient was reported to have become withdrawn, confused, and depressed. She also had frequent slips and falls and was generally clumsy. She had further admissions in 1971, 1972, and 1973 essentially for depressed affect, withdrawal, psychomotor retardation, urinary incontinence, inability to care for herself, refusal to eat, and crying spells. In her admissions to this hospital from 1968 until 1973, the patient was treated symptomatically with various phenothiazines and antidepressants. Her recurrent chest and urinary tract infections were treated with antibiotics. She was given small doses of anticholinergic drugs for her Parkinsonism.

The patient's complete blood count revealed mild anemia (12 g/100ml), sedimentation rate of 28 mm/hour, serum folic acid 2.4 g/ml (normal = 2.5 to 12). The patient's serum B_{12}, thyroid function test, urinalysis, and glucose tolerance test were within normal limits. Venereal disease and tuberculin tests were negative. X ray of chest, skull, and cervical spine were essentially within normal limits. Cerebrospinal fluid examination revealed mild elevation of proteins. EEG showed generalized dysfunction. EKG, brain scan, and right carotid angiogram were within normal limits. Psychological tests showed nonlateralizing organic brain impairment.

In view of the patient's history of repeated episodes of neurologic dysfunction with resolution over a period of time, she was considered to have multiple sclerosis. She seemed also to have recurrent affective illness, either associated with or uncovered by multiple sclerosis. As she had no cardiac, renal, or hepatic contraindication for lithium therapy, she was put on this treatment and has now been on it for some 20 months without complications and is doing quite well. It would appear that some patients with organic brain impairment and recurrent affective disorder might benefit from lithium.

DINESH B. MEHTA, M.D.
St. Louis, Mo.

Correction

There is an error in the article "A Hierarchy of Drug Use in Adolescence: Behavioral and Attitudinal Correlates of Substantial Drug Use," by Beatrix A. Hamburg, M.D., Helena C. Kraemer, Ph.D., and William Jahnke (November 1975 issue). In figure 1, page 1159, the authors inadvertently interchanged the labels of the curves representing hard liquor and tobacco usage.

BOOK REVIEWS

Why Survive? Being Old in America, *by Robert N. Butler, M.D. New York, N.Y., Harper & Row, 1975, 482 pp., $15.00.*

This is a sad and disturbing book, yet it is timely and of real importance. It is an excellent work that is difficult to review because one would like to quote extensively from it. Written by a man who obviously loves old people and who champions their cause, the book awakens deep emotional reactions; Dr. Butler demonstrates that in many instances old age in these United States is indeed a tragedy. He recognizes that some old folks are sick and that some are well, that some are rich and many more are poor, and he lifts a curtain so that we may see a side of life that is usually hidden from us or that we have not cared to see. He reports that at least 30% of the nation's more than 20 million elderly live in substandard housing and that over 10 million of them live on less than $75 a week—$10 a day.

Just as racism and sexism are abroad in the land, so too, says Dr. Butler, is another form of bigotry that he calls "ageism"—a widespread prejudice against the elderly in government, industry, housing, and, saddest of all for us, in medical practice, including psychiatry.

The myth of aging, the idea of inflexibility, unproductivity, and senility as inevitable accompaniments of age, is unfortunate; there are great differences to be found among the older age groups. Old women fare worse than old men, for instance. Many elderly people who suffer from reversible medical conditions are labeled senile and packed off to institutions. Many respond to the prejudice: some act senile, others try to conceal their age, and some attempt to appear young.

Every day more than 1,000 people pass over that artificial barrier which marks old age—65 years. This age group now comprises over 10% of the population of the United States; in 1903 this figure was 3%. Negative attitudes toward this age group range from pity to direct hostility, from infantilization to neglect or avoidance. Old age, the author says, has become an absurdity.

In the chapter titled "How To Grow Old and Poor in an Affluent Society," Dr. Butler notes that single women, widows, and members of minority groups fare worst of all of the elderly groups economically. The minority groups are in double jeopardy. We hear very little about these people because they do not court attention. They escape notice by staying in their homes or in their lonely rooms, locked in by timidity, illness, disability, lack of money, poor transportation, or fear of crime. In low-cost restaurants (if they can be found) they linger over a cup of coffee or a roll, often to the distress of the proprietor and the other customers. Their alternatives are hot dog stands or a hot plate in a furnished room.

Dr. Butler discusses the difficulties encountered in trying to collect pensions and the disheartening hunt for a place to live. He describes how old people wait endlessly for services essential for survival and the capacity to live independently. He observes that many of the elderly cannot afford proper medical care and that doctors and other health care personnel have not been trained to deal properly with them.

Direct as well as general prejudices against the elderly exist because they require more time from physicians and, often, are unlovely as individuals. Under difficult circumstances the elderly may at times become disoriented and demonstrate fluctuating awareness. This may be caused by congestive heart failure, head trauma, alcohol intake, dehydration, or cerebral vascular accidents. Given proper treatment, their chances of recovery are high. The danger, of course, is that they are often regarded as only senile or arteriosclerotic and sent to a nursing home. Once that magic word "senile" is uttered, the cause is regarded as lost.

Dr. Butler explores in depth the degradation that old patients experience in hospitals: they are not regarded as desirable patients. They get what he calls the "emergency room hustle" and "the transfer": "shut the door and wait." The final indignity is to isolate them when they are dying: one of the greatest fears of the elderly is that of dying alone.

Psychiatry does not do any better than general medicine in taking care of the elderly, although research reports indicate that older patients can benefit from mental health care. Dr. Butler points out that as many as 75% of patients over 65 in private hospitals can return to their own homes in two months. The difficulty, of course, is that most of the elderly cannot afford the level of care provided in private hospitals. Butler also speaks of the "Yavis syndrome," which is the therapist's interest in patients who are "young, attractive, verbal, intelligent, and successful." Therapists who do not work in public hospitals or clinics see patients from only a narrow band of the American population.

The book also deals with nursing homes, the victimization of the elderly, and the crimes of violence and frauds that are perpetrated against them. Politicians make promises to the elderly to pacify them, but in the end they neglect them. It all makes for a sorry story of man's inhumanity to man.

The final chapter, "Growing Old Absurd," ends with the following statement:

> After one has lived a life of meaning, death may lose much of its terror. For what we fear most is not really death but a meaningless and absurd life. I believe most human beings can accept the basic fairness of each generation's taking its turn on the face of the planet if they are not cheated out of the full measure of their own turn. The tragedy of old age in America is that we have made absurdity all but inevitable. We have cheated ourselves. But we still have the possibility of making life a work of art.

Thus the author examines the subject of aging in all of its complexities. He becomes personally involved with his subject, and his deep feelings are evident. The book is a labor of love. One leaves it depressed, but to understand the overall problems of the aged one of necessity has to read it.

F.J.B.

The Alternate Services: Their Role in Mental Health, *by Raymond M. Glasscote, M.A., James B. Raybin, M.D., Clifford B. Reifler, M.D., and Andrew W. Kane, Ph.D. Washington, D.C., Joint Information Service of the American Psychiatric Association and the National Association for Mental Health, 1975, 329 pp., $8.00.*

In a style that is now familiar, Glasscote and his collaborators report a study of a small but representative group of service delivery systems that typify a larger area of service systems in the mental health field. In this book 14 programs of recent vintage were studied through questionnaires and site visits. The findings have been summarized in separate chapters for each of the 14 programs. The service units range from hotlines and walk-in counseling centers to runaway houses, free clinics, and a residential center using the encounter techniques of therapeutic communities.

The authors' descriptions of these alternate services are well written, vivid, and as comprehensive as the various services would permit. In their words, "Alternate services are more or less targeted to teenagers and young adults whereas in traditional helping services, both medical and psychiatric, there has been in many places a distinct lack of services for this age group." They recognize that many of the alternative services had their origins in the drug crisis, epidemic, and panic of the past decade. Moreover, they note that these alternate services have increasingly shifted their focus from drug problems to the problems in living faced by juvenile offenders and by the depressed, lonely, or alienated members of society who are not drug users. The authors note, "It seems likely for the foreseeable future that our society will produce an ample supply of clients for the alternate services."

Perhaps one of the advantages of this book lies in the fact that it represents an interesting chronicle of the origins, struggles for survival, use of volunteers, and, in some cases, the demise of alternate services, which have been so enthusiastically developed throughout the United States in the past few years. It appears that the authors have been extremely fair in their approach; to the casual reader it might seem that they were lending their full support to these varied treatment programs. It seems possible that an uncritical lay reader might assume that alternate services show the way for the future.

For example, in the preface to this book Senator Birch E. Bayh, Jr., chairman of the Senate Subcommittee to Investigate Juvenile Delinquency, views this book as an inspiration, as an affirmation of the American tradition of volunteering to help one's fellows. Senator Bayh also states that the book documents "that as a nation we are able to respond quickly and appropriately to newly recognized and emerging needs." Senator Bayh was impressed that "the unit cost of services has been correspondingly low" in the alternate services at a time when "the cost of helping services seems to escalate at least as rapidly as the cost of everything else." He is encouraged to read that free clinics and telephone counseling services are still growing in number. He is "reassured to learn that originally highly specialized services have recognized the need to learn how to respond skillfully to the great variety of troubles and stresses that are part of growing up."

However, an important attitude of the authors toward the alternate services is best revealed in the following statements about various programs:

> As with other programs, their efficacy in bringing about lasting and desirable change in behavior is unknown. . . . Unfortunately, not one of the 14 programs could furnish data that would allow a combined compilation of the clientele. . . . We cannot even comment on the effectiveness or suitability of the response that these 14 alternative services made since none of them have follow-up data. . . .
>
> If the center has its distinctive qualities, it has one that it shares with everyone else. It has no good data concerning the effectiveness of the services it provides. . . .
>
> If outcome data for this interesting and rich program should ever become available they will result from efforts that are, at present, not even under consideration. . . .
>
> It would be astonishing if this program had any systematic information about the young people who leave it. And it does not. . . .
>
> The data available . . . are not exactly a researcher's paradise.

Inasmuch as more enthusiasm than research interest has surrounded the development of the alternate services, and inasmuch as efficacy data for these services are largely nonexistent at the present time, one might take the attitude that many of these services represent a special form of human research. It would be surprising to me if the guidelines for human research of the U.S. Department of Health, Education, and Welfare (HEW) were familiar, in any way, to most of the services described by Glasscote and his colleagues.

Because several of these services have received federal or state grants, one could ask what business the federal and state governments have in providing treatment funds for services that have no documented effectiveness. The answer to this question could probably be found in the therapeutic optimism of some funding agencies. Perhaps even more of the answer could be found in an amorphous HEW, in which agency heads responsible for dispersing treatment dollars may not be familiar with human research or their own agency's guidelines for the conduct of human research.

Some readers with a more traditional outlook might wonder what happened to demonstration research. It remains clear that a new and innovative service system that is unstudied contains the possibility of providing services that are not effective or are, in fact, destructive. This note of concern can be read between the lines in this book. Nevertheless, the genuine concern of volunteers for helping those who are in trouble represents a source of time, money, and manpower that is invaluable in the field of human services. The enthusiastic supporters of alternate services should recognize that a prolonged failure to study the effectiveness and suitability of these services may eventually be the most destructive course that can be followed in regard to the long-term survival of these services.

No reader of this book will miss the desperate financial plight and struggle for survival that has been common to the several services described. It seems clear that solid, respectable efficacy data could be most helpful in ensuring a less troubled existence for those selected services which should survive on the grounds of their excellence and high quality.

For those interested in a further pondering of the sociologic mysteries surrounding American youth over the past decade this book will provide fascinating background material. For those who wonder about the quality and acceptability of care in America's large medical institutions this book provides information for further speculation.

Finally, for those who subscribe to the concept of *primum non nocere*, this book will raise a lingering doubt that the alternate services have been overbalanced with therapeutic optimism and antiscience.

VERNON D. PATCH, M.D.
Boston, Mass.

Supportive Care: Theory and Technique, 2nd ed., *by Werner M. Mendel. Los Angeles, Calif., Mara Books, 1975, 237 pp., $12.00.*

Dr. Mendel provides us with a clear, vivid description of how he cares for chronic mentally ill patients, especially chronic schizophrenic patients. His book is concrete, practical, richly illustrated with clinical examples, free of jargon, and easy to follow. It will also be easy for experienced therapists to identify just where they do or do not agree with him and to trace the consequences of his basic philosophy of treatment throughout his technical advice.

"Supportive care" is defined as "that management which relieves suffering, prevents complications, and allows the patient to function at or near his capacity." It is sharply differentiated from "expressive" treatment, which is seen as contradictory and contraindicated for chronic patients: "Expressive techniques are contraindicated with the psychotic patient or for the patient classified as borderline psychotic." This sharp polarization of the two different treatments rather than a conceptualization of them as two different themes found in varying proportion in every psychotherapy may seem unwise to some readers. However, it is pedagogically effective, and it is associated with Dr. Mendel's view of chronic mental illness (essentially chronic schizophrenia) as a disability rather than a disease.

Dr. Mendel's treatment is a psychotherapy aimed at helping patients adjust and adapt to their disability; it holds no expectation of altering any basic underlying process. He is particularly effective in discussing therapeutic problems encountered in treating the chronically disabled, such as transference and countertransference issues, dependency and trust, the emerging "real" relationship with the therapist, change of therapists, and institutional transference. In keeping with his model, there is no discussion of therapeutic maneuvers designed to alter the primary deficits themselves.

Dr. Mendel's method is also based on his somewhat idiosyncratic view of schizophrenia:

> There are three primary defects which are the basis of the chronic schizophrenic illness and which are seen in such schizophrenic existences. These are (1) *the failure of historicity*, (2) *expensive and ineffective management of anxiety*, and (3) *disastrously painful and unsuccessful interpersonal transactions*. All other signs and symptoms of schizophrenia are secondary to these three defects.

By "historicity" (an unfortunate term and concept, in my opinion), he means the "lived history which the person brings to the task of conducting his life and which influences his perception of himself and others, his behavior, his emotional response, and his attitudes." This view of schizophrenia is not elaborated, tested against other views, or defended in the book, but it is always implicit in the technical recommendations. I suspect that it stems not so much from a theoretical refinement of prevailing views as from an extrapolation backward from the clinical maneuvers that the author has found useful. As such, like the book as a whole, it is heuristically valuable but scientifically unsatisfying. In a way, it is an antidote to the current popular discussion of expressive dynamic psychotherapies for schizophrenic and borderline patients, but a true synthesis of these two approaches is still wanting.

Although there is much with which to agree or disagree in this book, there is only one point on which I found the author's view unreasonably prejudiced—the role of the psychiatric hospital. He has apparently had some bad experiences with hospitals and seems to have generalized from them. In one passage he discusses the therapist's expectations as self-fulfilling prophecies; perhaps that complicates his use of hospitals. His views speak for themselves: in discussing the suicidal patient, "It is always best for the patient not to be hospitalized," and in discussing the patient's family, "Even a disorganized family, with proper management, is more therapeutic than a psychiatric hospital." The latter statement is sometimes true, but it hardly represents an ironclad principle.

The book has many lists and many rules. Not always theoretically satisfying, they more than make up for this by their clarity and pedagogic value. One suspects that we are being educated supportively. By the constant gentle push toward pragmatic action in the real world, we get a taste of the experience of a patient receiving supportive care. For those who profit from supportive education, or for those who are free to stand back from it, the approach is excellent. We learn that all psychotherapy can be resolved in 5 essential steps, that there are 16 indications for hospitalization, that there are 4 antitherapeutic factors always part of hospitalization, that appointments must never be canceled (to me a dangerous rule—it will inevitably get broken and, if treated as an absolute, the consequences will be magnified), and finally, that "it can be stated categorically that if a member of a hospital staff is struck by a patient he has erred in the performance of his therapeutic task." This last statement is certainly not true with patients who have brain syndromes.

The book includes a chapter on medication by Robert Allen, M.D., and one on ECT by Max Pepernik, M.D. The latter is usually optimistic about results, going far beyond the usually accepted view of the role of ECT in chronic schizophrenia. The bibliography, which is nicely annotated, essentially stops in 1968 (about the time of the first edition).

This is a valuable book for those who work with the chronically mentally ill. It will be most useful for training those who work within the author's frame of reference and for broadening the perspectives of experienced therapists with other points of view. It is not a general text and does not pretend to be one. The author's skill and experience as both therapist and teacher are clear, and his willingness to describe what he does in detail is most commendable.

ROBERT MICHELS, M.D.
New York, N.Y.

The Residential Psychiatric Treatment of Children, *edited by Philip Barker, M.B., D.P.M., D.C.H. New York, N.Y., Halsted Press (John Wiley & Sons), 1974, 346 pp., $22.50.*

This is a fine presentation of the current state of the art and practice of the field of residential psychiatric treatment of children in Great Britain. An extra dividend for the American reader is a review of the accomplishments and mean-

derings of the United States in this field. The British came later into this scene, after World War II; they could select and digest from our experience what seemed useful to them. Their judgment becomes an unusual opportunity to see ourselves as others see us.

Dr. Barker has put together a collection of clear and logical chapters written by himself and others concerned with the residential treatment of disturbed children. There is very little overlapping of subject matter. The writing styles are lucid and direct. There is little exploitation of innovation for innovation's sake or for grantsmanship's sake. The book could serve as a solid checklist for the fundamental ingredients of proper inpatient care and treatment of children.

The book shows sensitivity not only for the children themselves but for staff members as well. Careful attention is given to diagnosis and classification and also to the need for medical appraisal. Staff nurses seem to carry the main responsibilities with the children rather than the child-care workers relied on so much in the United States. Schooling is stressed as an integral part of the program. Treatment modalities lean toward behavioral modification, but psychoanalytic interpretations are employed judiciously and medications are used when really needed.

A significant difference between treatment programs in Britain and the United States is the relative smallness of the residential units in Britain. This seems to contribute appreciably to more individual attention without producing undue strains on the staff. Considerable emphasis is placed on careful, continual integration of the hospital services with all of the other family and community resources before, during, and after the hospital stay. The writers deplore the fact that there is only one academic unit of child psychiatry in Britain as well as the fact that the National Health Service accords rather scanty recognition to child psychiatry. We in the United States know these problems, too. The authors offer regrets for the absence from most treatment units of any staff for research and for the paucity of vigorously scientific studies of the actual treatment processes.

For perspective, Dr. Barker suggests the following:

> Long-term follow-up studies of child psychiatric patients should be treated with reserve. Inpatient care might be compared with one instrument in an orchestra, the music of which should be judged with and without that instrument. Consideration of inpatient treatment on its own is liable to lead to results as fallacious as judging the potential of a double-bass simply on what it can achieve played as a solo instrument.

ROBERT P. KEMBLE, M.D.
South Hadley, Mass.

The Psychoanalytic Forum, Vol. 5, *edited by John A. Lindon, M.D. New York, N.Y., International Universities Press, 1975, 430 pp., $15.00.*

The jacket of this volume carries the names of 68 "authors," which may be a record of some sort. In spite of this fact, the book does not come across in the usual desultory fashion of multiauthored books because the editor, John A. Lindon, has done his work well. In my opinion, he has successfully marshaled the contributions of these many and varied psychoanalysts into a readable and valid book.

Dr. Lindon has strictly limited the writing to nine clearly defined topics. Each topic is introduced by one well-known person, who presents his or her view of the particular issue. Then follow 6 to 10 shorter essays, all directly related to the primary topic, although often at variance with the point of view of the major presentation.

The quality of the comments varies, of course, but all address the central issue, a tribute to the editor as well as to each contributor. One feels when reading these papers that one is sharing an open meeting with people of strong but respectfully varying opinions. It is the sort of thing one wants from national meetings but, sadly, seldom gets. Each section gives the main author space for rebuttal, which makes for much more interesting and rounded reading. Because the topics are so varied, each of the nine will be identified in this review.

Harold F. Searles writes on "Violence in Schizophrenia." He stirs up the most controversial issue of the entire volume, namely, how much the personality of the therapist has to do with violence in the patient. Charles W. Wahl then presents some thoughtful material on "Psychoanalysis of the Rich, the Famous and the Influential," a subject about which all therapists must be concerned, to some degree at least. Again, the countertransference aspects come in for some heavy debate.

John C. Solomon addresses himself to "The Incredible Sixties: Psychodynamics of Youth," about which little is really known, even after one reads the seven responses. Stuart S. Asch presents some carefully thought out clinical and theoretical data on "Some Superego Considerations in Crime and Punishment." The respondents to his paper are well tuned to his tempo. Robert J. Stoller initiates some difficult polemics on the topic "Healthy Parental Influences on the Earliest Development of Masculinity in Baby Boys." Socarides, Mahler, De Saussure, Jossely, and Bowlby each has a great deal to say in response to this article.

Joseph M. Natterson writes on "Extra-Analytic Transference: A Two-Way Tide." This entire section is most delicately done by all of the writers. It will provoke thought in the careful reader. Frederick Kurth writes on "Projective Identification, Analyzability and Hate." The same range of theoretical comments, including the Kleinian perspective, can be found here as in other sections of the book. Melitta Sperling writes, in the way only she could have written, about "Somatic Symptomatology in Phobia." Her article is followed by interested praise and strong disclaimers by the discussants, all written before news of her death.

The last section of the book is the weakest: Hanna Segal writes on "Delusions and Artistic Creativity: Some Reflections on Reading William Golding's *The Spire*." This part of the book is interesting but of very little practical value to the average reader of this journal. Those who like this sort of thing have all they want in the analytic journals. It just did not seem to belong with the rest of this book. For the practitioner who must rely on reading to keep his perspectives clear, however, the bulk of this volume will prove valuable.

RAYMOND HEADLEE, M.D.
Milwaukee, Wis.

Principles of Family Psychiatry, *by John G. Howells, M.D., D.P.M. New York, N.Y., Brunner/Mazel, 1975, 446 pp., $17.50.*

Principles of Family Psychiatry is the latest in a scholarly series covering a wide variety of psychiatric subjects by Dr. Howells. In this volume he continues to display a depth of

knowledge and creativity that casts light not only on the field of family therapy but on psychiatry in general.

The scope of the book is quite broad—broader in fact than the title implies. What makes it possible to cover so much territory in one book is the excellent organization. One has the feeling that any information in the book could be relocated at a later date with very little effort.

As one would expect, the book contains an extensive analysis of the family—its functions, its dynamics, its psychology, and its psychopathology. Considerable space is devoted to this subject, with examinations of such areas as the role of individuals, communication among family members, and communication between the family and the rest of society. Dr. Howells presents a conceptual model of the family and then uses this model to help elucidate some ambiguous areas of psychiatric theory.

For example, family process is related to personality development and normal functioning as well as to somatic complaints, problems of the elderly, and many other areas of psychiatric disorder. In addition to examining the etiology of these disorders, Dr. Howells offers concrete suggestions for treatment, effective therapy goals, and organization of a family psychiatric service.

Probably the most innovative aspect of this volume is the section on the nosology of psychopathological conditions. Dr. Howells offers an extensive, well-grounded alternative to the traditional categories of diagnosis. It seems unlikely that this nosology will replace the terms currently in use, but it does provide the clinician with a valuable conceptual framework and concrete criteria for the evaluation of various "borderline" states. There is also an interesting section on schizophrenia in which this condition is subdivided in such a way as to make the diagnostic label more useful in the choice of treatment.

In summary, Dr. Howells offers an overview of family theory and therapy that should prove quite useful to those who wish to become more familiar with the area. He also offers a conceptual framework and a standardization of terms that should prove valuable to those of us already engaged in the practice of family psychiatry.

DAVID MENDELL, M.D.
Houston, Tex.

Training in Child Psychiatry in Canada, *edited by Dr. Paul Steinhauer. Toronto, Ont., Canada, Laidlaw Foundation, 1975, 114 pp., $3.00 (paper).*

To read this book is to regret that one was not part of its development. It records a two-and-a-half-day workshop at Val-David, Que., Canada, where 41 participants from all but one of the psychiatric training centers in Canada met in May 1974 to discuss four background papers that had been circulated in advance. Those position papers as well as the final presentation by a fifth contributor are reproduced in full in the second section of this book.

The first section is a masterful summary of the discussions; it is concise, terse, pithy, and yet reflective of the diversity of opinions expressed and the ardor of their protagonists. Dr. Steinhauer organized his overview of the workshop around nine key themes. He presents not only positions that all the participants could agree upon but also the dissenting views that many issues evoked. The result is a cornucopia of rewards for any reader. By no means confined to problems of training in Canada or to controversies within child psychiatry, the discussions range over the entire field of psychiatry and its place within medicine, within the broader fields of health and mental health, and within the society that supports it.

To imply that a review could give more than a suggestion of what this small volume contains would be an injustice to its editor and a disservice to its potential readers. The following are no more than isolated samplings of the kinds of questions the participants grappled with:

Recognizing that little has been achieved to eliminate mental health problems in the community, how can one argue for the relevance, efficiency, or even the validity of the specialty of psychiatry? What is the appropriate place for the psychodynamic/psychoanalytic model? Has the family model been oversold? Is the concept of effective consumer participation in planning and organizing services only a myth perpetuated by sociologists? How is training in child psychiatry best woven into the fabric of general psychiatric residency training? What is the ideal relationship between child psychiatry and pediatrics, between psychiatry and medicine, and between psychiatry and other mental health professions? What should residents learn, how should they learn it, and how should they organize and apply what they have learned?

The book is short, its content is full, and its style is easy. It is highly recommended.

ROBERT J. CAMPBELL, M.D.
Katonah, N.Y.

Practical Psychiatry for the Primary Physician, *by James R. Hodge, M.D. Chicago, Ill., Nelson-Hall, 1975, 345 pp., $15.00.*

Any psychiatrist who provides consultation to or teaches nonpsychiatric physicians soon accumulates a bookshelf of resources to which he can refer them. A practicing nonpsychiatric physician who wishes to read psychiatry is a rare treasure to be treated carefully, with respect and thoughtful guidance. To refer such a person to standard introductory psychiatry texts usually serves to drown his curiosity and enthusiasm in a flood of irrelevant detail.

The medical practitioner usually wants something that is clinically relevant and practical, uses a minimum of jargon, avoids unnecessary emphasis on psychopathology and theoretical dogma, and focuses primarily on doctor-patient relationships. By far the best book available for this purpose is Michael Balint's classic, *The Doctor, His Patient and the Illness* (1). The principal drawback to Balint's book is that one needs a fair amount of clinical sophistication and psychological mindedness to actually read it through and to be able to profit from the experience.

Dr. Hodge has provided a very useful single-author volume for the medical practitioner who is rediscovering psychiatry but is not yet ready to read Balint, or who needs an understanding of basic practical psychiatry, which Balint does not provide in quite the same way. In comparing this book with Balint's I do Hodge no disservice: Hodge acknowledges his debt to Balint throughout this book.

The strengths of Hodge's book lie in its practicality and commonsense approach. His presentation of the material is fair, balanced, and heavily laced with personal opinions and experiences, which are carefully labeled as such. In an engaging style, the book emanates practical clinical know-how. Although everyone will find things to disagree with here, I particularly liked the author's chapters titled "Principles of

Psychotherapy," "Strategies of Psychotherapy," and "Office Psychotherapy." Although the book is written for the nonpsychiatrist, I myself frankly found these chapters very instructive.

Features that may be less attractive to some readers stem from the author's tendency to skimp on detail and depth, presumably to enhance readability. Chapters particularly lacking in this regard are "Psychoactive Medications" and "Marital Conflict." Other chapters, although limited in scope and perhaps elementary in presentation, seem appropriate for the intended audience.

In summary, this is a good book for psychiatrists to skim if they wish to be familiar with potential resources for nonpsychiatrists. It is highly recommended for psychiatrists who want a good model of format and style for instructing nonpsychiatric physicians. It should also be popular with any physician who enjoys reading how an obviously skilled clinician deals with everyday problems of medical practice.

REFERENCE

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MARTIN R. LIPP, M.D.
San Francisco, Calif.

Down's Syndrome (Mongolism): Research, Prevention and Management, *edited by Richard Koch, M.D., and Felix F. de la Cruz, M.D. New York, N.Y., Brunner/Mazel, 1975, 235 pp., $10.00.*

Books that result from conferences are frequently eclectic in their treatment of the alleged common theme; this book is no exception. Nevertheless, it should not be dismissed lightly by the practicing physician, the medical educator, or anyone else who is interested in the mental health of families who have to cope with the presence of a chronic congenital handicap in one of their members. Its eclectic aspects stem directly from its subject matter, which is many faceted.

The conference from which these papers proceed was organized by two pediatricians and the father of a youngster with Down's syndrome. It was sponsored by the National Association for Retarded Citizens, organizations of parents of children with Down's syndrome, and the Mental Retardation Branch of the National Institute of Child Health and Human Development. The papers were prepared for the purpose of answering many of the questions that intelligent and concerned parents ask these days about Down's syndrome and about the prospects of children born with this condition.

Three of the papers deal with aspects of biochemical balance. Three deal with topics relevant primarily to the adult with Down's syndrome—employment, community mobility, and sexuality. Four papers deal with early intervention strategies based on psychoeducational techniques, two deal with longitudinal studies indicating the norms of development for this group of children as they grow up, and one deals very candidly with "Present Approaches to Therapy in Down's Syndrome." As a documentation of the state of the art in 1973, this book has both current and historical significance for physicians and child development specialists as well as parents.

Twenty-five years ago it was the usual practice for physicians to recommend early institutionalization of the mongoloid baby. Indeed, it was not uncommon for the child's father to be advised to have the infant placed directly from the newborn nursery so as not to let his wife become attached to "it." Until about 10 years ago, most data reported about the potentials for development of infants with Down's syndrome were based on observations made on children in institutions. We now know that the very process of caring for the children in this way considerably reduced their effective functioning. One of the contributions of this volume is to provide documentation based on creditable research rather than on isolated instances showing the changed expectations for developmental and social quotients obtained at various ages by children and adolescents with Down's syndrome raised at home. It makes clear that the physician jeopardizes his own credibility if he glibly tells parents that their infant will never walk or talk, as has so often happened in the past.

Parents have always been and continue to be intensely interested in the etiology of their children's disabilities. This parental interest is reflected in several of the papers included in this book. These papers also contain an implicit lesson for parents in the methods and conduct of research. Some of the very parents represented in this book lobbied the Senate Subcommittee on Appropriations for the U.S. Department of Health, Education, and Welfare in 1969. They wanted to be sure that Mary Bazelon Coleman would be funded for her studies on the role of serotonin in Down's syndrome. At the conference in 1973 they heard her give a straightforward and detailed account of the results of treatment with 5-hydroxytryptophan; the results were largely negative.

Nevertheless, hidden in Dr. Coleman's paper is an important observation:

> One of the major conclusions of our study relates to the depression and despair that pediatricians induce in the parents of Down's Syndrome patients by informing them at birth of the coming disability of their child. . . . The whole area of informing and supporting parents regarding the diagnosis and prognosis of a Down's Syndrome child during the early months of life is identified by this study as an area which needs research. (p. 114)

Psychiatry has little to contribute to the prevention of Down's syndrome, and it falls relatively infrequently to the psychiatrist to communicate this diagnosis. However, this process is fraught with risk for parents directly and for their infants indirectly. Not a single psychiatrist contributed to the body of this book; paradoxically, this constitutes the prime reason why psychiatrists, especially those in teaching positions, should both own and loan it.

ELIZABETH M. BOGGS, PH. D.
Hampton, N.J.

What is Psychotherapy? Parts I and II. Proceedings of the 9th International Congress of Psychotherapy, *edited by T.-E. Mogstad and F. Magnussen. New York, N.Y., S. Karger, 1975, 617 pp., $67.50.*

This collection of papers from the 9th International Congress on Psychotherapy in Oslo, Norway, is both extensive and expensive, the one attribute presumably justifying the other. The more than 100 very brief articles by as many different authors do not explore their topics in any great depth. The theme of nearly all of the papers seems to be that Freud is not enough.

The articles begin by labeling psychoanalytic therapy

"pseudoscience," note the "bankruptcy in the individual dynamic model," and conclude that psychotherapy today has refused to be limited to conventional interviews but has spilled over into activities beyond the individual patient. The participants of the conference seem motivated to elaborate beyond the plain and simple Freudian meaning of psychotherapy, with the evident goals of reaching a consensus, attaining greater efficiency, and providing greater adaptability to various social situations.

A few writers plead for training in self-restraint and for gaining new insights without losing sight of the values and knowledge provided by the pioneers of analysis. Chessick seems to make the most notable effort to integrate traditional psychotherapy and the newer therapies.

The expense of the book may be justified by the fact that it demonstrates the amazing range of variations in psychotherapy that exist worldwide. Two opening lectures state that psychotherapy molds the individual into his society—into a system of secular ethics, which, according to Szasz, is coercive, or into a way of realizing the image of man in actual socialistic life circumstances, which, according to a Marxist, makes life useful. The following contributions cover such remotely related issues as family therapy, systems theory, behavior therapy, philosophy, treatment of children, and social psychiatry.

Most of the well-known contemporary authorities (Frank, Strupp, Sifneos, etc.) are included in this volume; most of them restate what they have said before. It is interesting that the most quoted authors are (in order of frequency of quotations) Freud, Malan, Frank, Wolpe, Binswanger, and Wolberg.

The greatest reader interest will be attracted to the articles on autogenic training, Zen, and the roles of the shaman and the folkhealer in Africa and India.

The greatest weakness in the field of psychotherapy today appears to be the scarcity of articles on research in psychotherapy. Except for the work of a group in Boston, the research articles presented at the Oslo meeting appear to be limited to narrative reports of individual clinical experiences. In spite of their great fascination in revealing unique situations, these solitary studies simply show that psychotherapy today has yet to mount a program of decisive research to establish its real worth.

C. E. SCHORER, M.D.
Detroit, Mich.

Depression: Concepts, Controversies, and Some New Facts, *by Eugene E. Levitt and Bernard Lubin. New York, N.Y., Springer Publishing Co., 1975, 163 pp., $8.50.*

The authors of this book would have done well to select a more appropriate title than simply *Depression*. This presentation is not really a book but a monograph; it deals with the results and interpretations of a special type of study. Using a questionnaire method of investigation, the authors sampled a population of 3,011 individuals throughout the United States to determine whether significant correlates to depression could be identified.

Their findings can be briefly summarized. Depression seemed to be more prevalent among older people and more evident in women than men. The authors also found a negative correlation between depression and education level and a higher evidence of reported depression among people of lower or moderate economic status who had recently suffered economic reverses. Black women in particular showed a high degree of depression. It was equally distributed among married and single people. Contrary to recent reports, people in high-prestige occupations, such as medicine, showed less depression than those in low-prestige occupations.

The difficulties inherent in this type of investigation are well known to anyone who has attempted epidemiological research. To begin with, in spite of the authors' lengthy review of the relevant literature and their discussion of methodology, the validity of their findings can be questioned on several levels. For instance, the levels of significance are frequently weak: the male-female correlation achieved only a 1% level, and the income correlations achieved only a 0.1% level.

Second, it is always difficult to know what is actually being detected in a study of this type. The introductory discussion of the nature of depression—what it may be as a mood, as a reaction, as a clinical condition—is insufficient and simultaneously too extensive to set the stage for the report that follows. This problem stems from the fact that rather than reconceptualizing the problem of depression in the light of "some new facts," as they set out to do, the authors have produced, essentially, an extended journal-type article with findings worth publishing and clues to further research but without the depth they originally promised.

It would have been much more satisfying if they had let the reader know early on what he was going to encounter, rather than preparing him for something that never happened. "Mountains will be in labor," said Horace, "and an absurd mouse will be born."

There is an important place in the psychiatric literature for the monograph format. Reorganized, the material presented in this book would have been better published as such rather than in its present form.

FREDERIC F. FLACH, M.D.
New York, N.Y.

The Diagnosis and Treatment of Alcoholism, *by Gary G. Forrest, Ed.D. Springfield, Ill., Charles C Thomas, 1975, 251 pp., no price listed.*

The author, who supervises a military alcoholism rehabilitation program, addresses himself to the broad field of clinical workers in alcoholism treatment programs. He provides background information as well as "how I do it" chapters that should be helpful, especially to alcoholism counselors with limited professional background and professionals coming into the field with limited experience with alcoholic patients. Experienced professionals will find the book helpful, especially if it supports their particular viewpoints, and quite useful in staff training.

The first section of the book presents background information about the definition of alcoholism, the stages of alcoholism, and personality characteristics of the alcoholic patient. One might criticize Forrest's definition of "alcoholism" as distinct from "problem drinking" as being a bit too restrictive and placing too much emphasis on physical complications rather than evidence of true addiction. His use of the term "chronic alcoholism" for the most advanced stage is a redundancy because alcoholism is chronic by definition. In discussing the meaning of the drinking experience, emphasis is given to relief of pain; the pleasure provided by alcohol is not given sufficient importance. However, Forrest says

the following, with which I most heartily agree: "It is my impression that the addiction per se is the focal point to be dealt with therapeutically" (p. 60).

The second section of the book, Strategies of Treatment, discusses individual and group psychotherapy, Alcoholics Anonymous, residential treatment, and behavior therapy. The chapters on psychotherapy are the best in the book; these will be quite helpful to new counselors because they offer optimism and encourage an open, involved approach. Forrest emphasizes warmth, empathy, and genuineness; he is a directive Rogerian. His description of his results might sound like "transference cures" to many, but he does not address countertransference issues. Nevertheless, the techniques he suggests are sound, although he may be a bit overenthusiastic when he claims 85% effectiveness (p. 73) without substantiating data.

Two interesting practical suggestions can be found in the chapters on psychotherapy: no more than 40% of a treatment staff for alcoholic patients should be made up of "recovered" alcoholics, to avoid one-sidedness; in group treatment, there should be some group members with at least six months' sobriety to serve as role models.

Other discussions in this section give superficial coverage to such topics as detoxification, genetics, hospital programs, and behavior therapy.

The third section of the book is a mixed bag of brief theoretical essays and small pilot research studies. Forrest discusses the need for self-disclosure by the alcoholic for recovery, drinking periods as symbolic death and recovery as rebirth, and the "hide-and-seek" game of the hidden alcoholic in the military. He does better when he gets back to treatment, especially in the chapter titled "Resolution of the Alcoholic Power Fantasy." The research studies on treatment effectiveness are of the pilot variety—e.g., with small samples and self-selected "controls." These studies suggest the capability of the author and his staff to do more definitive research on their clinical work.

The book contains certain errors, e.g., the following: "Significant numbers of alcoholics die annually as a result of the DT's, in spite of medical intervention" (p. 21); diazepam and chlordiazepoxide do not reduce anxiety but alcohol does (p. 42); high blood pressure and diabetes are "clearly a part of the overall clinical picture" in late-stage alcoholism (p. 58); and "Intravenous and oral fluid ingestion [should be used] to counteract dehydration" (p. 125).

With prompt medical attention, the mortality rate from delirium tremens is very low, and a dropping alcohol blood level leads to overhydration. However, taken as a whole, with its good and bad points, this book is a useful addition to a staff training library.

ROBERT A. MOORE, M.D.
San Diego, Calif.

Growth and Change of Schizophrenic Children: A Longitudinal Study, *by William Goldfarb, M.D., Ph.D. Washington, D.C., V.H. Winston & Sons (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1975, 271 pp., $10.95.*

In their studies of schizophrenic children and their families during the past two decades, William Goldfarb and his collaborators at the Ittleson Center have shown how clinical research and humane care can enrich each other (1, 2). This book is a report on changes in adaptive functions of 40 schizophrenic children who received residential treatment when they were between the ages of 7 and 10 years. The children were tested and rated on a variety of measures at several points during the course of treatment. The results were assessed for trends over time and comparison with normal children. The 40 measures included IQ tests, neurological assessments, academic achievement scales, perceptual tasks, and a variety of rating scales.

On many dimensions of conceptual behavior, communication, motor coordination, and perceptual skill, the schizophrenic children showed definite improvement in response to intensive educational, social, and therapeutic influences. However, most of the children did not reach normal levels, and there were great individual differences. On several dimensions the children's initially marked abnormalities persisted: muscle tone (hypertonia and hypotonia), activity (hyperactivity and hypoactivity), and receptor function (underreaction or overreaction to delayed auditory feedback and sudden noise). The children's deviant receptor responses were consistent with the idea that their difficulties in self-regulation may be related to disturbances in meaningfully organizing perceptual information and deploying attention. Goldfarb suggests, however, that deficits even in these areas may be remedied with newer educational techniques.

Anyone who has conducted longitudinal clinical research will appreciate the difficulties that must have been encountered in conducting this study: changes in therapeutic and educational ideas and methods, staff turnover, and, above all, the need to treat each child as an individual. These factors must be kept in mind in evaluating the results. One would like to cite this study as evidence of the value of intensive residential treatment, which it supports. However, the study is limited because we do not know about the universe of disturbed children from whom these fortunate 40 were drawn, and there is no adequate control group of schizophrenic children who were treated in other ways (e.g., day schools with active parent involvement).

In his previous studies as well as this one, Goldfarb has clarified some dimensions of the heterogeneity of the population of children who are diagnosed as schizophrenic. They come from different kinds of families, differ in neurological integrity, and present with a broad range of intellectual, social, and emotional disabilities. How they differ, as demonstrated in this book, may be more apparent than how they are the same. The phenotypic variance probably reflects important genotypic distinctions.

Until the fundamental lines are drawn between subgroups of the childhood schizophrenias or along the schizophrenic or atypical spectrum, all research on severe psychiatric disturbances of childhood will be hampered. One contribution to this effort is Goldfarb's attempts at distinguishing subgroups clinically. Advances in biological research may be dependent on this clinical investigation and, in turn, may provide new criteria for subgrouping.

For most readers, the main attraction of this book will be its summary of Goldfarb's balanced viewpoint on ego disturbances in childhood schizophrenia. The book has two relatively minor limitations. First, the title may imply that it is a long-term longitudinal or outcome study of childhood schizophrenia rather than a study of the impact of residential treatment during three years. Second, the 130 pages of tables will be of little interest to any but the most specialized researcher. Perhaps they could have been made available in another way to those interested. A more substantial concern has to do with the analysis of the data. With so many measures, many of which tended to cluster together, I wonder why

more sophisticated, multivariate analysis was not attempted to explicate underlying dimensions and, perhaps, to assist in defining subgroups.

REFERENCES

1. Goldfarb W: Childhood Schizophrenia. Cambridge, Mass, Harvard University Press, 1961
2. Goldfarb W, Mintz I, Stroock KW: A Time To Heal. New York, International Universities Press, 1969

DONALD J. COHEN, M.D.
New Haven, Conn.

Mental Health: The Public Health Challenge, *edited by E. James Lieberman, M.D. Washington, D.C., American Public Health Association, 1975, 287 pp., no price listed.*

The general purpose of this book appears to be to stimulate greater interest in mental health services among public health administrators. It probably will succeed in this endeavor because it presents the mental health dimensions of subject areas that are familiar to public health personnel—e.g., prevention, training, services, policy making, and social issues. The last chapter, on information sources, is comprehensive and of value to everyone interested in the field of mental health.

By using a definition of prevention that includes primary (the removal of causes), secondary (early treatment), and tertiary (rehabilitation), the book makes all mental health services legitimate for public health personnel. Some of the articles give the impression that the field of mental health has the responsibility and the capability to solve social and environmental problems that have theoretical, direct, or indirect impact on the well-being of individuals or communities. These include, but are not limited to, the following: poverty, discrimination, unemployment, crime, delinquency, education, and divorce. This may attract and challenge some public health personnel, but it might create a serious reluctance for others.

The field of public health has been concerned traditionally with primary prevention. Lemkau, with his usual candor, states that "the pathological mechanisms for the most important 'functional' mental illnesses, affective disorder, schizophrenia, neuroses, and personality disorders remain conjectural or unknown." He also states that "some causes of mental disorders are known, but the causes of most are unknown, or at least unproven." He then lists as known causes poison, infection, genetics, nutrition, physical trauma, and general systemic disease. Another author in the volume, however, is undaunted by such knowledge, writing of his interest in improving the quality of life and well-being of patients, "not merely averting pathology." This author states that his approach to primary prevention is based on a "socio-psycho-cultural-educational rather than a medical model."

It is well to remember, while reading the expositions of the various authors, that the admonition at the beginning of the book says that the ideas and opinions presented by the authors are their own and do not necessarily represent the view or policies of the American Public Health Association. That is what makes the various articles interesting.

JAMES V. LOWRY, M.D.
San Diego, Calif.

Studies of Schizophrenia. British Journal of Psychiatry Special Publication 10, *edited by M.H. Lader. Ashford, Kent, England, Headley Brothers, 1975, 160 pp., $9.00 (paper).*

This publication makes available in convenient form the papers read at the World Psychiatric Association symposium Current Concepts of Schizophrenia, which was held in London in November 1972. The authors represent 14 countries of Europe and 3 of the western hemisphere; the reader in the United States thus obtains access to a great deal of unfamiliar work. As Lader points out in his foreword, there are great differences in the concepts used in different countries.

Opening remarks by Dr. Howard Rome and Sir Martin Roth outline recent developments in research on schizophrenia. There follow four chapters on diagnostic concepts, five on genetic and nature-nurture aspects, five on schizophrenia and the community, four on psychotherapy, and nine of other aspects of treatment. All are brief and concise presentations of useful material.

Particularly worthy of notice is the chapter by Tienari, of Oulu, Finland, who summarizes results of concordance rates of monozygotic twin studies reported between 1928 and 1970 and presents up-to-date results of his study of all pairs of male twins born in Finland between 1920 and 1929. His 1963 report found no concordance in 16 pairs of monozygotic twins with 1 schizophrenic member. A total of 1,432 twin pairs were reviewed again in 1969 and 1970: preliminary results now support a concordance rate of 15% in monozygotic pairs and 7.5% in dizygotic pairs for schizophrenia or paranoid psychosis.

Another chapter of special interest is that by Kolyaskina and Vartanian of Moscow, who report findings that suggest that patients with continuous (nonrecurrent) schizophrenia and their relatives show brain antibodies in their systems.

The topic of schizophrenia and the community is covered in articles from England, the United States, Canada, Hungary, and Rumania; it is evident that similar problems in attempts to return schizophrenia patients to the community arise in different countries.

This is an admirable compendium of papers that shows the diversity of approaches to schizophrenia throughout the world. Although uneven in scientific caliber, it fulfills its stated goal of presenting the views of leading researchers in the field in a relatively concise and readable form. It can be recommended to residents in psychiatry as a source of information on schizophrenia that is not found in standard textbooks or American journals.

A. ARTHUR SUGERMAN, M.D.
Belle Mead, N.J.

This Month's Reviewers

VERNON D. PATCH, M.D., is Associate Professor of Psychiatry, Harvard Medical School, Boston, Mass.

ROBERT MICHELS, M.D., is Barklie McKee Henry Professor of Psychiatry and Chairman of the Department of Psychiatry, Cornell University Medical College, and Psychiatrist-in-Chief, New York Hospital, New York, N.Y.

ROBERT P. KEMBLE, M.D., is Chief of the Mental Hygiene Clinic, Veterans Administration Hospital, Northampton, Mass.

RAYMOND HEADLEE, M.D., is Clinical Professor of Psychiatry, Medical College of Wisconsin, and Professor of Psychology, Marquette University, Milwaukee, Wis.

DAVID MENDELL, M.D., is Clinical Professor of Psychiatry, University of Texas at Houston.

ROBERT J. CAMPBELL, M.D., is Medical Director, Four Winds Hospital, Katonah, N.Y.

MARTIN R. LIPP, M.D., is Assistant Clinical Professor of Psychiatry and of Ambulatory and Community Medicine, University of California, San Francisco, School of Medicine, San Francisco, Calif.

ELIZABETH M. BOGGS, Ph.D., lives in Hampton, N.J.

C. E. SCHORER, M.D., is Assistant Director in Charge of Education, Lafayette Clinic, Detroit, Mich.

FREDERIC F. FLACH, M.D., is Associate Clinical Professor of Psychiatry, Cornell University Medical College, and Attending Psychiatrist, Payne Whitney Clinic, New York Hospital, New York, N.Y.

ROBERT A. MOORE, M.D., is Medical Director, Mesa Vista Hospital, San Diego, Calif.

DONALD J. COHEN, M.D., is Associate Professor of Pediatrics, Psychiatry, and Psychology, Yale University School of Medicine, New Haven, Conn.

JAMES V. LOWRY, M.D. is a Mental Health Services Consultant in San Diego, Calif.

A. ARTHUR SUGERMAN, M.D., is Medical Director and Director of Research, Carrier Clinic, Belle Mead, N.J.

Books Received

This listing acknowledges the receipt of recent books. Books of special interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Depression and Human Existence, *edited by E. James Anthony, M.D., and Therese Benedek, M.D. Boston, Mass., Little, Brown and Co., 1975, 546 pp., no price listed.*

The Foundations of Psychiatry. American Handbook of Psychiatry, 2nd ed., Vol. I, *edited by Silvano Arieti; Silvano Arieti, editor-in-chief. New York, N.Y., Basic Books, 1974, 1,215 pp., $29.50.*

Child and Adolescent Psychiatry, Sociocultural and Community Psychiatry. American Handbook of Psychiatry, 2nd ed., Vol. II, *edited by Gerald Caplan; Silvano Arieti, editor-in-chief. New York, N.Y., Basic Books, 1974, 836 pp., $24.50.*

Adult Clinical Psychiatry. American Handbook of Psychiatry, 2nd ed., Vol. III, *edited by Silvano Arieti and Eugene B. Brody; Silvano Arieti, editor-in-chief. New York, N.Y., Basic Books, 1974, 860 pp., $24.50.*

Organic Disorders and Psychosomatic Medicine. American Handbook of Psychiatry, 2nd ed., Vol. IV, *edited by Morton F. Reiser; Silvano Arieti, editor-in-chief. New York, N.Y., Basic Books, 1975, 922 pp., $32.50.*

Treatment. American Handbook of Psychiatry, 2nd ed., Vol. V, *edited by Daniel X. Freedman and Jarl E. Dyrud; Silvano Arieti, editor-in-chief. New York, N.Y., Basic Books, 1975, 954 pp., $32.50.*

New Psychiatric Frontiers. American Handbook of Psychiatry, 2nd ed., Vol. VI, *edited by David A. Hamburg and H. Keith H. Brodie; Silvano Arieti, editor-in-chief. New York, N.Y., Basic Books, 1975, 1,022 pp., $32.50.*

Your Child's Self-Esteem: The Key to Life, *by Dorothy Corkille Briggs. Garden City, N.Y., Dolphin Books (Doubleday & Co.), 1975, 334 pp., $2.95 (paper).*

The Biofeedback Syllabus: A Handbook for the Psychophysiologic Study of Biofeedback, *edited by Barbara E. Brown, Ph.D. Springfield, Ill., Charles C Thomas, 1975, 495 pp., $26.50.*

The Patient and the Therapist: New Light on the Psychotherapist, *by Arthur Burton. Sacramento, Calif., Hamilton Psyche Press, 1975, 168 pp., $8.95.*

Mental Disorder in Earlier Britain: Exploratory Studies, *by Basil Clarke. Cardiff, Wales, University of Wales Press, 1975, 327 pp., £10.00.*

Nebraska Symposium on Motivation 1974, *edited by James K. Cole and Theo B. Sonderegger. Lincoln, Neb., University of Nebraska Press, 1975, 292 pp., $6.95 (paper).*

Psychiatry and Mysticism, *edited by Stanley R. Dean, M.D. Chicago, Ill., Nelson-Hall, 1975, 398 pp., $15.00.*

Language and Interpretation in Psychoanalysis, *by Marshall Edelson, M.D., Ph.D. New Haven, Conn., Yale University Press. 1975, 229 pp., $12.50.*

Preparation for Breast Feeding, *by Donna and Rodger Ewy. Garden City, N.Y., Dolphin Books (Doubleday & Co.), 1975, 118 pp., $2.95 (paper).*

Don't Say Yes When You Want To Say No, *by Herbert Fensterheim, Ph.D., and Jean Baer. New York, N.Y., Dell Books, 1975, 298 pp., $1.95 (paper).*

Annual Review of Behavior Therapy Theory and Practice 1975, *edited by Cyril M. Franks, Ph.D., and G. Terence Wilson, Ph.D. New York, N.Y., Brunner/Mazel, 1975, 827 pp., $25.00.*

Biology of the Major Psychoses: A Comparative Analysis. Association for Research in Nervous and Mental Disease Research Publication, Vol. 54, *edited by Daniel X. Freedman, M.D. New York, N.Y., Raven Press, 1975, 358 pp., $22.00.*

The Cycles of Sex, *by Warren J. Gadpaille, M.D.; edited by Lucy Freeman. New York, N.Y., Charles Scribner's Sons, 1975, 472 pp., $17.50; $6.95 (paper).*

Psychopathology: A Cognitive View, *by Zvi Giora, Ph.D. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1975, 175 pp., $12.95.*

The Movies on Your Mind, *by Harvey R. Greenberg, M.D. New York, N.Y., Saturday Review Press/E.P. Dutton & Co., 1975, 264 pp., $4.95 (paper).*

Daughters and Mothers: Mothers and Daughters, *by Signe Hammer. New York, N.Y., Quadrangle/New York Times Book Co., 1975, 175 pp., $7.95.*

Drug Interactions: Clinical Significance of Drug-Drug Interactions and Drug Effects on Clinical Laboratory Results, 3rd ed., *by Philip D. Hansten, Pharm.D. Philadelphia, Pa., Lea & Febiger, 1975, 378 pp., $11.50 (paper).*

Perinatal Addiction, *edited by Raymond D. Harbison. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1975, 384 pp., no price listed.*

Counseling and Psychology in Elementary Schools, *by Henry Kaczkowski and C.H. Patterson. Springfield, Ill., Charles C Thomas, 1975, 355 pp., $22.50.*

Personal Effectiveness: Guiding People To Assert Themselves and Improve Their Social Skills, *by Robert Paul Liberman, Larry W. King, William J. DeRisi, and Michael McCann. Champaign, Ill., Research Press, 1975, 169 pp., $5.95 (paper).*

The Practice of Mental Health Consultation, *edited by Fortune V. Mannino, Beryce W. MacLennan, and Milton F. Shore. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1975, 241 pp., $12.95.*

Legal Challenges to Behavior Modification: Trends in Schools, Corrections and Mental Health, *by Reed Martin. Champaign, Ill., Research Press, 1975, 179 pp., $5.95 (paper).*

The Hyperactive Child with Minimal Brain Dysfunction: Questions and Answers, *by J. Gordon Millichap, M.D. Chicago, Ill., Year Book Medical Publishers, 1975, 146 pp., $10.95.*

Addictions Can Be Cured: The Treatment of Drug Addiction by Neuro-Electric Stimulation, An Interim Report, *by Margaret A. Patterson, M.B.E., M.B., Ch.B. Berkhamsted, Herts, England, Lion Publishing, 1975, 95 pp., £2.95.*

Identity Group Psychotherapy with Adolescents, *by Arnold W. Rachman, Ph.D. Springfield, Ill., Charles C Thomas, 1975, 299 pp., $16.75.*

Self-Destructive Behavior, *compiled and edited by Albert R. Roberts. Springfield, Ill., Charles C Thomas, 1975, 210 pp., $17.50.*

Opening Doors: What Happens in Gestalt Therapy, *by Daniel Rosenblatt. New York, N.Y., Harper & Row, 1975, 144 pp., $7.95.*

The Politics of Drugs: Marijuana to Mainlining, *by Richard C. Schroeder. Washington, D.C., Congressional Quarterly, 1975, 205 pp., no price listed (paper).*

The Making of the Modern Family, *by Edward Shorter. New York, N.Y., Basic Books, 1975, 362 pp., $15.00.*

Handbook of Psychiatric Emergencies: A Guide for Emergencies in Psychiatry, *by Andrew E. Slaby, M.D., M.P.H., Julian Lieb, M.B., B.Ch., and Laurence R. Tancredi, M.D., J.D. Flushing, N.Y., Medical Examination Publishing Co., 1975, 187 pp., no price listed (spiral bound).*

Psychological Care of the Medically Ill: A Primer In Liaison Psychiatry, *by James J. Strain, M.D., and Stanley Grossman, M.D. New York, N.Y., Appleton-Century-Crofts (Englewood Cliffs, N.J., Prentice-Hall), 1975, 214 pp., $12.50.*

The Cognitive Unconscious: A Piagetian Approach to Psychotherapy, *by Melvin L. Weiner. Davis, Calif., International Psychological Press, 1975, 188 pp., $12.95.*

Marital Separation, *by Robert S. Weiss. New York, N.Y., Basic Books, 1975, 326 pp., $11.95.*

The Excitement of Change: A Book of Personal Growth, *by Benjamin V. White, M.D., and Helen White, M.S. New York, N.Y., Crossroad Book (Seabury Press), 1975, 214 pp., $7.95.*

Rypins' Medical Licensure Examinations: Topical Summaries and Questions, 12th ed., *by Arthur W. Wright, M.D., with the collaboration of a review panel. Philadelphia, Pa., J.B. Lippincott Co., 1975, 999 pp., $25.00.*

Program Evaluation: Alcohol, Drug Abuse, and Mental Health Services, *edited by Jack Zusman and Cecil R. Wurster. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1975, 272 pp., $14.00.*

OFFICIAL ACTIONS

The American Board of Psychiatry and Neurology

The following are those who successfully completed the Board examination given in St. Louis, Mo., Oct. 6–7, 1975.

PSYCHIATRY

Abad, Vicente, M.D., New Haven, Conn.
Abramson, Ronald, M.D., Wayland, Mass.
Agrawal, Prema, M.D., Poughkeepsie, N.Y.
Ahmed, Mohammad Basheeruddin, M.D., New Rochelle, N.Y.
Albert, Harry Dean, M.D., New York, N.Y.
Albert, Joel Stuart, M.D., Weston, Conn.
Alfie, Salomon, M.D., Riverside, R.I.
Arana, Jose D., M.D., Baltimore, Md.
Arnold, Larrie Jean Willis, M.D., Dallas, Tex.
Aviles-Roig, Francisco, M.D., Houston, Tex.

Babigian, Haroutun M., M.D., Rochester, N.Y.
Baer, Karl Gunter, M.D., San Diego, Calif.
Bakhtiar, Jamshid A.H., M.D., Shiraz, Iran
Balsam, Stephen Jay, M.D., New York, N.Y.
Bapat, Vijay Narayan, M.D., Columbia, Mo.
Barakonski, Alina M., M.D., Beverly Hills, Calif.
Baumer, Robert John, M.D., Mililani Town, Hawaii
Beitman, Bernard D., M.D., Seattle, Wash.
Bell, George S., M.D., Rydal, Pa.
Bergman, Robert L., M.D., Albuquerque, N.M.
Berkowitz, David A., M.D., Newton, Mass.
Bidwell, Robert E.G., M.D., Kansas City, Mo.
Blair, Walter B., M.D., Roanoke, Va.
Blanton-Green, Gloria N., M.D., Columbia, S.C.
Bockner, Andrew Charles, M.D., Washington, D.C.
Boekelheide, Priscilla Day, M.D., Durham, N.C.
Bongiorno, Joseph A., M.D., Lincolnshire, Ill.
Boruch, Frank, M.D., Washington, D.C.
Bradshaw, C. Marshall, M.D., Fort Worth, Tex.
Brown, Walter A., M.D., Wickford, R.I.
Brownlow, Elizabeth, M.D., Cambridge, Mass.
Bueno, Jose A., M.D., Warren Township, N.J.

Cain, Marsha L., M.D., Federal Way, Wash.
Cain, Nancy Napier, M.D., Rochester, N.Y.
Cameron, Alan S., M.D., Vienna, Va.
Carpenter, Kenneth B., M.D., Knoxville, Tenn.
Carter, J. Stephen, M.D., Greensburg, Pa.
Carter, James H., M.D., Raleigh, N.C.
Cepeda, Manuel Lawrence, M.D., Ocean Springs, Miss.
Chanel, Philippe E., M.D., Washington, D.C.
Chapin, Frederick J., M.D., Newberry, Mich.
Charuvastra, V. Charles, M.D., Los Angeles, Calif.
Chenier, Yves Edouard, M.D., New York, N.Y.
Chien, Ching-Piao, M.D., Delmar, N.Y.
Chowdhrey, M. Salim, M.D., West Orange, N.J.
Claycomb, J. Barry, M.D., Manchester, Mass.
Cline, William R., M.D., San Francisco, Calif.
Cloninger, C. Robert, M.D., St. Louis, Mo.
Cohen, Ruth Paula, M.D., New York, N.Y.
Cohen, Steven R., M.D., Pipersville, Pa.
Condron, Brian P., M.D., Lancaster, Pa.
Conklin, Thomas Joseph, M.D., Manchester, Conn.
Cook, Dennis D., M.D., San Diego, Calif.
Cooke, John Cary III, M.D., Abilene, Tex.
Cosma, Guillermo Washington, M.D., Clearwater, Fla.
Cotton, Paul Gerald, M.D., Boston, Mass.
Cowl, John Geoffrey, M.D., Pikesville, Md.
Croll, Douglas, M.D., Pomona, N.Y.
Crowder, John Edward, M.D., Cerritos, Calif.
Cunningham, Lynn Ashley, M.D., Springfield, Ill.
Curtiss, David Pierce Jr., M.D., Natick, Mass.

Dakin, Alice Richardson, M.D., Oak Park. Ill.
Daruna, Paul A., M.D., Coconut Grove, Fla.
Davis, Amos, M.D., Los Angeles, Calif.
Davis, Charles Monroe, M.D., Charlottesville, Va.
de La Chapelle, Alain, M.D., New York, N.Y.
DeLano, James Edwards Jr., M.D., Barksdale AFB, La.
De Tobal, Clara Aisenstein, M.D., Washington, D.C.
Dogan, Kutas Tavlan, M.D., Weston, Ont., Canada
Donnellan, Edmond J., M.D., Gaithersburg, Md.
Duboczy, John B., M.D., West Hartford, Conn.
Dubovsky, Steven L., M.D., Denver, Colo.
Dugan, Stanley Paul, M.D., Charleston, Ill.
Dunn, George C. Jr., M.D., Riverdale, Ga.

Ebert, Michael Hobart, M.D., Bethesda, Md.
Edmonson, Alan L., M.D., Dayton, Ohio
Edwards, Charles H., M.D., Atlanta, Ga.
Eichten, John Gregory, M.D., Minneapolis, Minn.
El-Beshir, Abdel Monem, M.D., Ellicott City, Md.
El-Yousef, M. Khaled, M.D., Indian Rocks Beach, Fla.
Etezady, M. Hossein, M.D., Gulph Mills, Pa.

Fabiani, Cesar A., M.D., Philadelphia, Pa.
Fedders, David Leo, M.D., Cincinnati, Ohio
Feldstein, Robert M., M.D., Woodland Hills, Calif.
Ferrell, Richard Bradley, M.D., Hanover, N.H.
Files, Lee Sid III, M.D., San Diego, Calif.
Fisher, Thomas Leonard, M.D., Bryn Mawr, Pa.
Flashman, Alberta R., M.D., Annapolis, Md.
Fox, David A., M.D., Brookline, Mass.
Fox, Ruth Anne, M.D., South Laguna, Calif.
Fox-Baker, Jack, M.D., Charleston Heights, S.C.
French, Alfred P., M.D., Sacramento, Calif.
Friedman, Matthew R., M.D., Arlington, Mass.

Friend, Linda Lee, M.D., San Francisco, Calif.

Gallo, Arnold J., M.D., New York, N.Y.
Gamblin, Kenneth Ray, M.D., Colorado Springs, Colo.
Garrett, Robert Wales, M.D., Okemos, Mich.
Gelenberg, Alan J., M.D., Waban, Mass.
Gibson, Robert W. Jr., M.D., Morganton, N.C.
Glicklich, Lucille Barash, M.D., Milwaukee, Wisc.
Goldin, Michael R., M.D., New York, N.Y.
Gomberg, Herbert L., M.D., New York, N.Y.
Grad, Gary Jordan, M.D., New York, N.Y.
Green, Edmon L., M.D., Nashville, Tenn.
Green, Jacquelyn K., M.D., Los Angeles, Calif.
Green, Ronald Lloyd, M.D., Thetford Hill, Vt.
Green, Wayne Hugo, M.D., New York, N.Y.
Griffin, John Joseph, M.D., Anchorage, Alaska
Groves, Gerald A., M.D., Philadelphia, Pa.
Gruen, Peter H., M.D., Larchmont, N.Y.
Gunawardhana, Hemachandra Pemasiri, M.D., St. Louis, Mo.
Gurnack, William, M.D., Boston, Mass.

Halbert, John T., M.D., Beaumont, Tex.
Hall, John Lee, M.D., Garland, Tex.
Hanson, Norman Peter, M.D., Rochester, Minn.
Hargrave, Robert L., M.D., Austin, Tex.
Harley, Ned Richard, M.D., Boulder, Colo.
Harris, G. Christian, M.D., Seattle, Wash.
Hayashida, Motoi, M.D., Moorestown, N.J.
Head, William B. Jr., M.D., Bronx, N.Y.
Heilman, Roger Harvey, M.D., Pasadena, Calif.
Hellman, Jesse M., M.D., Baltimore, Md.
Hemaya, Rizk Mishriky, M.D., Leawood, Kans.
Henderson, Harry Convery III, M.D., La Mesa, Calif.
Herrera, Edward A., M.D., Orlando, Fla.
Herron, Richard I., M.D., Chicago, Ill.
Hiatt, Barry A., M.D., St. Louis, Mo.
Hiatt, John Frederick, M.D., Berkeley, Calif.
Hill, David P., M.D., Wheeling, W. Va.
Hill, Thomas M., M.D., Hartford, Conn.
Hochberg, Richard Alan, M.D., New City, N.Y.
Hoffman, Leon, M.D., New York, N.Y.
Hogue, James C., M.D., Salem, Ore.
Husain, Syed Arshad, M.D., Columbia, Mo.
Hutzler, Jeffery Conway, M.D., Upper Arlington, Ohio

Ilaria, R., M.D., Chesapeake, Va.
Ittiara, T.L., M.D., Pontiac, Mich.

Jacobs, Gretchen Huntly, M.D., Vineyard Haven, Mass.
Jamarik, Blair Dissette, M.D., Vienna, Va.
Janes, Robert Grant, M.D., Mercer Island, Wash.
Jenks, Elisabeth R., M.D., New York, N.Y.
Jobe, Thomas Harmon, M.D., Chicago, Ill.
Johnson, Roger Alan, M.D., Burnsville, Minn.
Jones, H. Bruce, M.D., Hollywood, Fla.
Joseph, Mary, M.D., Binghampton, N.Y.
Joseph, Moolamannil, M.D., West Brentwood, N.Y.

Kang, Jae Ryong, M.D., Kings Park, N.Y.
Kaplan, Kenneth H., M.D., New York, N.Y.
Kapsalas, Angelos G., M.D., Richmond, Va.
Karush, Ruth K., M.D., New York, N.Y.
Kasuboski, David Allen, M.D., Madison, Wisc.
Kaufman, Benjamin, M.D., Sherman Oaks, Calif.
Kavka, Georgine Rotman, M.D., Chicago, Ill.
Keiter, Robert H., M.D., Richmond, Va.
Keskiner, Ali, M.D., Tarpon Springs, Fla.
Kim, Byung-Suk, M.D., Staten Island, N.Y.
Kim, Suk Sik, M.D., Woodside, N.Y.
Kleban, Carl H., M.D., New York, N.Y.
Kogan, Alan Otto, M.D., Chevy Chase, Md.
Kothari, Nauttam J., M.D., New York, N.Y.
Kriebel, George W. Jr., M.D., Providence, R.I.
Kury, Wendell M., M.D., Mt. View, Calif.
Kuster, Gordon Iverson, M.D., Orange, Conn.

Laddis, Andreas, M.D., Baltimore, Md.
Lakshminarayanan, Natarajan, M.D., St. Louis, Mo.
Lal, Shobhan, M.D., Kings Park, N.Y.
Lansky, Shirley B., M.D., Overland Park, Kans.
Lear, Charles E., M.D., Novato, Calif.
Lee, Jong Sung, M.D., Williamsville, N.Y.
LeFevre, Clinton A., M.D., Palo Alto, Calif.
Licht, Arnold L., M.D., Brooklyn, N.Y.
Lieberman, Richard Sheldon, M.D., Mill Valley, Calif.
Liem, Han, M.D., Delran, N.J.
Lipsitch, Ian I., M.D., Atlanta, Ga.
Long, William B., M.D., Omaha, Neb.
Looker, Andrew N., M.D., New York, N.Y.
Loschen, Earl Lee, M.D., Chatham, Ill.

McLaughlin, Allan Evert, M.D., Silver Spring, Md.
McMurry, Bryce E., M.D., Seattle, Wash.
Maany, Iradj, M.D., Philadelphia, Pa.
MacDonald, Richard M., M.D., San Francisco, Calif.
Malitz, Eugene, M.D., Downey, Calif.
Maltbie, Allan Armstrong, M.D., Chapel Hill, N.C.
Marcus, Michael William, M.D., Boston, Mass.
Margini, Lorenzo Marcello, M.D., New York, N.Y.
Mas, Francis G., M.D., New York, N.Y.
Maxmen, Jerrold S., M.D., New York, N.Y.
Maxwell, Olen D., M.D., Stillwater, Okla.
Merowitz, Martin, M.D., Wellesley, Mass.
Michals, Timothy John, M.D., Philadelphia, Pa.
Milke, Denis J., M.D., Camp Hill, Pa.
Miller, Grant Durward, M.D., San Luis Obispo, Calif.
Milliken, A. Donald, M.D., Edmonton, Alta., Canada
Mittleman, Frederick Stuart, M.D., Topeka, Kans.
Monnelly, Edward P., M.D., Ipswich, Mass.
Montero, J.A., M.D., Oklahoma City, Okla.
Mudd, John W., M.D., New York, N.Y.
Mundassery, Sarala C., M.D., Princeton Junction, N.J.
Murkofsky, Charles A., M.D., New York, N.Y.
Mushrush, Grace Johannette, M.D., Brookline, Mass.
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Beg, Raihana, M.D., Melrose Park, Pa.
Desai, Prakash, M.D., Chicago, Ill.
Elizan, Teresita S., M.D., New York, N.Y.
Escueta, Antonio V., M.D., Durham, N.C.
Hossain, S.M. Moazzem, M.D., Worcester, Mass.
Lee, Ho Young, M.D., Westfield, N.J.
Lee, Soo Ik, M.D., Charlottesville, Va.
Nakhla, Fayek, M.D., New York, N.Y.
Nazari, Habib Vaziri, M.D., Birmingham, Mich.
Oh, Shin Joong, M.D., Birmingham, Ala.
Pati, Prasanna K., M.D., Salem, Ore.
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Lederle Laboratories introduces

Loxitane™

LOXAPINE SUCCINATE

Lederle

A NEW CHEMICAL ENTITY FOR THE MANIFESTATIONS OF SCHIZOPHRENIA

Product of a decade of research and clinical evaluation by Lederle Laboratories, LOXITANE is a dibenzoxazepine, representing a new subclass of tricyclic antipsychotic agents. It is chemically distinct from the phenothiazines, thioxanthenes and butyrophenones.

A program of 31 studies[1] by 18 investigators included 11 controlled studies of 441 adult patients to determine efficacy of LOXITANE by comparing it with two reference agents, chlorpromazine and trifluoperazine.

Patient types	No. of Studies	No. of Loxapine Subjects	No. of Chlorpromazine Subjects	No. of Trifluoperazine Subjects
Acute Schizophrenic*	5	98	70	27
Chronic Schizophrenic†	6	123	39	84
Totals	11	221	109	111

*Studies carried out with newly admitted patients with acute schizophrenic symptoms.
†Studies carried out with patients resident in psychiatric wards for at least a year.

Psychiatric and behavioral symptoms were assessed by the Brief Psychiatric Rating Scale (BPRS) and the Nurses' Observation Scale for Inpatient Evaluation (NOSIE). Response was measured by statistical analysis comparing symptom severity before and after treatment. The controlled studies demonstrated the efficacy of LOXITANE and the two reference agents. The graphs that follow show the substantial reduction in schizophrenic symptoms achieved by the LOXITANE patient group as expressed in terms of percent of Maximum Possible Improvement.

1. Data on file, Clinical Research Department, Lederle Laboratories



Now in schizophrenia—efficacy that speaks for itself..

SEE LAST PAGE OF THIS ADVERTISEMENT FOR FULL PRESCRIBING INFORMATION.

Lederle Laboratories introduces

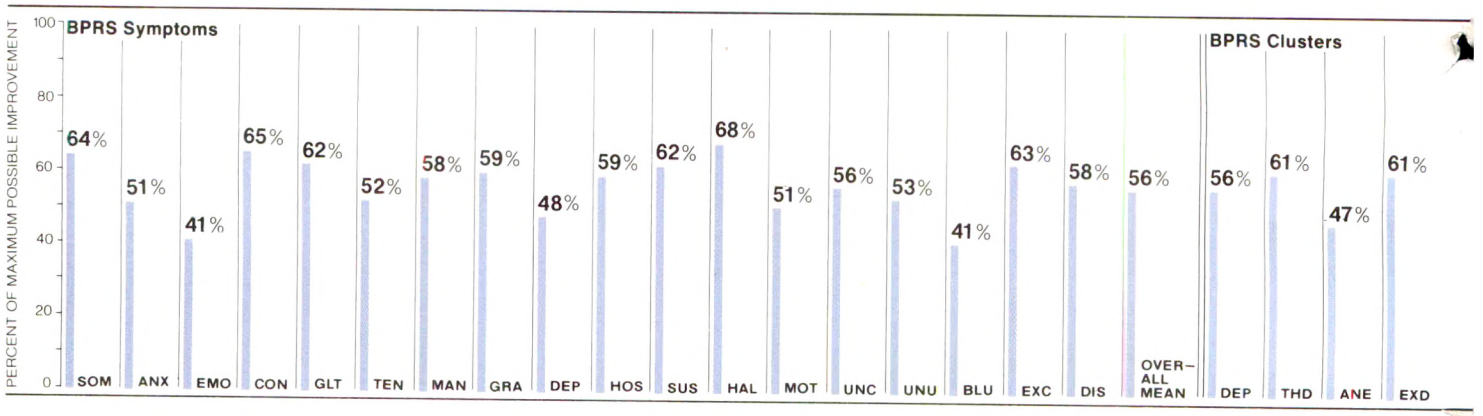


Loxitane™
LOXAPINE SUCCINATE
Lederle

SUBSTANTIALLY REDUCES THE SYMPTOMS OF SCHIZOPHRENIA

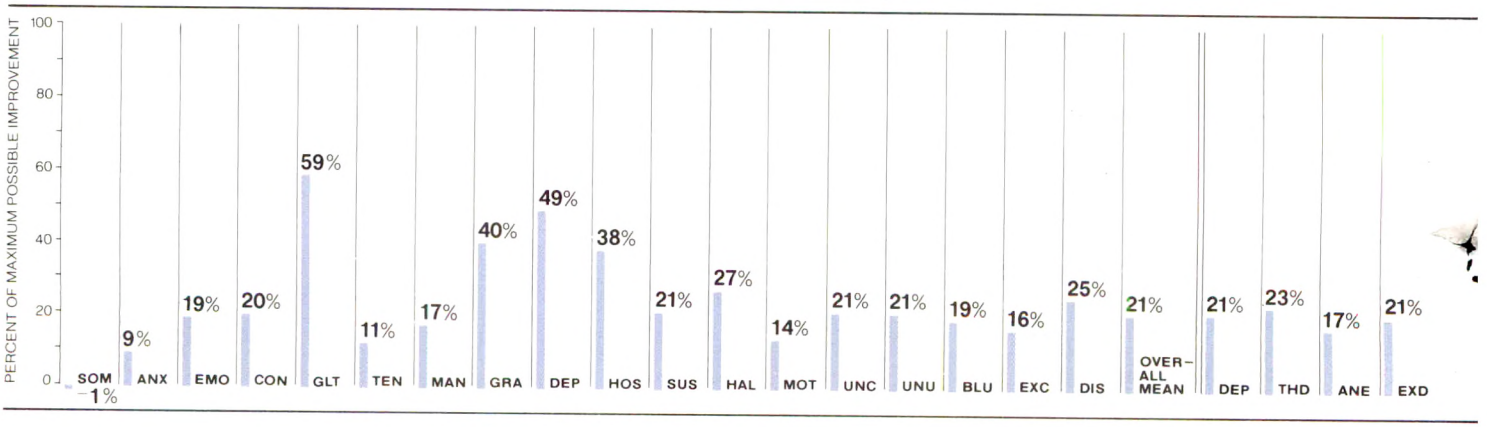
In 5 studies involving 98 acute schizophrenic patients treated for 3 to 6 weeks...

Patients treated with LOXITANE Loxapine reached an average of 56% of Maximum Possible Improvement in 18 symptoms and symptom clusters



In 6 studies involving 123 chronic schizophrenic patients treated for 8 to 12 weeks...

Severity of schizophrenic symptoms was appreciably reduced despite the typically lower responsiveness of the chronic patient



In 31 long-term, dose-range finding and controlled efficacy studies involving 469 patients...

Certain favorable trends were exhibited in the LOXITANE side effects profile, but these require further tests and broader clinical experience for confirmation

No endocrine abnormalities have been reported with LOXITANE. Hepatotoxicity manifested by jaundice or biliary stasis has not been observed. Transient liver enzyme changes, however, have been reported, but it has not been determined whether they are related to LOXITANE administration.

Although a few cases of changes in ECG have been reported, a causal relationship to LOXITANE has not been established. Clinical experience with LOXITANE has not demonstrated ocular toxicity; however, the possibility of its occurrence cannot be ruled out at this time. Manifestations of adverse effects on the central nervous system other than extrapyramidal symptoms have been encountered infrequently, and drowsiness, when it occurs, is usually mild and subsides with continued therapy. Skin rashes of uncertain etiology have been observed in a few patients during the hot summer months; therefore the possibility of phototoxicity and/or photosensitivity cannot be excluded.

In general, LOXITANE presents a profile of extrapyramidal side effects similar to that of other agents used in the treatment of schizophrenia. Parkinson-like symptoms and akathisia are usua not severe and can be controlled by reduction in LOXITANE dosage or by administration of antiparkinson drugs. Dystonia and dyskinetic reactions, while less frequent, may be more severe and may require reduction or temporary withdrawal of LOXITANE dosage, as well as treatment with anticholinergic agents. As with all antipsychotic agents, tardive dyskinesia ma appear in some patients on long-term therapy or after drug therapy has been discontinued. It is suggested that treatment w any antipsychotic agent be suspended should this reaction occ

Cardiovascular effects such as hypotension, hypertension, light headedness and syncope have been reported. Increased pulse rate has been reported in a majority of patients receiving antipsychotic doses. Anticholinergic effects seen with LOXITANE include dry mouth, nasal congestion, constipation and blurred vision.

See LOXITANE package insert for warnings and precautions ar for more detailed information concerning side effects.



Loxitane™
LOXAPINE SUCCINATE
Lederle

A range of dosage forms to suit the patient's individual needs

Supplied

Capsules	10 mg Green and Yellow	25 mg Green Two Tone	50 mg Green and Blue

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d. or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 Days	Increase dosage until psychotic symptoms are controlle Dosage should not exceed 250 mg/day Usual dosage during titration 50 to 150 mg/day		
Maintenance Dosage	Adjust to lowest effective level Usual maintenance dosage 60 to 100 mg/day Many patients are controlled with dosages as low as 2(to 60 mg/day		

SEE LAST PAGE OF THIS ADVERTISEMENT FOR FULL PRESCRIBING INFORMATION.

FOR ORAL USE

PRODUCT INFORMATION

DESCRIPTION

LOXITANE® *loxapine*, a dibenzoxazepine compound, represents a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines. Chemically, it is 2 - chloro - 11 - (4 - methyl - 1 - piperazinyl) dibenz [b,f] [1,4] oxazepine. It is present in capsules as the succinate salt. Each 1.36 mg of loxapine succinate is equivalent to 1 mg of loxapine.

ACTIONS

Pharmacologically, loxapine is a tranquilizer for which the exact mode of action has not been established. However, changes in the level of excitability of subcortical inhibitory areas have been observed in several animal species in association with such manifestations of tranquilization as calming effects and suppression of aggressive behavior.

In normal human volunteers, signs of sedation were seen within 20 to 30 minutes after administration, were most pronounced within 1½ to 3 hours, and lasted through 12 hours. Similar timing of primary pharmacologic effects was seen in animals.

Absorption of loxapine following oral or parenteral administration is virtually complete. The drug is removed rapidly from the plasma and distributed in tissues. Animal studies suggest an initial preferential distribution in lungs, brain, spleen, heart, and kidney. Loxapine is metabolized extensively and is excreted mainly in the first 24 hours. Metabolites are excreted in the urine in the form of conjugates and in the feces unconjugated.

INDICATIONS

LOXITANE *loxapine succinate* is indicated for the manifestations of schizophrenia.

CONTRAINDICATIONS

LOXITANE is contraindicated in comatose or severe drug-induced depressed states (alcohol, barbiturates, narcotics, etc.)

LOXITANE is contraindicated in individuals with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Safe use of LOXITANE *loxapine succinate* during pregnancy or lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the benefits of treatment be weighed against the possible risks to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from midpregnancy with doses of 0.6 and 1.8 mg/kg, doses which approximate the usual human dose but which are considerably below the maximum recommended human dose.

Usage in Children: Studies have not been performed in children; therefore this drug is not recommended for use in children below the age of 16.

LOXITANE, like other tranquilizers, may impair mental and/or physical abilities, especially during the first few days of therapy. Therefore, ambulatory patients should be warned about activities requiring alertness (eg, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

PRECAUTIONS

LOXITANE loxapine succinate should be used with extreme caution in patients with a history of convulsive disorders since it lowers the convulsive threshold. Seizures have been reported in epileptic patients receiving LOXITANE at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy.

Loxapine has an antiemetic effect in animals. Since this effect may also occur in man, loxapine may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

LOXITANE should be used with caution in patients with cardiovascular disease. Increased pulse rates have been reported in the majority of patients receiving antipsychotic doses; transient hypotension has been reported. In the presence of severe hypotension requiring vasopressor therapy, the preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine.

The possibility of ocular toxicity from loxapine cannot be excluded at this time. Therefore, careful observation should be made for pigmentary retinopathy and lenticular pigmentation since these have been observed in some patients receiving certain other antipsychotic drugs for prolonged periods.

Because of possible anticholinergic action, the drug should be used cautiously in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

ADVERSE REACTIONS

CNS Effects: Manifestations of adverse effects on the central nervous system, other than extrapyramidal effects, have been seen infrequently. Drowsiness, usually mild, may occur at the beginning of therapy or when dosage is increased.

It usually subsides with continued LOXITANE therapy. The incidence of sedation has been less than that of certain aliphatic phenothiazines and slightly more than the piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported.

Extrapyramidal Reactions—Neuromuscular (extrapyramidal) reactions during the administration of LOXITANE *loxapine succinate* have been reported frequently, often during the first few days of treatment. In most patients, these reactions involved Parkinson-like symptoms such as tremor, rigidity, excessive salivation, and masked facies. Akathisia (motor restlessness) also has been reported relatively frequently. These symptoms are usually not severe and can be controlled by reduction of LOXITANE dosage or by administration of antiparkinson drugs in usual dosage. Dystonic and dyskinetic reactions have occurred less frequently, but may be more severe. Dystonias include spasms of muscles of the neck and face, tongue protrusion, and oculogyric movement. Dyskinetic reaction has been described in the form of choreo-athetoid movements. These reactions sometimes require reduction or temporary withdrawal of LOXITANE dosage in addition to appropriate counteractive drugs.

Persistent Tardive Dyskinesia—As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movement of the tongue, face, mouth, or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia; antiparkinson agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Cardiovascular Effects: Tachycardia, hypotension, hypertension, light-headedness, and syncope have been reported.

A few cases of ECG changes similar to those seen with phenothiazines have been reported. It is not known whether these were related to loxapine administration.

Skin: Dermatitis, edema (puffiness of face), pruritus, and seborrhea have been reported with loxapine. The possibility of photosensitivity and/or phototoxicity occurring has not been excluded; skin rashes of uncertain etiology have been observed in a few patients during hot summer months.

Endocrine Effects: No endocrine abnormalities have been reported.

Anticholinergic Effects: Dry mouth, nasal congestion, constipation, and blurred vision have occurred; these are more likely to occur with concomitant use of antiparkinson agents.

Other Adverse Reactions: Nausea, vomiting, weight gain, weight loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, and polydipsia have been reported in some patients.

DOSAGE AND ADMINISTRATION

LOXITANE *loxapine succinate* is administered orally usually in divided doses two to four times a day. Daily dosage (in terms of base equivalents) should be adjusted to the individual patient's needs as assessed by the severity of symptoms and previous history of response to antipsychotic drugs. Initial dosage of 10 mg twice daily is recommended, although in severely disturbed patients initial dosage up to a total of 50 mg daily may be desirable. Dosage should then be increased fairly rapidly over the first seven to ten days until there is effective control of psychotic symptoms. The usual therapeutic and maintenance range is 60 mg to 100 mg daily. However, as with other antipsychotic drugs, some patients respond to lower dosage and others require higher dosage for optimal benefit. Daily dosage higher than 250 mg is not recommended. For maintenance therapy, dosage should be reduced to the lowest level compatible with symptom control; many patients have been maintained satisfactorily at dosages in the range of 20 mg to 60 mg daily.

HOW SUPPLIED

LOXITANE *loxapine succinate* is supplied in the following base equivalent strengths:

CAPSULES Hard Shell Printed "Lederle"

10 mg—Green and Yellow; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5360.

25 mg—Green Two Tone; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5361.

50 mg—Green and Blue; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5362.

REV. 3/75

A new chemical entity for the manifestations of schizophrenia



Loxitane™
LOXAPINE SUCCINATE
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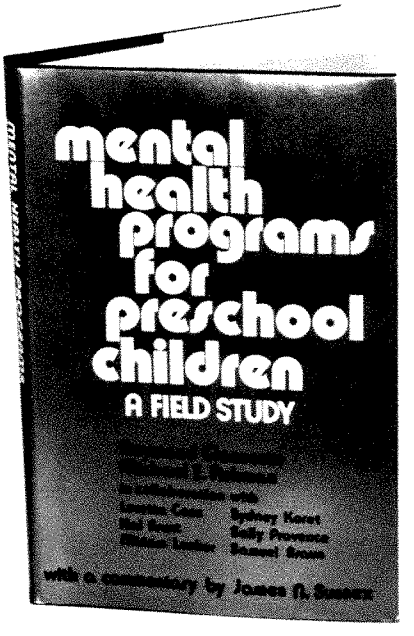
Because schizophrenia is symptoms

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A Timely New Book from The Joint Information Service

This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



An articulate exposition of the immense problems and encouraging accomplishments in a particularly difficult area of endeavor . . . . Beyond their concern with the manifestly ill child, the authors intriguingly explore what may be needed to enhance the mental good health of all young children . . . . A significant contribution.

James N. Sussex, M.D.
President, American Association of Psychiatric Services for Children

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Suspicious and hostile?



Consider the advantages of starting her on HALDOL® (haloperidol)

Acts promptly to control hostility and suspiciousness

Several clinicians have cited the special value of HALDOL haloperidol in controlling hostility, suspiciousness, ideas of persecution and other delusions associated with psychoses.[1-6] Symptom control is achieved rapidly, with many patients showing distinct improvement in a few days to a week[1,6]—frequently within a few hours when the intramuscular form is used for initial control of acutely agitated psychotic states.[7,8]

Usually leaves patients relatively alert and responsive

Although some instances of drowsiness have been observed, marked sedation with HALDOL haloperidol is rare. In fact, this drug has been reported to increase activity in some underactive patients, while in hyperactive patients it usually reduces activity to a normal level without somnolence. HALDOL haloperidol has been found to "normalize" behavior and produce a sensitivity to the environment that allows more effective use of the social milieu and the therapeutic community.[4]

Reduces likelihood of certain adverse reactions

HALDOL haloperidol, a butyrophenone, avoids or minimizes many of the problems associated with the phenothiazines. The most commonly noted side effects of HALDOL haloperidol—extrapyramidal reactions—are usually dose-related and readily controlled with antiparkinson drugs.

Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. HALDOL haloperidol is also unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes. Ocular damage and pigmentation changes have not been reported.

References: 1. Towler, M.L., and Wick, P.H.: Int. J. Neuropsychiat. 3: Suppl. 1,62 (Aug.) 1967. 2. Crane, G.E.: Int. J. Neuropsychiat. 3: Suppl. 1,111 (Aug.) 1967. 3. Ban, T.A., and Lehmann, H.E.: Int. J. Neuropsychiat. 3: Suppl. 1,79 (Aug.) 1967. 4. Haward, L.R.C.: Clin. Trials J. 2:135 (May) 1965. 5. Yun, B.S., et al.: Mich. Med. 67:1349 (Nov.) 1968. 6. Rubin, R.: Alabama J. Med. Sci. 8:414 (Oct.) 1971. 7. Reschke, R.W.: Dis. Nerv. Syst. 35:112 (Mar.) 1974. 8. Palestine, M.L., and Alatorre, E.: Quart. J. Stud. Alcohol 34:185 (Mar.) 1973.

HALDOL®
(haloperidol)
tablets/concentrate/injection

highly specific control of disordered and disruptive behavior

For information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions, please turn page.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

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A rapid-acting injection for psychiatric emergencies: 5 mg. per cc., with 0.5 mg. methylparaben and 0.05 mg. propylparaben per cc., and lactic acid for pH adjustment to 3.4±0.2.

Indications: HALDOL haloperidol is indicated for use in the management of manifestations of psychotic disorders.

It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9/74

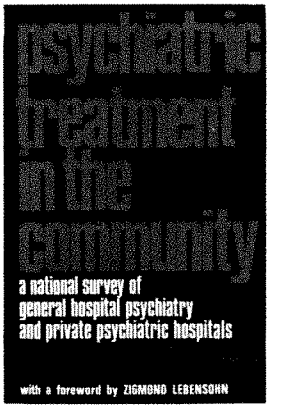
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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.
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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.
Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
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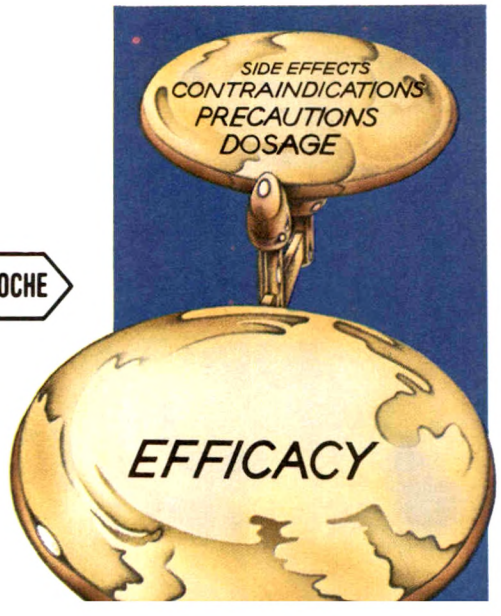
The first is more easily established. The second may be ascertained by comparison. In other words, with safety there is the question of balancing a drug's potential benefits against any potential risks.

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Indications: Norpramin (desipramine hydrochloride tablets N.F.) is indicated for the relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Desipramine hydrochloride should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Contraindications include the acute recovery period following myocardial infarction and hypersensitivity to the drug. Cross sensitivity with other dibenzazepines is a possibility.

Warnings: 1. Extreme caution should be used in patients: (a) with cardiovascular disease. (b) with a history of urinary retention or glaucoma. (c) with thyroid disease or those on thyroid medication. (d) with a history of seizure disorder. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. *Use in Pregnancy:* Safe use during pregnancy and lactation has not been established. 4. *Use in Children:* Norpramin is not recommended for use in children. 5. This drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. Therefore, the patient should be cautioned accordingly.

Precautions: This drug should be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with drugs of this class. If possible, dispense in child-resistant containers. It should be kept out of reach of children. Reduce dosage, or alter treatment, if serious adverse effects occur. Norpramin therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates and may cause exacerbation of psychosis in schizophrenic patients. Use cautiously with anticholinergic or sympathomimetic drugs. Response to alcoholic beverages may be exaggerated. In the concurrent administration of ECT and antidepressant drugs one should consider the possibility of increased risk relative to benefits. Discontinue as soon as possible prior to elective surgery because of possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy: the drug should be discontinued if there is neutropenia.

Adverse Reactions: *Cardiovascular:* hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke. *Psychiatric:* confusional states (especially in the elderly), hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis. *Neurological:* numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus. *Anticholinergic:* dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, hypotonic bladder. *Allergic:* skin rash, petechiae, urticaria, itching, photosensitization, edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs. *Hematologic:* bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue. *Endocrine:* gynecomastia, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels. *Other:* jaundice (simulating obstructive), altered liver function; weight gain or loss, perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia. *Withdrawal Symptoms:* Though not indicative of addiction, abrupt cessation after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: *The usual adult dose:* 50 mg. three times daily; increase if necessary after 7 to 10 days to maximum of 200 mg. daily. Dosages above 200 mg. per day are not recommended. *Maintenance:* At a lower dose adequate to maintain remission. *Adolescent and geriatric patient dose:* 25 to 50 mg. daily; increase to 100 mg. daily if necessary.

Overdosage: There is no specific antidote for desipramine, nor are there specific phenomena of diagnostic value characterizing poisoning by the drug. The principles of management of coma and shock by means of the mechanical respirator, cardiac pacemaker, monitoring of central venous pressure and regulation of fluid and acid-base balance are well known in most medical centers. If heart failure is imminent, digitalize promptly.

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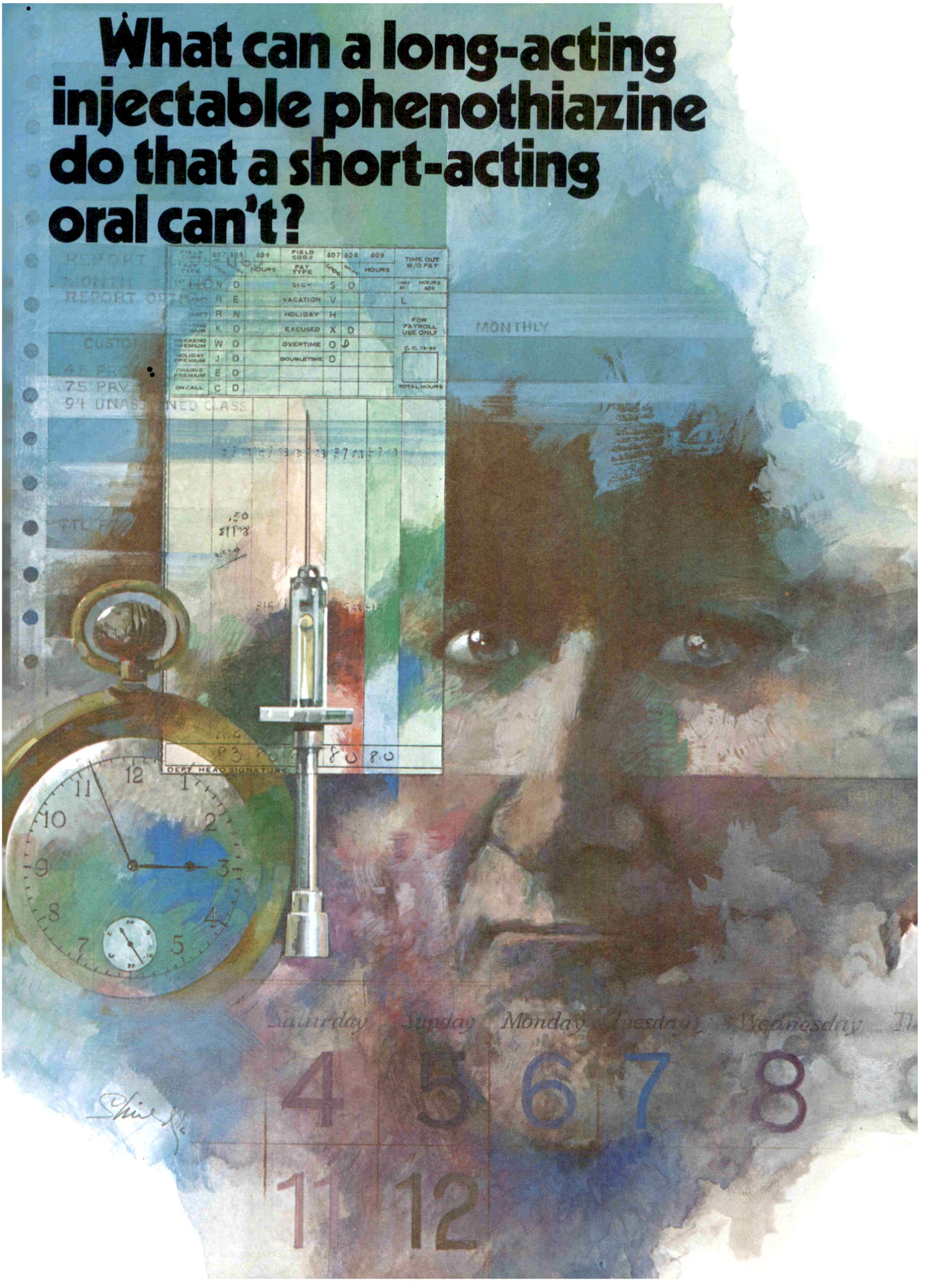
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It can save time, it can save money, and it can even save people – by reducing readmissions.

Time Prolixin Decanoate (Fluphenazine Decanoate Injection), with duration of action that may last up to 4 weeks or longer in patients on maintenance therapy, can effect important savings in nursing time.

Approximate Staff Time Required to Medicate 18 Schizophrenic Patients*

8 a.m. ¾ hr. 1 p.m. ¾ hr. 6 p.m. ¾ hr.	1 injection every 28 days for most patients
= 2¼ hours nursing time	4 minutes required for each injection
2¼ hrs. x 28 days = 63 hours of nursing time every 28 days	= 1 hr., 10 minutes nursing time in 28 days
NURSING TIME SAVED IN 28 DAYS	61 hours and 50 minutes or more than 7½ eight-hour working days

*Adapted from Platt R: Br J Social Psychiatry 2: 187-191, 1968.

Money Martin and Townend[1], who found from their study of Prolixin Decanoate that 15 of 39 patients could be maintained on 25 mg. every 4 weeks, also note that on this basis "a year's maintenance on chlorpromazine represents the order of 110 grams of phenothiazine as opposed to 0.33 gram of fluphenazine." Translating this into dollars shows the following potential hospital savings on the basis of patient population:

Agent and amount/year	Form	Cost/patient/year	Cost/500 patients/year
Chlorpromazine (SKF brand) 110 grams	8 oz. concentrate 100 mg./ml.†	$48.75*	$24,375
Prolixin Decanoate .33 grams	5 ml. vials 25 mg./ml.	23.17**	11,585
SAVINGS		$25.58 patient, year	$12,790 500 pts/yr.

* Calculated from prices published in 1975 Red Book.
** Calculated from Squibb 1975 price catalogue.
† With tablets, the annual cost is even greater.



People The controlled drug delivery system of Prolixin Decanoate helps get schizophrenic patients out of the hospital and helps them stay out. It promotes continuity of therapy—reducing outpatient drug defaulting from approximately 50% with oral medications according to one report[2] to about 16% according to another report[3].

It reduces the number and length of rehospitalizations. In one study of 103 patients maintained on injections of long-acting fluphenazines, total hospital readmissions for a year were cut from 191 to 50 and inpatient time from 8,713 days to 1,335 days[4].

It facilitates return to a productive life. A 12-month followup of 103 discharged patients revealed 77% in full-time employment or household duties and only 23% unemployed[4].

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may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

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References:
1. Martin ICA and Townend RA: Brit J Psychiat 124: 173-6, 1974.
2. Goldberg HL, DiMascio A, Chaudhary B: Psychosomatics 11: 173-177, 1970.
3. Medical World News, February 11, 1972, p. 58H.
4. Denham J and Adamson L: Can Psychiat Assoc J 18: 235-7, 1973.

For product brief summary see following page.

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PROLIXIN DECANOATE®

(Fluphenazine Decanoate Injection)

may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia, liver damage or renal insufficiency, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

FILMS ON PSYCHIATRIC MANAGEMENT AVAILABLE FROM SQUIBB

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No default insurance

Tofranil-PM®
imipramine pamoate

Geigy

In depression

Daily Dosage Chart



1 h.s.

A major reason for drug defaulting is multiple daily dosage schedules.

Ayd* reports that 70 percent of patients default on a q.i.d. regimen; 60 percent on t.i.d.; 30 percent on b.i.d., and only 7 percent on a single daily dose.

A single daily capsule that provides a full day's medication would logically assure better therapeutic results.

Because one capsule lasts from bedtime to bedtime, Tofranil-PM may markedly reduce the probability of missed doses.

For single-dose therapy in depression when the dosage is established.

Please review the prescribing information summarized on the back of this page.

*Ayd, F.J., Jr. (Ed.): Int. Drug Ther. Newslett. 7(9/10):35-50 (Nov./Dec.) 1972.

Tofranil-PM®
imipramine pamoate

One capsule lasts from bedtime to bedtime.

Tofranil-PM® brand of imipramine pamoate
Tofranil® brand of imipramine hydrochloride USP

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil, brand of imipramine hydrochloride, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

- patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
- patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
- hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
- patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
- patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Usage in Children: Pending evaluation of results from clinical trials in children, Tofranil, brand of imipramine hydrochloride, is not recommended for treatment of depression in patients under twelve years of age.

Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as operating an automobile or machinery, the patient should be cautioned accordingly.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil, brand of imipramine hydrochloride, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil, brand of imipramine hydrochloride, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine hydrochloride should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

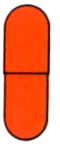
Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Tofranil-PM®
imipramine pamoate

In depression

Daily Dosage Chart



1 h.s.

Geigy

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: Lower dosages are recommended for elderly patients and adolescents. Lower dosages are also recommended for outpatients as compared to hospitalized patients who will be under close supervision. Dosage should be initiated with Tofranil, brand of imipramine hydrochloride, at a low level and increased gradually, noting carefully the clinical response and any evidence of intolerance. Following remission, maintenance medication may be required for a longer period of time, at the lowest dose that will maintain remission.

Once-a-day maintenance dosage can be provided with Tofranil-PM, brand of imipramine pamoate, capsules if this dosage has been established as explained above. This dose may be given at bedtime. For the occasional patient who manifests stimulation and insomnia with this dosage regimen, the capsules may be given in the morning.

Parenteral administration should be used only for starting therapy in patients unable or unwilling to use oral medication. The oral form should supplant the injectable as soon as possible.

How Supplied: Tofranil, brand of imipramine hydrochloride: Round tablets of 25 and 50 mg.; triangular tablets of 10 mg.; and ampuls, each containing 25 mg. in 2 cc. for I.M. administration.

Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)

Printed in U.S.A. (B) 98-146-850-R (2/74)

For complete details, including dosage and administration, please refer to the full prescribing information.

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Ardsley, New York 10502

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A Survey of Academic Resources in Psychiatric Residency Training

Author: Lee Gurel, Ph.D.

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Basic tools of primitive psychiatry



Konde: Incarnation of *Ndoki*, an evil spirit which causes disease. Wish-projection "causes" illness when a nail is driven into the statue. Also used to "drive out" an illness. Bakongo, Zaire.



Mask: Represents the spirit of the underworld. Used by the Ekpo male society, which honors dead ancestors and enforces the law. Anang Ibibio, Nigeria.

Objects from the collection of the Segy Gallery, New York City.

Basic tool of Western psychiatry

Thorazine®

brand of chlorpromazine

Tablets: 25 mg. of the HCl

- 'Thorazine' controls psychotic symptoms
- Especially useful in agitated, violent or anxious schizophrenic patients
- Unsurpassed clinical experience
- 18 dosage forms and strengths

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudo-parkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only). Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg., in bottles of 50; in Single Unit Packages of 100 (intended for institutional use only). Injection, 25 mg./ml.; Syrup, 10 mg./5 ml.; Suppositories, 25 mg. and 100 mg.; Concentrate (intended for institutional use only), 30 mg./ml. and 100 mg./ml.

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SK&F

Smith Kline & French Laboratories
Division of SmithKline Corporation
Philadelphia, Pa. 19101

Effective control of psychotic agitation

Tested by time and experience in

1962

"...a considerable decrease of hyperactivity...."[1]
Knobel, 1962



Over a decade of controlled studies and clinical experience has shown the effectiveness of Ritalin in reducing the hyperactivity,[1-3] distractibility,[1,4,5] and disorganized behavior[1-8] in the MBD child.

By lessening the effects of motor and attentional disorders, Ritalin can help the MBD child to better focus his attention on meaningful stimuli and thus can often improve cognition and promote learning.[6,9]

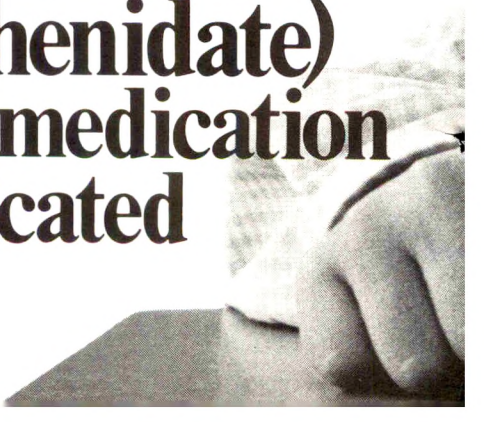
And side effects – insomnia and appetite loss – with Ritalin have occurred less frequently than with dextroamphetamine.[10,11]

Indeed, Ritalin is currently a drug of choice in many MBD situations,[10,12] and can prove to be an important element in many complete remedial programs for MBD.

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Drug treatment is not indicated for all children with MBD.

Dosage should be periodically interrupted. Often, these interruptions reveal some "stabilization" in the child's behavior even without medication, permitting a reduction in dosage and eventual discontinuance of drug therapy.

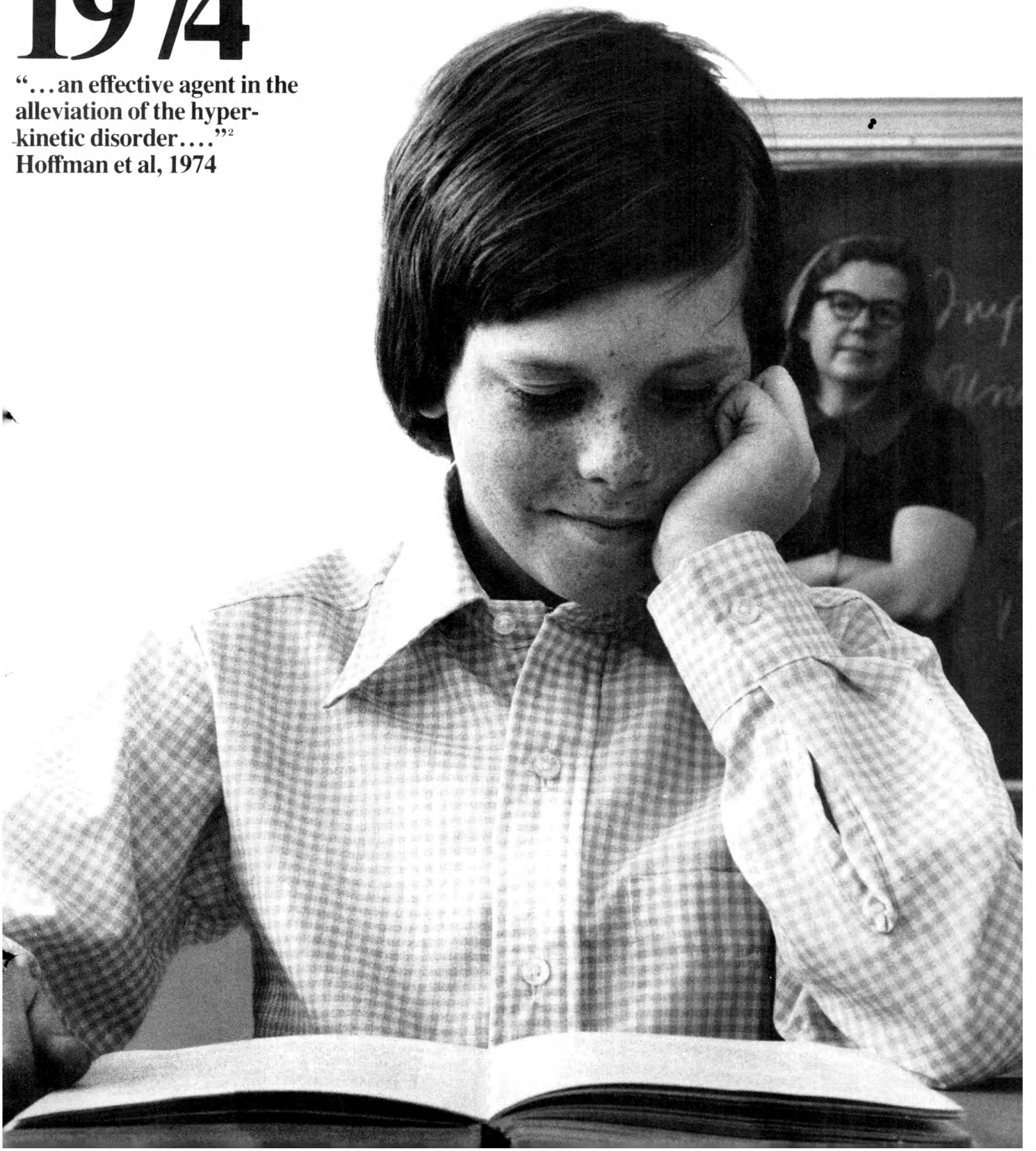
Ritalin®
(methylphenidate)
Only when medication is indicated

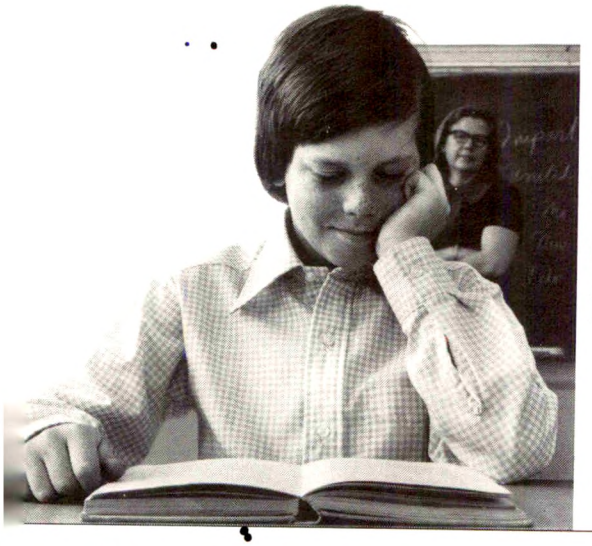


the treatment of MBD

1974

"...an effective agent in the alleviation of the hyperkinetic disorder...."[2]
Hoffman et al, 1974





Ritalin®
(methylphenidate)
Only when medication is indicated

Ritalin® hydrochloride ©
(methylphenidate hydrochloride)

TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over)

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

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2/5031 17

CIBA

SEXUAL DYSFUNCTION: BEHAVIOR THERAPY WORKSHOPS

Small group intensive training workshops emphasizing therapy techniques for use by mental health professionals and physicians who deal with sexual complaints.

The following workshops will be offered: Progressive Relaxation; Systematic Desensitization; The Use of Films in Therapy; Sexual Attitude Reassessment; The Treatment of Male Sexual Dysfunction; The Treatment of Female Sexual Dysfunction; Taking a Behavioral Sex History; Facilitating Intimate Communication.

PHILADELPHIA, MARCH 5-6, 1976 — sponsored by the Behavior Therapy Unit, Department of Psychiatry, Temple University Medical School, Philadelphia, PA.

For information, please contact: Ms. B. J. Foster, Behavior Therapy Unit, Room 702, c/o E.P.P.I., Henry Avenue, Philadelphia, PA 19129. Phone (215) 438-4298.

The Princeton Center for Behavioral Consultation, in conjunction with local universities will sponsor this program in:

SAN FRANCISCO, MARCH 12-13, 1976
TORONTO, APRIL 9-10, 1976
NEW YORK, APRIL 23-24, 1976
DALLAS, MAY 8-9, 1976
HILTON HEAD, MAY 21-22, 1976

For information on these workshops, please contact: Ms. Debora Phillips, Princeton Center for Behavioral Consultation, Room 202, 53 Wilton Street, Princeton, NJ 08540.

PRINCE GEORGE REGIONAL HOSPITAL

The Prince George Regional Hospital is requesting applications from PSYCHIATRISTS. The hospital currently expanded to a 370 bed, (including a 27 bed Psychiatric Unit), fully accredited; active treatment Regional referral hospital, located in Central B.C., is situated in the City of Prince George. The present population of Prince George is approximately 70,000, and serves an area of about 150,000. Prince George is one of the fastest growing communities in the province and offers excellent educational and recreational facilities with easy access to Vancouver and other parts of Canada. Please direct enquiries to: Dr. P.J. Konkal, Chairman, Medical Manpower Committee, Prince George Regional Hospital, Prince George, B.C.

Three Treatment Centers

Three treatment centers provide individually prescribed programs of care, education, treatment and training for children, adolescents and adults with learning disabilities, neurological impairment, mental retardation or emotional disturbance.

Advantage: the availability of a large, trained and experienced staff, exchanging ideas and insights in a comprehensive program of supportive environment and individualized treatment.

Advantage: geographically separated to provide the proper residential milieu . . . yet near enough to benefit from the combined training and experience of a wide range of staff members.

The Oaks — A residential psychiatric treatment center for emotionally disturbed children and adolescents between the ages of six and 18. Co-educational, Austin. In addition, the Center provides a Short Term Adolescent Center and a six bed intensive care unit.

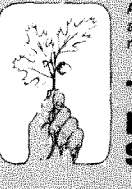
The San Marcos Treatment Center — a facility with three campuses designed for individuals with psychiatric, neurological, and intellectual difficulties who have evidenced a need for a 24-hour therapeutic milieu.

The Center offers a broad spectrum of services to both mentally retarded individuals and those with psychiatric disorders. In addition to a structured therapeutic environment, individual and group psychotherapy services are available to each resident. Clinical supervision and support services are provided by the Center psychiatrists.

The Ranch Treatment Center — located in the suburbs of Austin the Center provides both long-term and intermediate tenure services to multi-handicapped male adolescents and adults whose primary diagnostic classifications are in areas of mental retardation and mental illness. All residents receive program services which may permit their early discharge to the less restrictive settings of community-based programs.

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THE BROWN SCHOOLS

Effectiveness across the spectrum of most common forms of insomnia

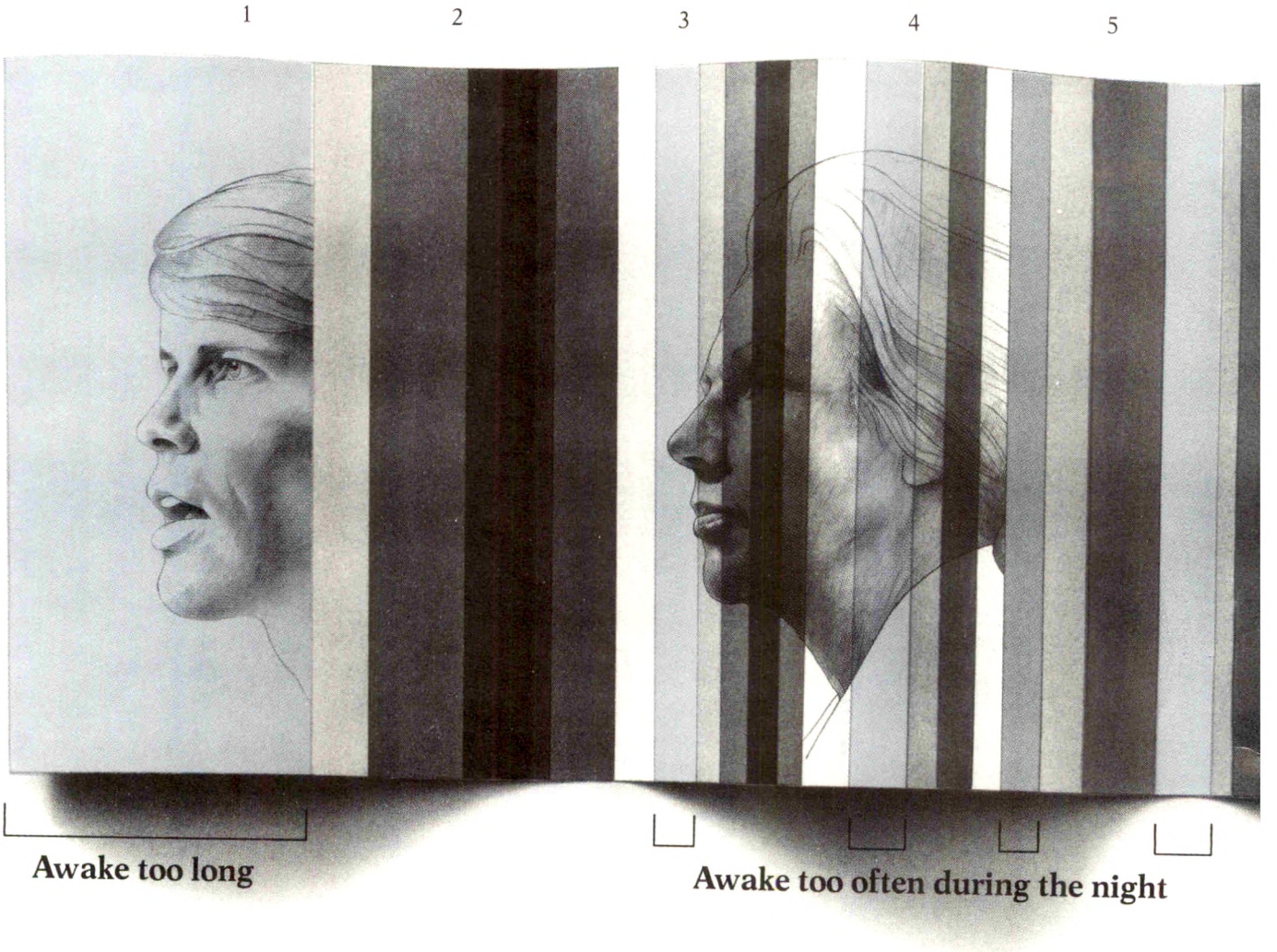
Awake too long, awake too often, awake too early...

These are the most common forms of insomnia, and may occur singly or in any combination. The night of troubled sleep depicted here comprises all three types. As the night progresses from left to right, each sleep stage is identifiable by its own shade of gray. Blue represents "Awake."

As you can see, this hypothetical "patient" takes well over an hour to fall asleep, awakens several times during the middle of the night and awakens too early in the morning.

Sleep Stages

- Awake
- REM
- Stage 1
- Stage 2
- Stage 3
- Stage 4



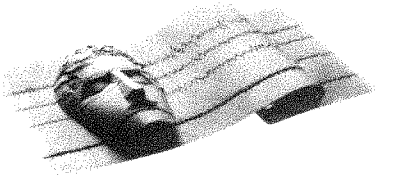
The insomnias most often occurring in young and older adults

For patients with trouble falling asleep (common in young adult insomnia patients), Dalmane (flurazepam HCl) 30 mg provides sleep within 17 minutes, on average. For those with trouble staying asleep or sleeping long enough (common in those over 50), Dalmane offers increased total sleep time with fewer nocturnal awakenings. These clinical results were demonstrated in studies conducted in four geographically separated sleep research laboratories[1-4]

The relative safety of Dalmane (flurazepam HCl) is well documented

Dalmane (flurazepam HCl) is relatively safe and well tolerated; morning "hang-over" has been infrequent. The usual adult dosage is 30 mg; in elderly or debilitated patients, limit initial dosage to 15 mg to preclude over-sedation, dizziness or ataxia. Caution patients about possible combined effects with alcohol and other CNS depressants.

Broad-spectrum medication for the most common forms of insomnia

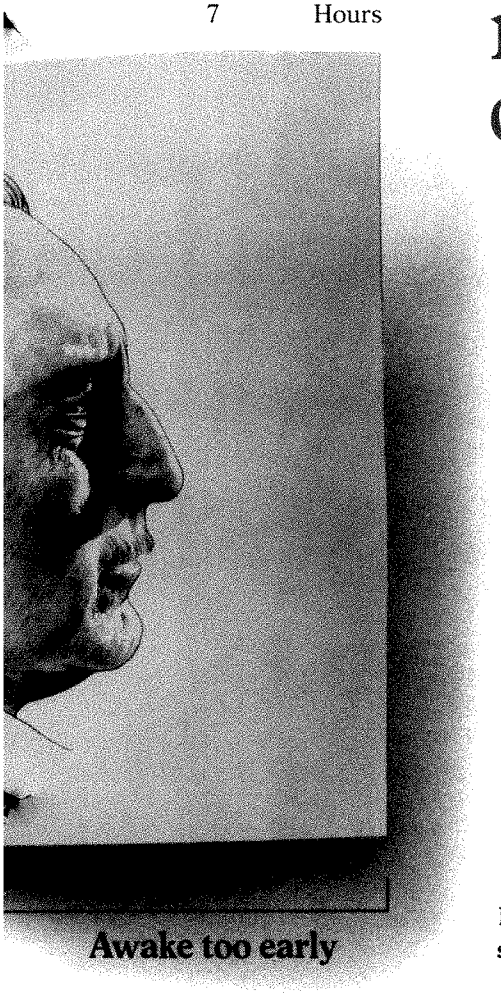


Dalmane®
(flurazepam HCl)

One 30-mg capsule *h.s.*— usual adult dosage
(15 mg may suffice in some patients).
One 15-mg capsule *h.s.*— initial dosage for elderly or debilitated patients.

- □ induces sleep rapidly
- □ reduces nighttime awakenings
- □ lengthens total sleep time

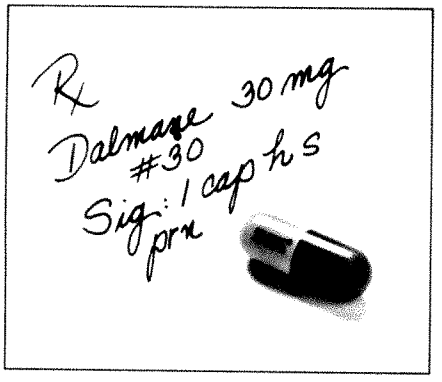
ROCHE



Please see following page for a summary of complete product information.

Broad-spectrum medication for the most common forms of insomnia

Dalmane®
(flurazepam HCl) ℞ IV



Objectively proved in the sleep research laboratory, Dalmane

- induces sleep within 17 minutes, on average
- reduces nighttime awakenings
- provides 7 to 8 hours sleep, on average, without repeating dosage

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; and in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Use in women who are or may become pregnant only when potential benefits have been weighed against possible hazards. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, initial dosage should be limited to 15 mg to preclude oversedation, dizziness and/or ataxia. If combined with other drugs having hypnotic or CNS-depressant effects, consider potential additive effects. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported were headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase. Paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, have also been reported in rare instances.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

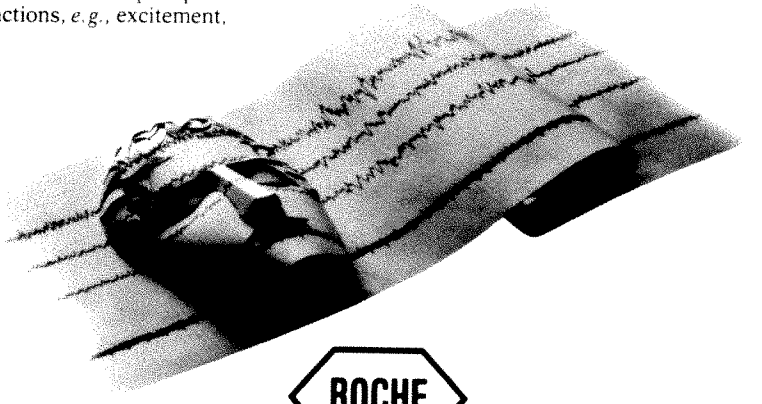
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2. Frost JD Jr: A system for automatically analyzing sleep. Scientific exhibit at the 24th Clinical Convention of the American Medical Association, Boston, Nov 29-Dec 2, 1970; and at the 42nd annual scientific meeting of the Aerospace Medical Association, Houston, Apr 26-29, 1971

3. Vogel GW: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ

4. Dement WC: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ



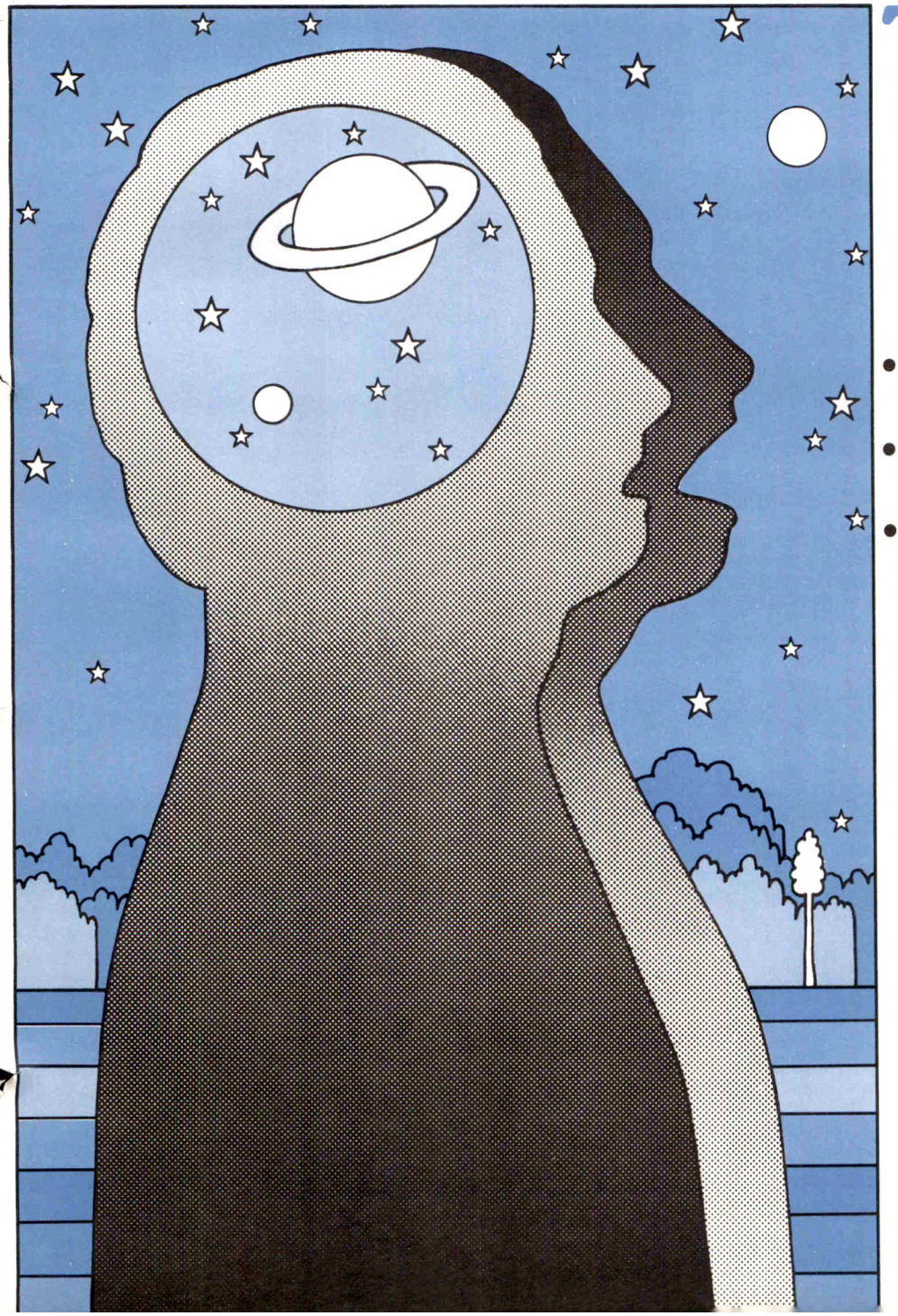
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SCHIZOPHRENIA

He believes he receives messages from outer space.



Trilafon®
brand of
perphenazine, NF

a low-dose high-potency phenothiazine for the management of disturbed behavior in schizophrenic and manic patients

- useful for in-patient and out-patient treatment
- can help orient psychiatric patients to their surroundings
- features minimal sedative effect, usually producing little lethargy or drowsiness

Versatile dosage

Injection
5 mg/ml, 1 ml ampule

Concentrate
16 mg/5 ml,
120 ml bottle

Tablets
2,4,8,16 mg

REPETABS®
Tablets 8 mg
brand of repeat-action tablets

Please see prescribing information on next page.

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Trilafon®

brand of
perphenazine, NF

for the management of schizophrenic and manic disorders

- in the psychiatric hospital
- in the mental health clinic
- in out-patient treatment

INJECTION
when you want *emergency* control for *uncooperative* in-hospital patients

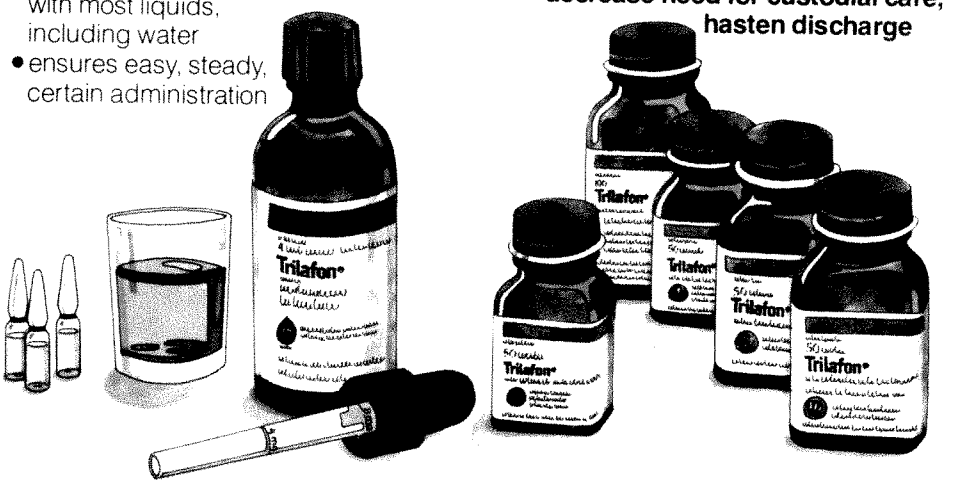
benefits
- usually takes effect in 10 minutes
- average duration of effect is 6 hours

CONCENTRATE
when you want *maintenance* control for *uncooperative* in-hospital patients

benefits
- tasteless, colorless, easily mixes with most liquids, including water
- ensures easy, steady, certain administration

TABLETS
when you want *maintenance* therapy for *cooperative* patients, in the hospital or at home

benefits
- **generally improve cooperation and communication**
- **decrease need for custodial care, hasten discharge**



Trilafon®
brand of perphenazine, NF
Tablets
REPETABS® Tablets
Concentrate
Injection

CONTRAINDICATIONS TRILAFON is contraindicated in drug-associated central nervous system depression (barbiturates, alcohol, narcotics, analgesics, antihistamines). Perphenazine is contraindicated in the presence of existing blood dyscrasias, bone marrow depression and pre-existing liver damage, and in patients who are hypersensitive to perphenazine.

TRILAFON **Injection** should not be given to patients in coma or severely depressed states.

WARNINGS **Usage in pregnancy:** Perphenazine should only be given to pregnant patients when, in the judgment of the physician, the potential benefits outweigh the possible risks.

Perphenazine can lower the convulsive threshold in susceptible individuals; it should be used with caution in patients with convulsive disorders. If the patient is being treated with an anticonvulsant agent, increased dosage of that agent may be required when perphenazine is used concomitantly.

Perphenazine should be used with caution in patients with psychic depression.

Perphenazine is not recommended for children under 12 years of age.

Perphenazine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery.

PRECAUTIONS As with any potent medication, patients receiving perphenazine should be under medical supervision, particularly if they are receiving high doses. Patients who have had any severe reaction to phenothiazines or to imipramine should be treated cautiously, under close medical supervision.

Although the following adverse reactions have not been reported in patients treated with perphenazine, the possibility that they might occur with TRILAFON should be considered: blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, eosinophilia); liver damage (biliary stasis); narrowing of the visual fields; pigmentation of the retina, cornea, or lens; cerebral edema; polyphagia; photophobia; hyperpyrexia.

If hypotension develops, levarterenol (norepinephrine) can be used, but not epinephrine, because epinephrine's action is blocked and partly reversed by perphenazine. Severe, acute hypotension has occurred with the use of phenothiazines and is of particular concern in patients with mitral insufficiency or pheochromocytoma.

A significant rise in body temperature may indicate an idiosyncratic reaction to perphenazine; treatment with perphenazine should be stopped if this occurs.

The antiemetic effect of perphenazine can obscure signs of toxicity due to overdosage of other drugs, or mask the symptoms of disease (eg, brain tumor or intestinal obstruction).

Contact dermatitis has been reported with a perphenazine solution; therefore, contact of hands or clothing by those handling perphenazine solutions should be avoided.

POTENTIATION Since phenothiazines can potentiate the central-nervous-system-depressant actions of opiates, antihistamines, barbiturates, and alcohol, less than the usual dosage of these agents is required when they are administered concomitantly with TRILAFON. Patients should be cautioned that their response to alcohol may be increased while they are being treated with TRILAFON.

Phenothiazines also potentiate the effects of atropine, heat, and phosphorus insecticides, and should be used with caution in persons exposed to these agents.

ADVERSE REACTIONS *Extrapyramidal reactions:* dystonia including protrusion, discoloration, aching and rounding of the tongue; tonic spasm of the masticatory muscles, tight feeling in the throat, slurred speech, dysphagia, oculogyric crisis, trismus, torticollis, retrocollis, muscle weakness, and aching and numbness of the limbs; akathisia; motor restlessness; dyskinesia, parkinsonism; hyperreflexia; and ataxia. The incidence and severity of these reactions usually increase with increased dosage, but have occurred in some patients receiving low dosage. Reduction in dosage or treatment with an antispasmodic agent will usually control extrapyramidal reactions. In some instances, however, these reactions may persist after discontinuation of treatment with perphenazine.

Persistent tardive dyskinesia: As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities. There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine, vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Allergic reactions: erythema, pruritus, urticaria, eczema, anaphylactoid reactions, and local and generalized edema. In extremely rare instances, individual idiosyncrasy or hypersensitivity to phenothiazines has resulted in cerebral edema, circulatory collapse, and death. Photosensitization, asthma, and exfoliative dermatitis have also occurred in patients treated with phenothiazines.

Autonomic reactions: blurred vision, dry mouth or salivation, nasal congestion, nausea, vomiting, hypertension, tachycardia, hypotension, anorexia, urinary frequency or incontinence, and constipation. Significant autonomic effects have been infrequent in patients receiving less than 24 mg. perphenazine daily.

Other reactions: endocrine disturbances (lactation, gynecomastia, galactorrhea, disturbances in the menstrual cycle), headaches, mild insomnia, altered cerebrospinal fluid proteins, ECG abnormalities, reactivation of psychosis, paradoxical excitement, paranoid-like reactions, catatonia, and systemic lupus erythematosus-like syndrome. Hypnotic effects appear to be minimal, particularly in patients who are permitted to remain active. The following adverse reactions, though rare, have also been reported to be associated with perphenazine treatment: agranulocytosis; jaundice; hyperpigmentation of the skin; grand mal convulsions; failure of ejaculation; hyperglycemia.

Side effects with intramuscular TRILAFON **Injection** have been infrequent and transient. Dizziness or significant hypotension after treatment with TRILAFON **Injection** is a rare occurrence.

NOVEMBER 1972
Schering Corporation
Kenilworth, N.J. 07033

SWW-5030

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for clinically significant depression . . .

Merck Sharp & Dohme introduces

75- and 100-mg

TABLETS

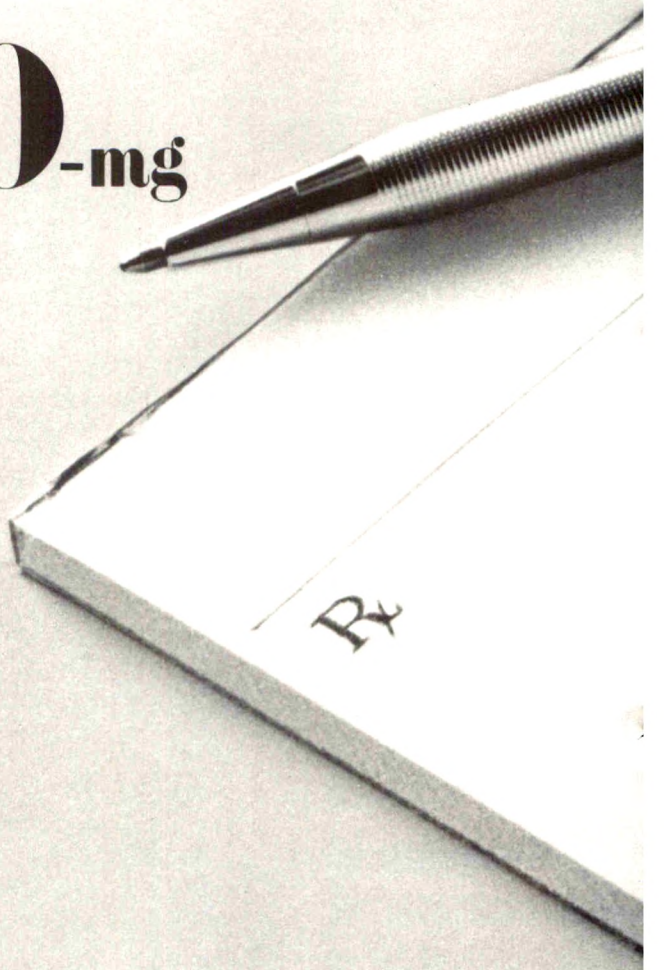
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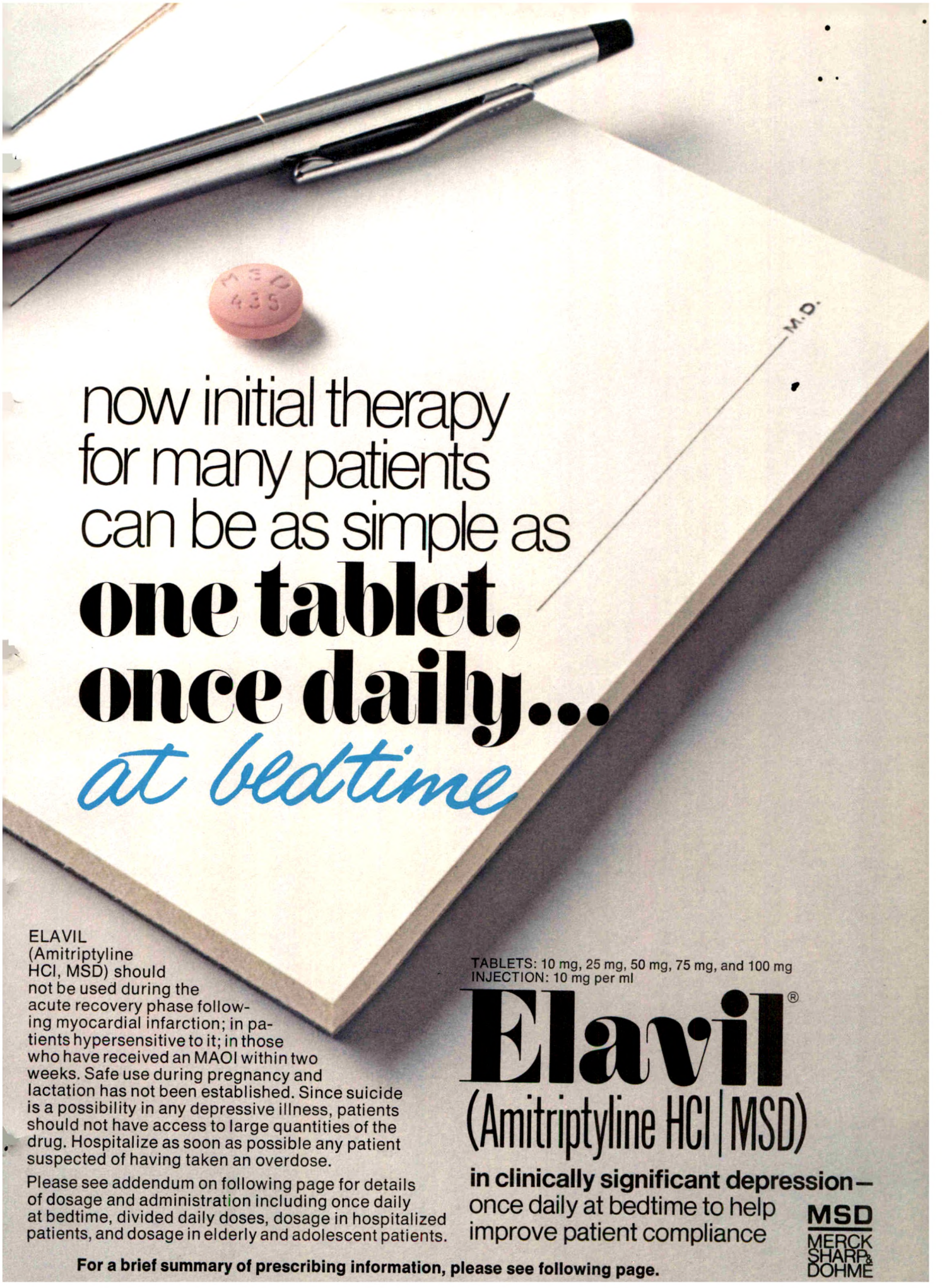
(Amitriptyline HCl | MSD)

to further simplify once-daily

h.s.

therapy





now initial therapy for many patients can be as simple as **one tablet, once daily...** *at bedtime*

ELAVIL (Amitriptyline HCl, MSD) should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks. Safe use during pregnancy and lactation has not been established. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

Please see addendum on following page for details of dosage and administration including once daily at bedtime, divided daily doses, dosage in hospitalized patients, and dosage in elderly and adolescent patients.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg
INJECTION: 10 mg per ml

Elavil®
(Amitriptyline HCl | MSD)

in clinically significant depression— once daily at bedtime to help improve patient compliance

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see following page.

NEW TABLETS

Elavil®

(Amitriptyline HCl | MSD)

75 mg and 100 mg

to further simplify once-daily

h.s.

therapy

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor or within at least 14 days following the discontinuance of a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

addendum

MSD
MERCK SHARP & DOHME

Elavil®

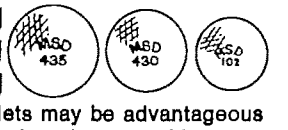
(Amitriptyline HCl | MSD)

Five tablet strengths for differing patient needs. The variety of dosage strengths available with ELAVIL makes possible great prescribing flexibility.

Once-a-day dosage at bedtime is an appropriate way to start—and maintain—many patients on therapy. The simplicity of this regimen helps improve patient compliance. ELAVIL may also be prescribed in divided daily doses.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

100 mg
75 mg
50 mg



These tablets may be advantageous when initiating therapy with once-daily bedtime dosage in adult outpatients. The usual starting dosage is 75 mg daily. Therapy may be initiated with 50 to 100 mg daily. This may be increased by 25 or 50 mg as necessary in the bedtime dose to a total of 150 mg per day.

These tablets may also be convenient for many hospitalized patients. These patients may need 100 mg a day initially which can be increased gradually to 200 mg a day if necessary. A small number of such patients may need as much as 300 mg a day.

25 mg



This tablet may prove useful when initiating therapy with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime doses.

10 mg



Because lower doses are generally recommended for adolescents and elderly patients, the 10-mg tablets may be most serviceable. Ten mg three times a day with 20 mg at bedtime may be satisfactory in adolescent and elderly patients who do not tolerate higher doses.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

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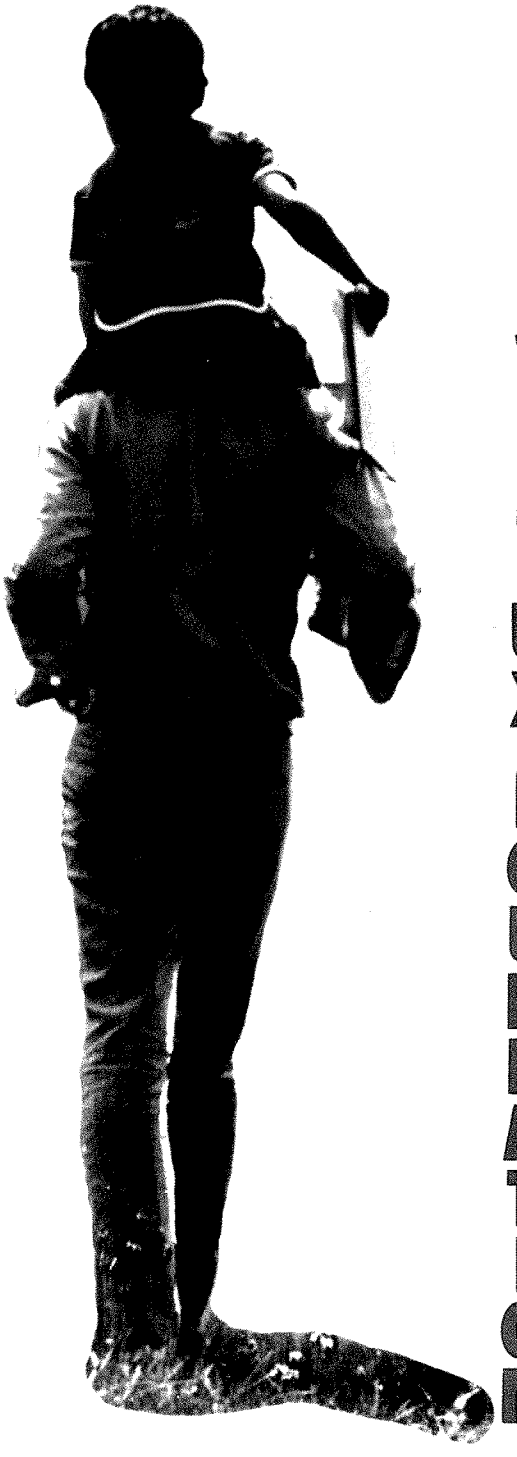
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Released from symptoms of psychotic depression...



Productive again.

By helping to reduce the frequency and intensity of psychotic symptoms, Navane (thiothixene) often permits resumption of more normal, more productive living.

The antipsychotic effectiveness of Navane—with relatively little drowsiness—helps patients remain more active, more alert, better able to meet the ordinary demands of life.

Cardiovascular side effects such as hypotension and nonspecific EKG changes are relatively rare with Navane (thiothixene). Extrapyramidal symptoms have been reported, but are usually controlled by reduction in dosage and/or administration of antiparkinson drugs.

Once you've controlled acute psychosis or psychotic depression, some patients can be maintained on a simple once-a-day dose. This once-a-day regimen can reduce the risk of missed doses with no loss of efficacy.

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New York, New York 10017



Navane®

(thiothixene) (thiothixene hydrochloride)

Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg/Concentrate 5 mg/ml

Once-a-day–to help control symptoms of psychosis

For prescribing information, including adverse reactions and contraindications please see following page of this advertisement.

Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg. of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

More detailed professional information available on request.

ROERIG Pfizer
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New York, New York 10017

Three Treatment Centers

Three treatment centers provide individually prescribed programs of care, education, treatment and training for children, adolescents and adults with learning disabilities, neurological impairment, mental retardation or emotional disturbance.

Advantage: the availability of a **large, trained and experienced staff,** exchanging ideas and insights in a comprehensive program of supportive environment and individualized treatment.

Advantage: **geographically separated** to provide the proper residential milieu . . . yet near enough to benefit from the combined training and experience of a wide range of staff members.

The Oaks — A residential psychiatric treatment center for emotionally disturbed children and adolescents between the ages of six and 18. Coeducational, Austin. In addition, the Center provides a Short Term Adolescent Center and a six bed intensive care unit.

The San Marcos Treatment Center — a facility with three campuses designed for individuals with psychiatric, neurological, and intellectual difficulties who have evidenced a need for a 24-hour therapeutic milieu.

The Center offers a broad spectrum of services to both mentally retarded individuals and those with psychiatric disorders. In addition to a structured therapeutic environment, individual and group psychotherapy services are available to each resident. Clinical supervision and support services are provided by the Center psychiatrists.

The Ranch Treatment Center — located in the suburbs of Austin the Center provides both long-term and intermediate tenure services to multi-handicapped male adolescents and adults whose primary diagnostic classifications are in areas of mental retardation and mental illness. All residents receive program services which may permit their early discharge to the less restrictive settings of community-based programs.

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Jackson R. Day, M.D./Medical and Psychiatric Director
James L. Boynton, M.D./Director, The Oaks Residential Treatment Center
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Orrie L. Forbis, Jr., M.D./Staff Psychiatrist
Other Medical Staff: Thomas F. Caldwell, D.D.S./ Patrick A. Cato, M.D./Willis M. Thorstad, M.D.



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THE BROWN SCHOOLS

The April 1976 issue of

The American Journal of Psychiatry

will feature

- **Nancy A. Roeske on Women in Psychiatry**
- **Fall Committee Meetings**

for clinically significant depression. . .

Merck Sharp & Dohme introduces

75- and 100-mg

TABLETS

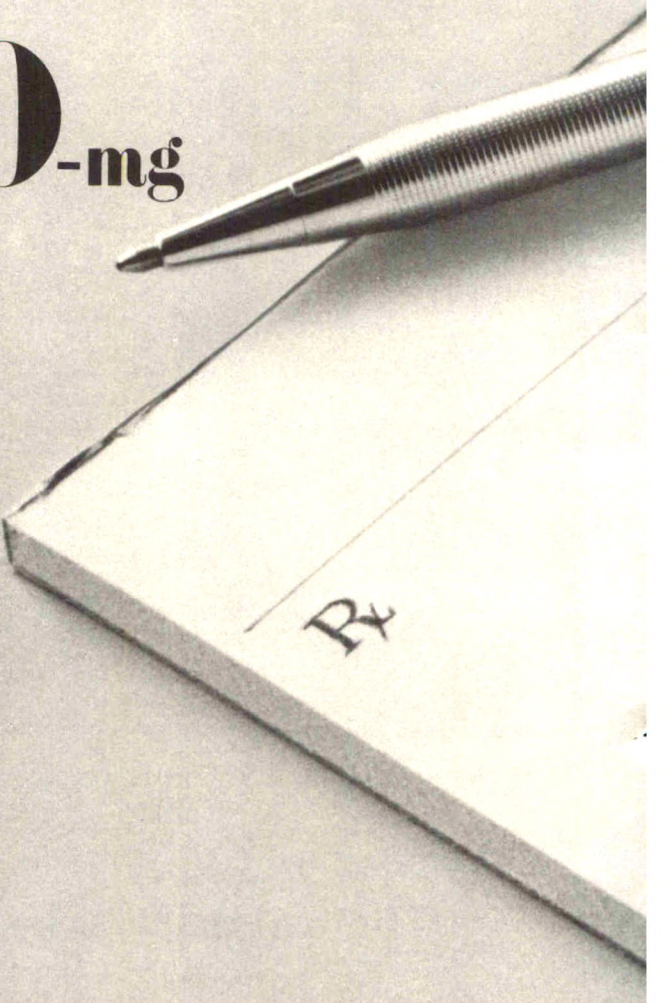
Elavil®

(Amitriptyline HCl | MSD)

to further simplify once-daily

h.s.

therapy





now initial therapy for many patients can be as simple as **one tablet, once daily...** *at bedtime*

ELAVIL (Amitriptyline HCl, MSD) should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks. Safe use during pregnancy and lactation has not been established. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

Please see addendum on following page for details of dosage and administration including once daily at bedtime, divided daily doses, dosage in hospitalized patients, and dosage in elderly and adolescent patients.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg
INJECTION: 10 mg per ml

Elavil®
(Amitriptyline HCl | MSD)

in clinically significant depression— once daily at bedtime to help improve patient compliance

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see following page.

NEW TABLETS

Elavil®

(Amitriptyline HCl | MSD)

75 mg and 100 mg

to further simplify once-daily

h.s.

therapy

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor or within at least 14 days following the discontinuance of a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

addendum

MSD
MERCK SHARP & DOHME

Elavil®

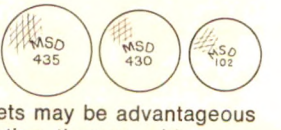
(Amitriptyline HCl | MSD)

Five tablet strengths for differing patient needs. The variety of dosage strengths available with ELAVIL makes possible great prescribing flexibility.

Once-a-day dosage at bedtime is an appropriate way to start—and maintain—many patients on therapy. The simplicity of this regimen helps improve patient compliance. ELAVIL may also be prescribed in divided daily doses.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

100 mg
75 mg
50 mg



These tablets may be advantageous when initiating therapy with once-daily bedtime dosage in adult outpatients. The usual starting dosage is 75 mg daily. Therapy may be initiated with 50 to 100 mg daily. This may be increased by 25 or 50 mg as necessary in the bedtime dose to a total of 150 mg per day.

These tablets may also be convenient for many hospitalized patients. These patients may need 100 mg a day initially which can be increased gradually to 200 mg a day if necessary. A small number of such patients may need as much as 300 mg a day.

25 mg



This tablet may prove useful when initiating therapy with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime doses.

10 mg



Because lower doses are generally recommended for adolescents and elderly patients, the 10-mg tablets may be most serviceable. Ten mg three times a day with 20 mg at bedtime may be satisfactory in adolescent and elderly patients who do not tolerate higher doses.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

80pp. Paperbound

Single copy, $4.00; 10–49 copies; $3.80 each; 50 or more copies, $3.20 each.

SPECIAL OFFER: One copy of this book (regular price, $4.00) and one copy of **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (regular price, $6.50) for $8.50.

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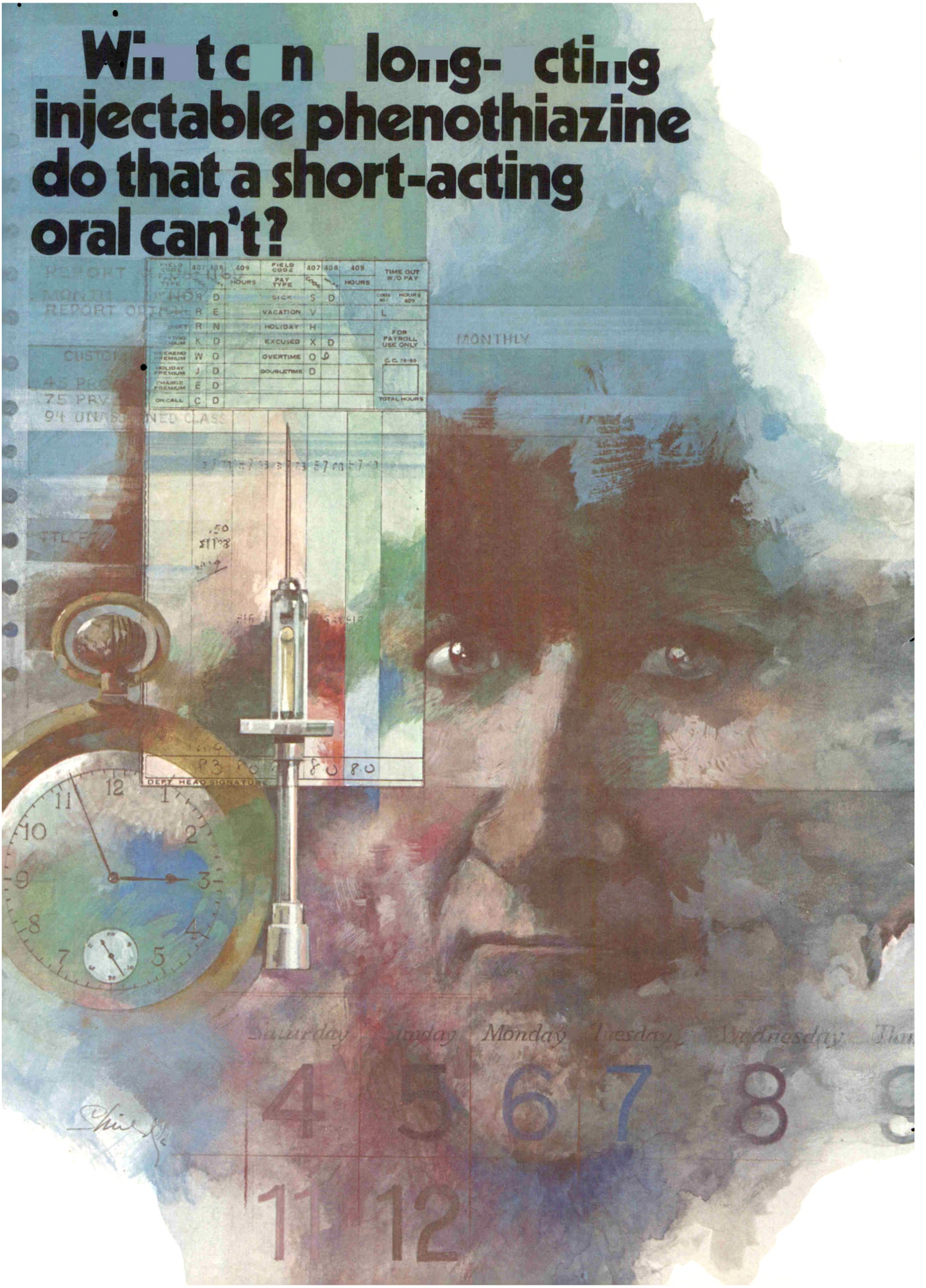
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W.. t.. c.. n .. lo..g- .cti..g injectable phenothiazine do that a short-acting oral can't?



It can save time, it can save money, and it can even save people – by reducing readmissions.

Time Prolixin Decanoate (Fluphenazine Decanoate Injection), with duration of action that may last up to 4 weeks or longer in patients on maintenance therapy, can effect important savings in nursing time.

Approximate Staff Time Required to Medicate 18 Schizophrenic Patients*

8 a.m. ¾ hr. 1 p.m. ¾ hr. 6 p.m. ¾ hr.	1 injection every 28 days for most patients
= 2¼ hours nursing time	4 minutes required for each injection
2¼ hrs. x 28 days = 63 hours of nursing time every 28 days	= 1 hr., 10 minutes nursing time in 28 days
NURSING TIME SAVED IN 28 DAYS	61 hours and 50 minutes or more than 7½ eight-hour working days

*Adapted from Platt R: Br J Social Psychiatry 2: 187-191, 1968.

Money Martin and Townend[1], who found from their study of Prolixin Decanoate that 15 of 39 patients could be maintained on 25 mg. every 4 weeks, also note that on this basis "a year's maintenance on chlorpromazine represents the order of 110 grams of phenothiazine as opposed to 0.33 gram of fluphenazine." Translating this into dollars shows the following potential hospital savings on the basis of patient population:

Agent and amount/year	Form	Cost/patient/year	Cost/500 patients/year
Chlorpromazine (SKF brand) 110 grams	8 oz. concentrate 100 mg./ml.†	$48.75*	$24,375
Prolixin Decanoate .33 grams	5 ml. vials 25 mg./ml.	23.17**	11,585
SAVINGS		$25.58 patient, year	$12,790 500 pts/yr.

* Calculated from prices published in 1975 Red Book.
** Calculated from Squibb 1975 price catalogue.
† With tablets, the annual cost is even greater.

People The controlled drug delivery system of Prolixin Decanoate helps get schizophrenic patients out of the hospital and helps them stay out. It promotes continuity of therapy—reducing outpatient drug defaulting from approximately 50% with oral medications according to one report[2] to about 16% according to another report[3].

It reduces the number and length of rehospitalizations. In one study of 103 patients maintained on injections of long-acting fluphenazines, total hospital readmissions for a year were cut from 191 to 50 and inpatient time from 8,713 days to 1,335 days[4].

It facilitates return to a productive life. A 12-month followup of 103 discharged patients revealed 77% in full-time employment or household duties and only 23% unemployed[4].

PROLIXIN DECANOATE®

(Fluphenazine Decanoate Injection)

may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

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References:

1. Martin ICA and Townend RA: Brit J Psychiat 124: 173-6, 1974.
2. Goldberg HL, DiMascio A, Chaudhary B: Psychosomatics 11: 173-177, 1970.
3. Medical World News, February 11, 1972, p. 58H.
4. Denham J and Adamson L: Can Psychiat Assoc J 18: 235-7, 1973.

For product brief summary see following page.

H425-001R

PROLIXIN DECANOATE®

(Fluphenazine Decanoate Injection)

may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia, liver damage or renal insufficiency, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

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The purpose of this volume is to examine the current status of the borderline concept as a diagnostic term or entity in psychiatry. The chapters reflect the use that various practitioners, approaching their clinical cases from differing perspectives and working in different settings, make of the borderline concept. Of particular interest are articles on the effects of drugs in borderline cases, and the problem of supervision of residents therapeutically involved with them.

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by Robert B. White, M.D. and Robert M. Gilliland, M.D.

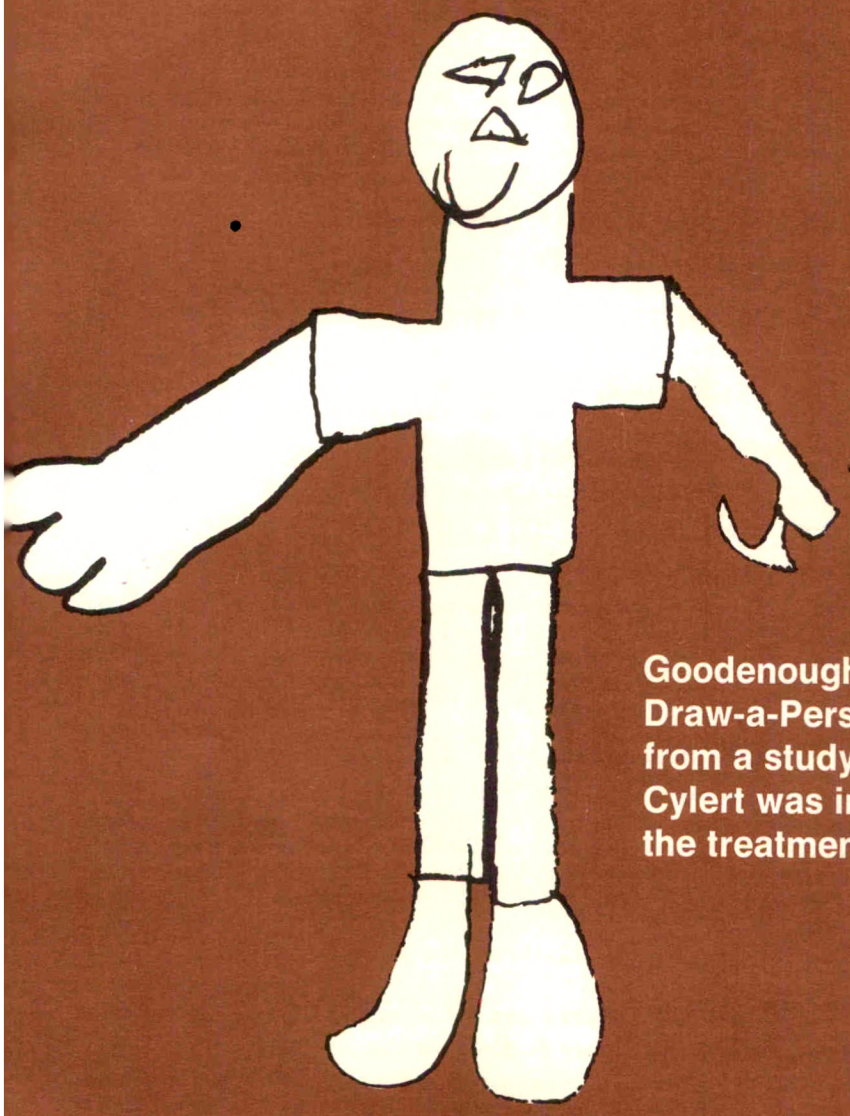
With emphasis on the mechanisms of defense, this presentation offers the student the means to gain the fundamental understanding of psychiatric disorders needed to develop skills for rational therapeutic intervention. All of the basic information needed to fully understand the mechanisms of defense is brought together in a most readable form. The book goes far beyond the early simplistic descriptions of defense mechanisms, putting them in the full context of current theories of anxiety, instinctual drives, ego and superego, and developmental principles including the epigenetic, maturational, and experiential.

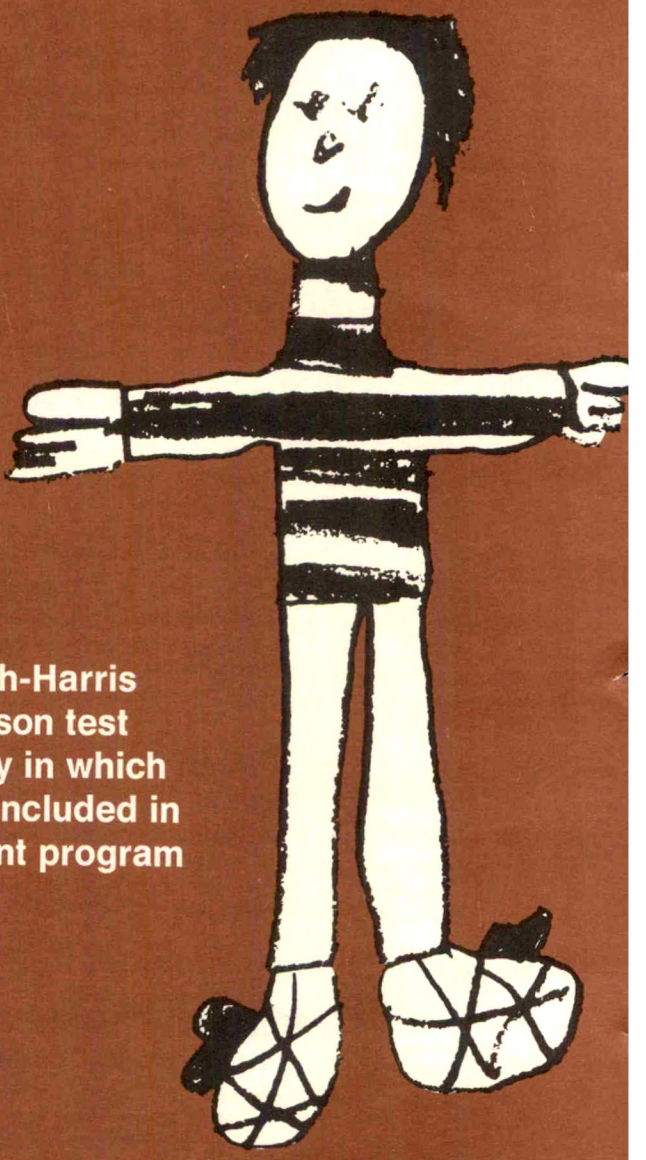
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Goodenough-Harris Draw-a-Person test from a study in which Cylert was included in the treatment program

Drawing made prior to treatment

Drawing made at week 8 during treatment

Cylert (pemoline) will not in itself "enhance learning" or resolve difficult behavioral problems. But it can increase attention span in the hyperkinetic child and reduce the impulsivity that often interferes with the learning process.

Cylert®
(pemoline)

offers these benefits in a treatment program for MBD

- **Single daily dose administration**
- **Minimal cardiovascular effects**
- **Mean dosage in long-term studies remained remarkably constant**

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EFFICACY

Multi-clinic study[1,2]
21 investigators from 10 states and two provinces in Canada took part in the clinical studies.

Double-blind, placebo control
413 patients were randomly assigned to Cylert or placebo groups. 238 patients met all criteria for evaluation of efficacy.

Psychological test results
Children on Cylert had significantly higher scores statistically than those on placebo on these psychological tests:

- The Wechsler Intelligence Scale for Children (WISC) and its performance IQ Sub-Component
- The Wide Range Achievement Test (WRAT) (reading and arithmetic)
- The Lincoln-Oseretsky Motor Performance Test Factor II

Overall results
Approximately two out of three patients were significantly improved by treatment with Cylert as reflected by global ratings.

1. Conners, C. K., ed., *Clinical Use of Stimulant Drugs in Children*, Excerpta Medica, 1974, p. 98.
2. Page, J. G., et al., J. Learning Disabilities, 7:498, Oct., 1974.

SAFETY

Multi-clinic study (9 weeks); safety data analyzed on 407 patients
There was no significant difference between Cylert and placebo groups in:

- Blood pressure
- Laboratory tests
- Pulse
- Neurological status

Insomnia and anorexia were the most frequently seen side effects and often improved with continuation of treatment or reduction of dosage.

Mean weight loss of 1.1 lbs. was demonstrated in the Cylert group during early weeks of treatment; long-term studies have shown that by 3-6 months, most children return to the normal rate of weight gain for their age group.

Long-term study on Cylert; up to 3 years and continuing

Mean dosage . . . remained remarkably constant.
Blood pressure . . no significant changes attributed to Cylert.
Pulse rate . . . . . no significant changes attributed to Cylert.

Laboratory examination—mild to moderate increase in transaminase (SGOT and SGPT) levels in 1-2% of patients (no clinical symptoms); levels returned to normal on withdrawal of medication.

No clinically significant abnormalities in the other tests.

Please see last page of this advertisement for Prescribing Information.

Importance of single daily dosage to the child, the parents and the teacher

For the child		For the adults
No drug in child's possession while at school	⟷	Control of medication remains with parents
Avoids situation in which child is repeatedly singled out as being "different"	⟷	Obviates need for nurse or teacher to supervise taking of mid-day doses
Helps prevent possible variations in effect caused by missed, forgotten or delayed doses	⟷	Helps assure that the prescribed dosage is being given each day



Cylert (pemoline), alone among CNS stimulants used to treat MBD, is inherently long-acting, permitting once-daily dosage.

Cylert can be taken with meals

You can prescribe Cylert a.c., p.c., or with meals. Although the speed of absorption is slightly slowed by food, the total absorption is not affected.

Dosage and administration

Cylert is given as a single oral dose each morning.

The recommended starting dose is 37.5 mg. per day. This daily dosage should be gradually increased at one-week intervals using increments of 18.75 mg. until the desired clinical response is obtained.

The mean daily effective dose ranges from 56.25 to 75 mg. per day. The maximum recommended daily dose of Cylert is 112.5 mg.

Using the recommended schedule of dose titration, significant benefits may not be seen until the third or fourth week of drug therapy. Side effects may be seen prior to optimum clinical results.

When not to use Cylert

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school-age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

The three dosage strengths of Cylert (pemoline)



Cylert, 18.75 mg. (yellow-colored, grooved)

Cylert, 37.5 mg. (orange-colored, grooved)

Cylert, 75 mg. (tan-colored, grooved)

Tablets are actual size.

Cylert
(pemoline) CIV



Prescribing Information

Description: Cylert (pemoline) is a white, tasteless, odorless powder which is relatively insoluble (less than 1 mg/ml) in water, chloroform, ether, acetone, and benzene. In 95% ethyl alcohol, the solubility of pemoline is 2.2 mg/ml.

Actions: Cylert (pemoline) is a central nervous system stimulant. The pharmacologic activity of pemoline is similar to that of other known stimulants but with minimal sympathomimetic effects. Pemoline is structurally dissimilar from the amphetamines and methylphenidate. Although the exact mode of pharmacodynamic action is undetermined in man, pemoline has been reported to increase the rate of synthesis of dopamine in rat brain.

In human subjects, Cylert produces peak blood levels within 2-4 hours. The serum half-life is approximately 12 hours. Multiple dose studies in adults at several dose levels indicate that serum levels plateau in approximately three days. Cylert and its metabolites are primarily excreted by the kidneys with approximately 75% of an oral dose appearing in the urine within a 24-hour period. Approximately 43% of pemoline is excreted unchanged. Metabolites include pemoline dione, conjugated pemoline and mandelic acid.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: MINIMAL BRAIN DYSFUNCTION IN CHILDREN—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: Specific etiology of minimal brain dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis includes the use not only of medical but of psychological, educational, and social resources.

Characteristics commonly reported include: A chronic history of moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindication: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See PRECAUTIONS)

Warnings: Cylert is not recommended for children under six years of age since safety and efficacy in this age group have not yet been established.

Since Cylert (pemoline) and its metabolites are excreted primarily by the kidneys, caution should be observed in administering the drug to children with significantly impaired renal function.

Sufficient data on safety and efficacy of Cylert administration for periods beyond two years duration in children with minimal brain dysfunction are not yet available. Although a definite causal relationship has not been established, some temporary suppression of predicted growth pattern(i.e., weight and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.

Drug Interactions: Interactions between Cylert and other drugs have not been studied in humans. As with most other drugs, concurrent administration with other agents, especially drugs with central nervous system activity, should be carefully monitored.

Usage in Pregnancy: Safety for use in pregnancy has not been established. Standard studies of fertility, teratology and reproduction were conducted in rats and rabbits. Daily oral doses of pemoline of 18.75 and 37.5 mg/kg beginning at conception produced no abnormalities in the fetuses and did not affect viability at birth. Further studies using similar dose levels with drug administration beginning 14 days before conception demonstrated an increased incidence of stillbirths in these animals.

Drug Dependence: Studies of the drug abuse potential of Cylert (pemoline) in primates have not demonstrated a potential for self-administration. However, the pharmacologic similarities between Cylert and other CNS stimulants with known abuse liability suggest that drug dependence of the stimulant type might occur. There have been isolated reports of transient psychotic symptoms in adults following long-term misuse of pemoline taken orally in excessive quantities. Therefore, caution should be observed in emotionally unstable patients considered to have a psychological potential for drug dependence.

Precautions: Delayed hypersensitivity reactions involving the liver have been reported in 1-2% of the patients receiving Cylert usually after several months of therapy. No clinical symptomatology has been observed, but mild to moderate increases in transaminase (SGOT and SGPT) levels have occurred in these cases. These effects appear to be completely reversible when drug treatment is discontinued. Transaminase levels should be determined periodically during therapy with Cylert to detect any such reactions.

Adverse Reactions: The most frequently reported adverse reaction with Cylert is insomnia. Insomnia has been observed prior to optimum therapeutic response and in the majority of cases was transient in nature or responded to dosage reduction. Anorexia with weight loss during the first few weeks of therapy has also been reported. With continuing therapy, a return to a normal weight curve usually occurred within three to six months. Other adverse reactions reported include stomachache, skin rash, irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations. Mild adverse reactions appearing early in treatment often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage reduction or discontinuation should be considered.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning. The recommended starting dose is 37.5 mg per day. This daily dosage should be gradually increased at one week intervals using increments of 18.75 mg until the desired clinical response is obtained. The mean daily effective dose ranges from 56.25 to 75 mg per day. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally to determine if behavioral symptoms sufficient to require continuing therapy recur.

Overdosage: Cylert overdosage has been reported to produce symptoms of tachycardia, hallucinations, agitation, or restlessness. The treatment of acute massive overdosage with pemoline is essentially the same as that for overdosage with any drug having CNS stimulatory effects. Management is largely symptomatic and may include induction of emesis, gastric lavage or other measures as appropriate.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (**NDC** 0074-6025-13)

37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)

75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13) 6013128R

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Effectiveness across the spectrum of most common forms of insomnia

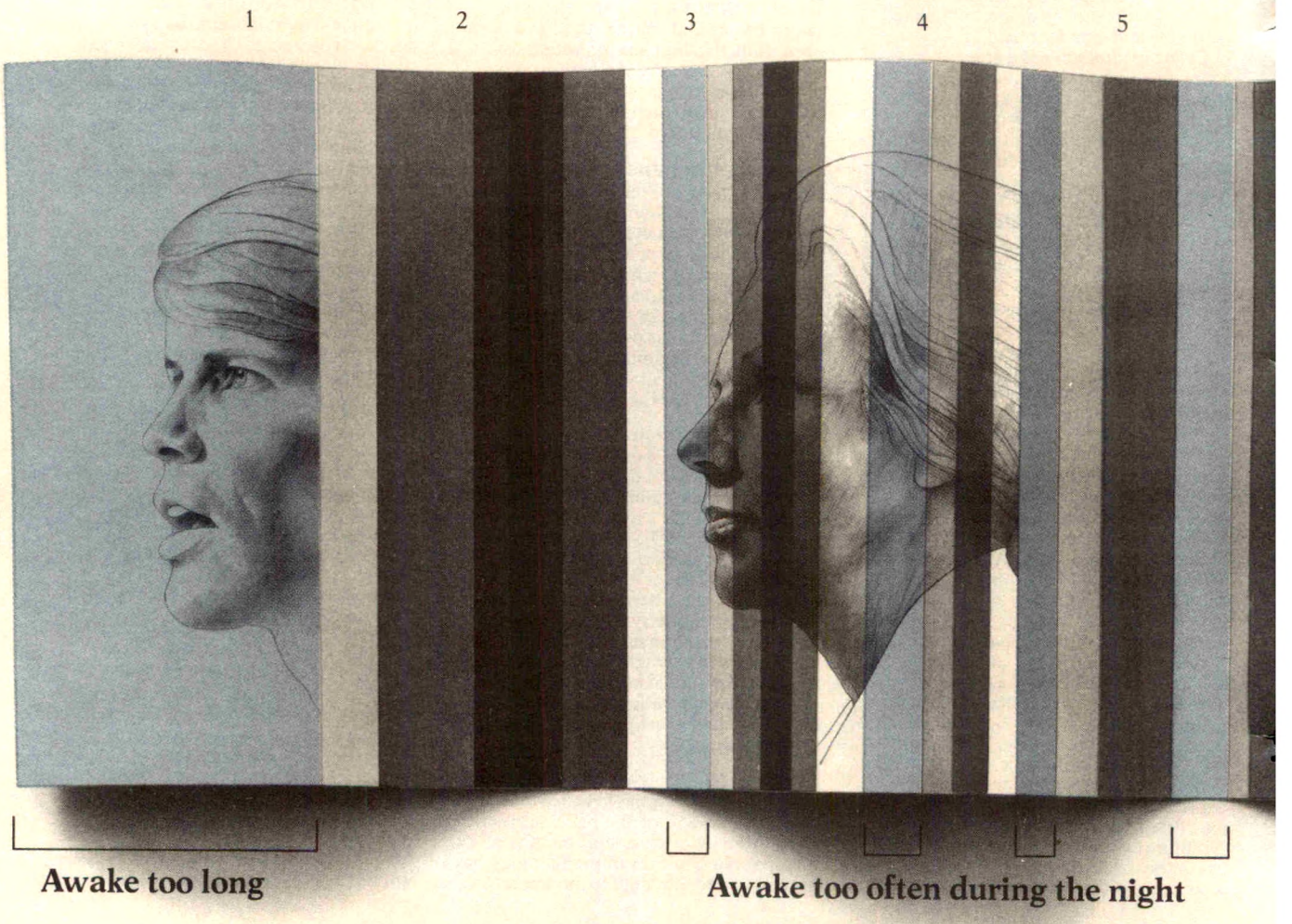
Awake too long, awake too often, awake too early...

These are the most common forms of insomnia, and may occur singly or in any combination. The night of troubled sleep depicted here comprises all three types. As the night progresses from left to right, each sleep stage is identifiable by its own shade of gray. Blue represents "Awake."

As you can see, this hypothetical "patient" takes well over an hour to fall asleep, awakens several times during the middle of the night and awakens too early in the morning.

Sleep Stages

- Awake
- REM
- Stage 1
- Stage 2
- Stage 3
- Stage 4



The insomnias most often occurring in young and older adults

For patients with trouble falling asleep (common in young adult insomnia patients), Dalmane (flurazepam HCl) 30 mg provides sleep within 17 minutes, on average. For those with trouble staying asleep or sleeping long enough (common in those over 50), Dalmane offers increased total sleep time with fewer nocturnal awakenings. These clinical results were demonstrated in studies conducted in four geographically separated sleep research laboratories.[1-4]

The relative safety of Dalmane (flurazepam HCl) is well documented

Dalmane (flurazepam HCl) is relatively safe and well tolerated; morning "hang-over" has been infrequent. The usual adult dosage is 30 mg; in elderly or debilitated patients, limit initial dosage to 15 mg to preclude over-sedation, dizziness or ataxia. Caution patients about possible combined effects with alcohol and other CNS depressants.

Broad-spectrum medication for the most common forms of insomnia

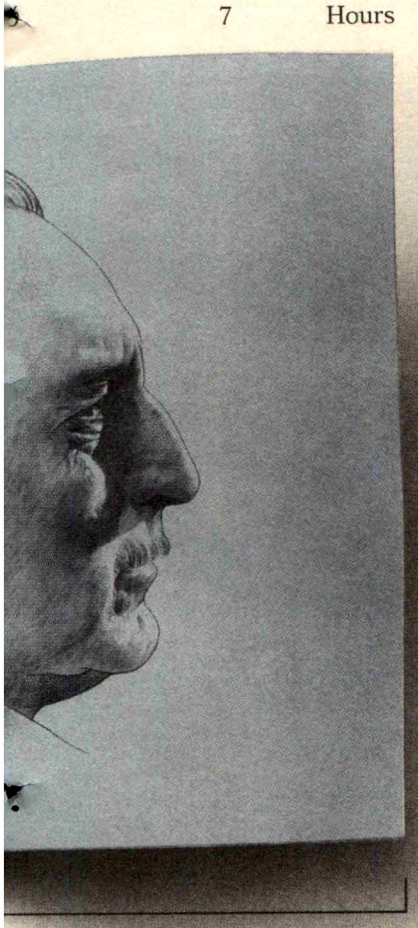


Dalmane®
(flurazepam HCl)

One 30-mg capsule *h.s.*— usual adult dosage
(15 mg may suffice in some patients).
One 15-mg capsule *h.s.*— initial dosage for elderly or debilitated patients.

- □ induces sleep rapidly
- □ reduces nighttime awakenings
- □ lengthens total sleep time

ROCHE

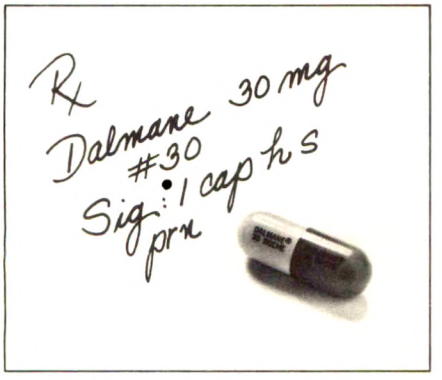


Awake too early

Please see following page for a summary of complete product information.

Broad-spectrum medication for the most common forms of insomnia

Dalmane®
(flurazepam HCl) Ⓒ IV



Objectively proved in the sleep research laboratory, Dalmane

- induces sleep within 17 minutes, on average
- reduces nighttime awakenings
- provides 7 to 8 hours sleep, on average, without repeating dosage

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; and in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Use in women who are or may become pregnant only when potential benefits have been weighed against possible hazards. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, initial dosage should be limited to 15 mg to preclude oversedation, dizziness and/or ataxia. If combined with other drugs having hypnotic or CNS-depressant effects, consider potential additive effects. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported were headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase. Paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, have also been reported in rare instances.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

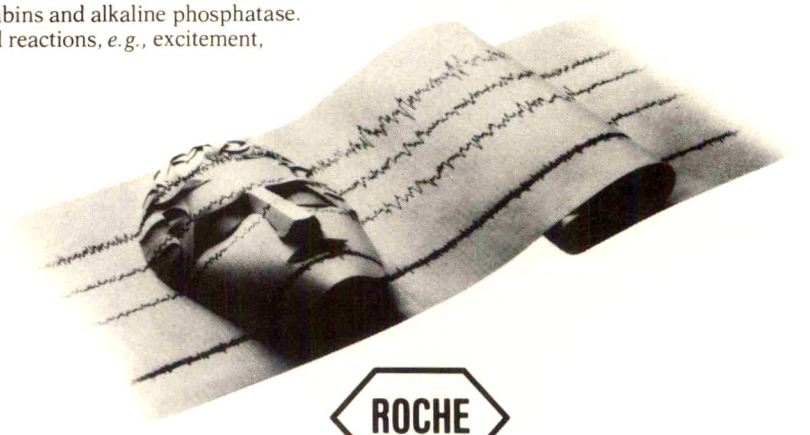
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4. Dement WC: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ



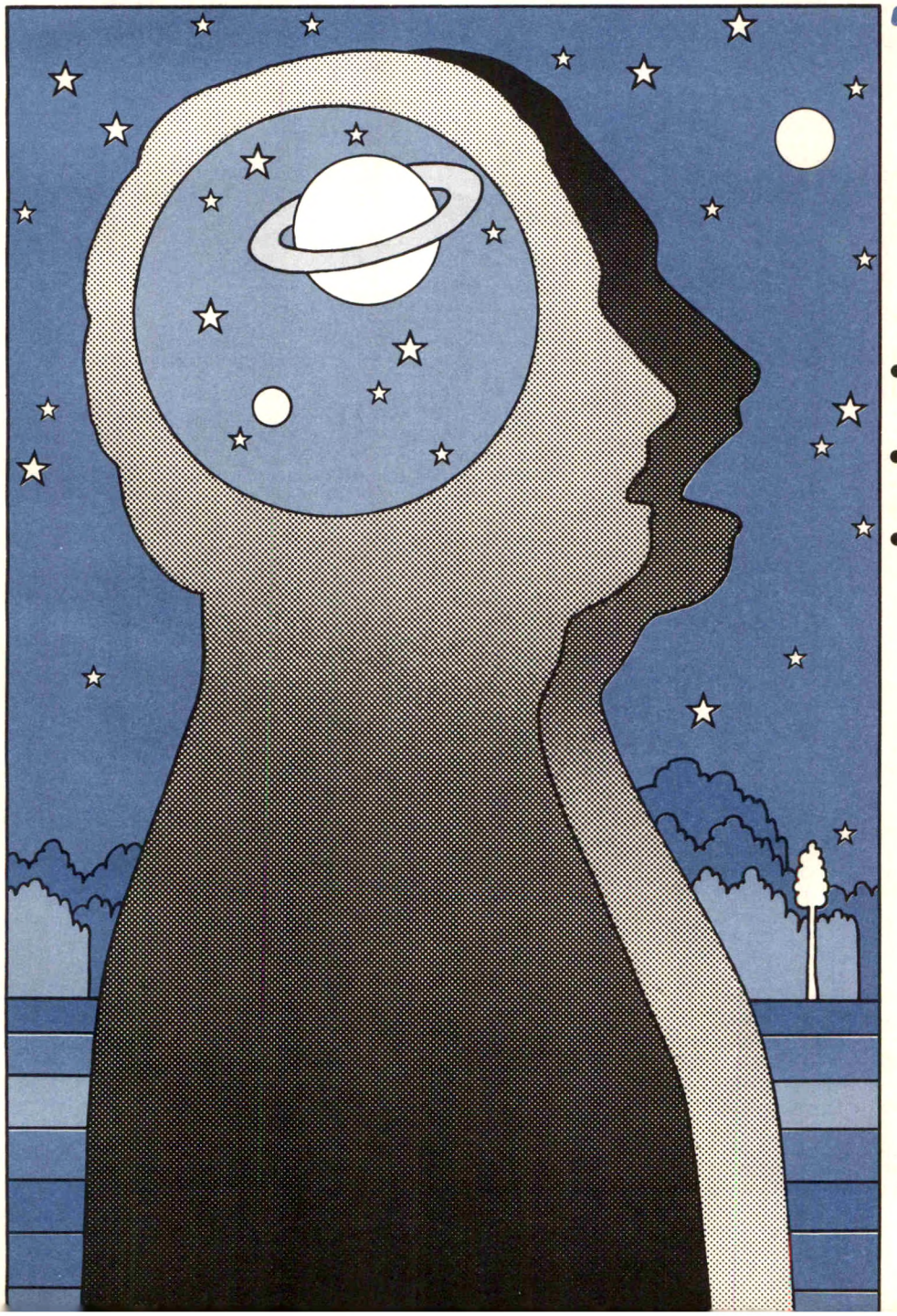
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SCHIZOPHRENIA

He believes he receives messages from outer space.



Trilafon®
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perphenazine, NF

a low-dose high-potency phenothiazine for the management of disturbed behavior in schizophrenic and manic patients

- useful for in-patient and out-patient treatment
- can help orient psychiatric patients to their surroundings
- features minimal sedative effect, usually producing little lethargy or drowsiness

Versatile dosage

Injection
5 mg/ml, 1 ml ampule

Concentrate
16 mg/5 ml,
120 ml bottle

Tablets
2,4,8,16 mg

REPETABS®
Tablets 8 mg
brand of repeat-action tablets

Please see prescribing information on next page.

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Trilafon®
brand of
perphenazine, NF

for the management of schizophrenic and manic disorders

- in the psychiatric hospital
- in the mental health clinic
- in out-patient treatment

INJECTION
when you want *emergency* control for *uncooperative* in-hospital patients

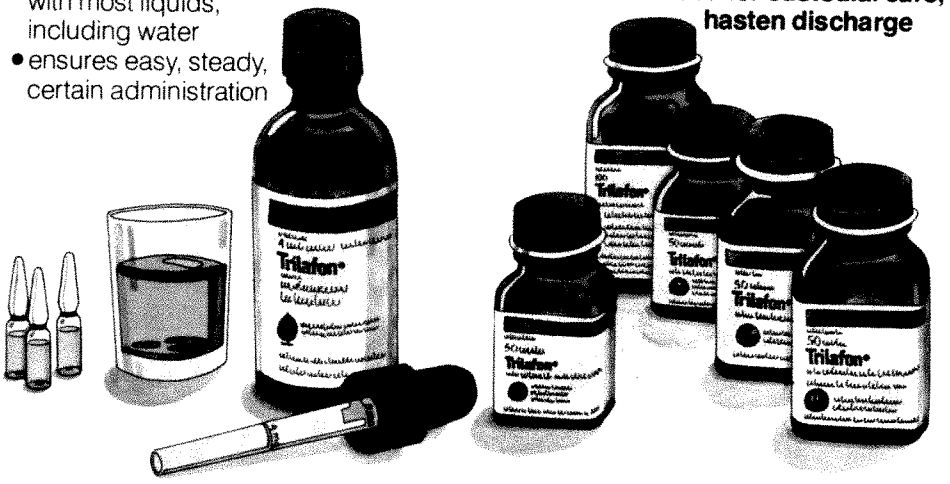
benefits
- usually takes effect in 10 minutes
- average duration of effect is 6 hours

CONCENTRATE
when you want *maintenance* control for *uncooperative* in-hospital patients

benefits
- tasteless, colorless, easily mixes with most liquids, including water
- ensures easy, steady, certain administration

TABLETS
when you want *maintenance* therapy for *cooperative* patients, in the hospital or at home

benefits
- **generally improve cooperation and communication**
- **decrease need for custodial care, hasten discharge**



Trilafon®
brand of perphenazine, NF
Tablets
REPETABS® Tablets
Concentrate
Injection

CONTRAINDICATIONS TRILAFON is contraindicated in drug-associated central nervous system depression (barbiturates, alcohol, narcotics, analgesics, antihistamines). Perphenazine is contraindicated in the presence of existing blood dyscrasias, bone marrow depression and pre-existing liver damage, and in patients who are hypersensitive to perphenazine.

TRILAFON **Injection** should not be given to patients in coma or severely depressed states.

WARNINGS **Usage in pregnancy:** Perphenazine should only be given to pregnant patients when, in the judgment of the physician, the potential benefits outweigh the possible risks.

Perphenazine can lower the convulsive threshold in susceptible individuals; it should be used with caution in patients with convulsive disorders. If the patient is being treated with an anticonvulsant agent, increased dosage of that agent may be required when perphenazine is used concomitantly.

Perphenazine should be used with caution in patients with psychic depression.

Perphenazine is not recommended for children under 12 years of age.

Perphenazine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery.

PRECAUTIONS As with any potent medication, patients receiving perphenazine should be under medical supervision, particularly if they are receiving high doses. Patients who have had any severe reaction to phenothiazines or to imipramine should be treated cautiously, under close medical supervision.

Although the following adverse reactions have not been reported in patients treated with perphenazine, the possibility that they might occur with TRILAFON should be considered: blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, eosinophilia); liver damage (biliary stasis); narrowing of the visual fields; pigmentation of the retina, cornea, or lens; cerebral edema; polyphagia; photophobia; hyperpyrexia.

If hypotension develops, levarterenol (norepinephrine) can be used, but not epinephrine, because epinephrine's action is blocked and partly reversed by perphenazine. Severe, acute hypotension has occurred with the use of phenothiazines and is of particular concern in patients with mitral insufficiency or pheochromocytoma.

A significant rise in body temperature may indicate an idiosyncratic reaction to perphenazine; treatment with perphenazine should be stopped if this occurs.

The antiemetic effect of perphenazine can obscure signs of toxicity due to overdosage of other drugs, or mask the symptoms of disease (eg, brain tumor or intestinal obstruction).

Contact dermatitis has been reported with a perphenazine solution; therefore, contact of hands or clothing by those handling perphenazine solutions should be avoided.

POTENTIATION Since phenothiazines can potentiate the central-nervous-system-depressant actions of opiates, antihistamines, barbiturates, and alcohol, less than the usual dosage of these agents is required when they are administered concomitantly with TRILAFON. Patients should be cautioned that their response to alcohol may be increased while they are being treated with TRILAFON.

Phenothiazines also potentiate the effects of atropine, heat, and phosphorus insecticides, and should be used with caution in persons exposed to these agents.

ADVERSE REACTIONS *Extrapyramidal reactions:* dystonia including protrusion, discoloration, aching and rounding of the tongue; tonic spasm of the masticatory muscles, tight feeling in the throat, slurred speech, dysphagia, oculogyric crisis, trismus, torticollis, retrocollis, muscle weakness, and aching and numbness of the limbs; akathisia; motor restlessness; dyskinesia, parkinsonism; hyperreflexia; and ataxia. The incidence and severity of these reactions usually increase with increased dosage, but have occurred in some patients receiving low dosage. Reduction in dosage or treatment with an antispasmodic agent will usually control extrapyramidal reactions. In some instances, however, these reactions may persist after discontinuation of treatment with perphenazine.

Persistent tardive dyskinesia: As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities. There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine, vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Allergic reactions: erythema, pruritus, urticaria, eczema, anaphylactoid reactions, and local and generalized edema. In extremely rare instances, individual idiosyncrasy or hypersensitivity to phenothiazines has resulted in cerebral edema, circulatory collapse, and death. Photosensitization, asthma, and exfoliative dermatitis have also occurred in patients treated with phenothiazines.

Autonomic reactions: blurred vision, dry mouth or salivation, nasal congestion, nausea, vomiting, hypertension, tachycardia, hypotension, anorexia, urinary frequency or incontinence, and constipation. Significant autonomic effects have been infrequent in patients receiving less than 24 mg. perphenazine daily.

Other reactions: endocrine disturbances (lactation, gynecomastia, galactorrhea, disturbances in the menstrual cycle), headaches, mild insomnia, altered cerebrospinal fluid proteins, ECG abnormalities, reactivation of psychosis, paradoxical excitement, paranoid-like reactions, catatonia, and systemic lupus erythematosus-like syndrome. Hypnotic effects appear to be minimal, particularly in patients who are permitted to remain active. The following adverse reactions, though rare, have also been reported to be associated with perphenazine treatment: agranulocytosis; jaundice; hyperpigmentation of the skin; grand mal convulsions; failure of ejaculation; hyperglycemia.

Side effects with intramuscular TRILAFON **Injection** have been infrequent and transient. Dizziness or significant hypotension after treatment with TRILAFON **Injection** is a rare occurrence.

NOVEMBER 1972
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CHARLES C THOMAS · PUBLISHER

ARMED ROBBERY: Offenders and Their Victims *by* **John M. Macdonald,** *Univ. of Colorado, Denver. Chapters by* **Lieutenant C. Donald Brannan.** The author of this book has conducted psychiatric evaluations for the courts of over 100 armed robbers, including twenty-five who killed during the robbery. Reasons for committing the crime as well as various methods of committing the crime are covered in detail. The actual words used by the robber to describe the crime, the use of violence and his experiences "on the run" are quoted at length. The victim also tells his own story and contributes to the understanding of the crime and its impact on victims. With an awareness of the admiration and fear of the armed robber which has existed in past centuries, the author gives lengthy consideration to the Robin Hood tradition. *'75, 456 pp., 8 tables, $18.50*

THE CLINICAL APPLICATION OF PROJECTIVE DRAWINGS (4th Ptg.) *edited by* **Emanuel F. Hammer,** *New York Univ., New York. (14 Contributors)* A classic in this field, this book provides fundamentals of projective drawings, and goes on to considerations of differential diagnosis, appraisal of psychodynamics, conflict and defense, psychological resources as treatment potentials, and projective drawing usage in therapy. In addition to Buck's H-T-P Technique and Machover's Draw-A-Person Test, the book also includes the Draw-A-Family Procedure, Harrower's Unpleasant Concept Test, Kinget's Drawing Completion Test, and the Eight Card Redrawing Test which is useful in disclosing the deepest layers of the subject's psychosexual identification. *'75, 688 pp., 360 il., $18.50*

A PRACTICAL HANDBOOK OF PSYCHIATRY *edited by* **Joseph R. Novello,** *Univ. of Michigan Medical School, Ann Arbor. (17 Contributors)* For the first time, information on continuing education for psychiatrists is brought together along with a compendium of training programs, referral hospitals, and professional organizations. Information is summarized in tables and outlined in the tradition of the popular handbooks that are available in medicine and surgery and other specialties. Part I, "A Guide to Clinical Practice" provides a discussion of practical problems, tools and techniques of the psychiatrist and includes the complete, official *Diagnostic and Statistical Manual.* Part II, "A Guide to Continuing Education" includes basic reading lists and journals in psychiatry, training opportunities and information on Board Examinations. Part III provides over 700 useful mailing addresses. *'74, 648 pp., 10 il., 47 tables, $16.75*

BEHAVIOR MODIFICATION WITH CHILDREN: A Clinical Training Manual *by* **Alexander J. Tymchuk,** *Univ. of California, Los Angeles.* This informative and easily read book is intended for professionals and parents who are interested in applying operant learning techniques with behaviorally disturbed, mentally retarded, or emotionally disturbed children. Certain chapters describe the parent and student training programs along with instructions on how to conduct a parent training group. Case studies include children who were aggressive, learning disabled or hyperactive. *'74, 144 pp., 20 il., 30 tables, cloth-$9.75, paper-$6.95*

ADOLESCENT MEDICINE: Principles and Practice *by* **I. Newton Kugelmass,** *Consultant to the Departments of Health and Hospitals, New York.* The movement of adolescent life in health and disease has its own hidden harmony of basic systems – biological, psychological and social. Problems with adolescents must be evaluated scientifically by applying the newer knowledge in experimental medicine and then clinically allowing the professional's own individuality to interact with that of the adolescent to lay bare such weaknesses as latent or manifest disorders in deficient development, delayed maturation and deviant function, body-mind derangement, objective-subjective disorder, inner-outer sense discord and self-nonself damage in youth who are poorly programmed for adult life. *'75, 584 pp., 3 il., 11 tables, $23.75*

UNUSUAL SEXUAL BEHAVIOR: The Standard Deviations *by* **David Lester,** *Richard Stockton State College, Pomona, New Jersey.* The primary concern in this book is with research literature and not clinical studies. However, occasional reference is made to the conclusions of clinical studies and to psychoanalytic hypotheses about sexual deviations. The author proposes a classification of sexual deviations which includes three kinds of deviant behavior: variation of mode which refers to deviations in the manner of obtaining sexual satisfaction, variation of object which refers to deviations in the choice of stimulus which provides sexual satisfaction, and variation in strength of the sexual response which refers to deviations in the frequency and intensity of sexual behavior. *'75, 256 pp., $16.75*

LESBIANISM: A Study of Female Homosexuality *by* **David H. Rosen,** *Univ. of California, San Francisco. Foreword by* **Doctor Evelyn Hooker.** This text takes lesbianism out of the subjective mythical and psychoanalytical theory stages and brings it into an objective phenomenological and existential realm. Judd Marmor states: "I found it of great interest as part of the growing list of recent efforts to study homosexuals as human beings without prejudiced preconceptions concerning 'mental illness' or 'mental abnormality.' . . . Doctor Rosen's study of twenty-six lesbians is a valuable addition to the literature in this area. Although his volume is a slender one it contains a great deal of useful and important information." *'74, 140 pp., 28 il., 3 tables, cloth-$7.95, paper-$4.95*

CHILDHOOD DEPRIVATION *compiled and edited by* **Albert R. Roberts,** *Coppin State College, Baltimore, Maryland. (13 Contributors)* The authors of this book have placed the focus on etiology, treatment methods and prevention with case histories often illustrating specific behavior disorders. Childhood experiences discussed which are likely to be traumatic for the child include lack of affection, foster home placement, institutionalization, divorce of parents, death of a loved one, and poverty. Other chapters focus on physical abuses and prolonged isolation of children. Detailed illustrations are provided of behavioral problems such as juvenile delinquency, acting out, temper tantrums, speech impediments and depression which can result from deprivation during childhood. *'74, 232 pp., 1 table, cloth-$9.75, paper-$6.95*

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Tested by time and experience in

1962

"...a considerable decrease of hyperactivity...."[1]
Knobel, 1962



Over a decade of controlled studies and clinical experience has shown the effectiveness of Ritalin in reducing the hyperactivity,[1-3] distractibility,[1,4,5] and disorganized behavior[1-8] in the MBD child.

By lessening the effects of motor and attentional disorders, Ritalin can help the MBD child to better focus his attention on meaningful stimuli and thus can often improve cognition and promote learning.[6,9]

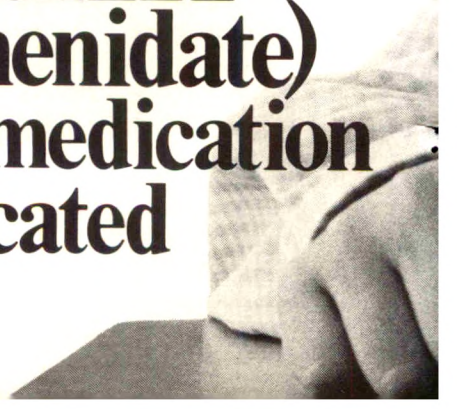
And side effects – insomnia and appetite loss – with Ritalin have occurred less frequently than with dextroamphetamine.[10,11]

Indeed, Ritalin is currently a drug of choice in many MBD situations,[10,12] and can prove to be an important element in many complete remedial programs for MBD.

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Drug treatment is not indicated for all children with MBD.

Dosage should be periodically interrupted. Often, these interruptions reveal some "stabilization" in the child's behavior even without medication, permitting a reduction in dosage and eventual discontinuance of drug therapy.

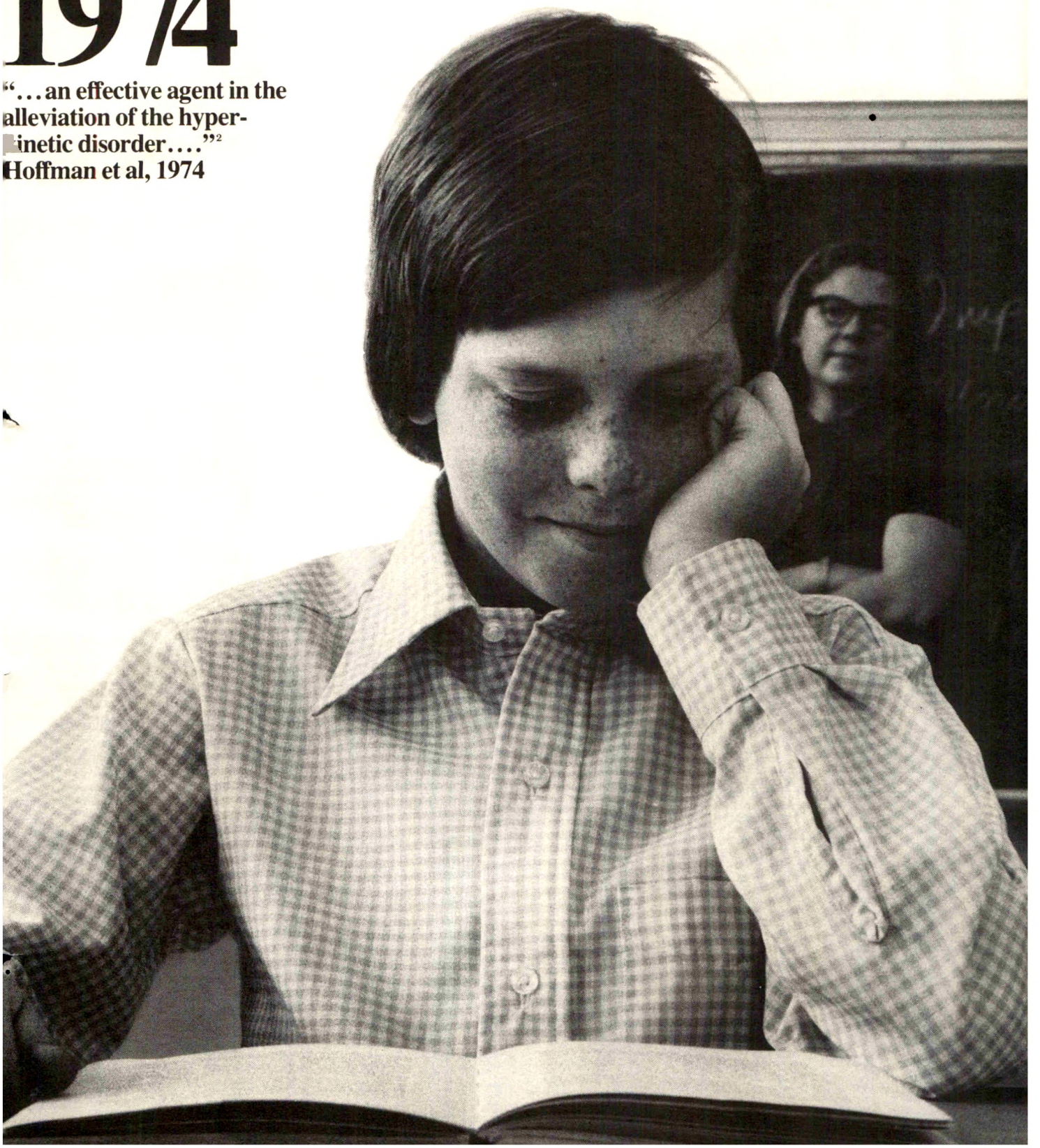
Ritalin®
(methylphenidate)
Only when medication is indicated



the treatment of MBD

1974

"...an effective agent in the alleviation of the hyperkinetic disorder...."[2]
Hoffman et al, 1974





Ritalin®
(methylphenidate)
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Ritalin® hydrochloride ℂ
(methylphenidate hydrochloride)

TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over)

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

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In depression

Daily Dosage Chart



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Ayd* reports that 70 percent of patients default on a q.i.d. regimen; 60 percent on t.i.d.; 30 percent on b.i.d., and only 7 percent on a single daily dose.

A single daily capsule that provides a full day's medication would logically assure better therapeutic results.

Because one capsule lasts from bedtime to bedtime, Tofranil-**PM**® may markedly reduce the probability of missed doses.

After therapy is initiated with the 75-mg. capsule, the 150-mg. capsule is the dose level at which optimum response is usually obtained.

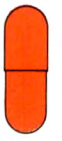
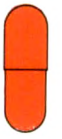
Please review the prescribing information summarized on the back of this page.

*Ayd, F.J., Jr. (Ed.): Int. Drug Ther. Newslett. 7(9/10):35-50 (Nov./Dec.) 1972.

Tofranil-PM®
imipramine

One capsule lasts from bedtime to bedtime

...ose For Maintenance Therapy

...arting ...ose	Usual Optimum Response Dose	A Full Range to Choose From*			
					
75 mg.	150 mg.	150 mg.	125 mg.	100 mg.	75 mg.

*Each capsule contains imipramine pamoate equivalent to 150, 125, 100 or 75 mg. of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg. day. This may be increased to 150 mg. day which is the dose level which usually obtains optimum response.If necessary, dosage may be increased to 200 mg. day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg. day and may be increased to 200 mg. day. Dosage should be increased to 250-300 mg. day if there is no response after two weeks. Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg. day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg. day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
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For complete details, including dosage and administration, please refer to the full prescribing information.

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Konde: Incarnation of *Ndoki*, an evil spirit which causes disease. Wish-projection "causes" illness when a nail is driven into the statue. Also used to "drive out" an illness. Bakongo, Zaire.



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Thorazine®

brand of chlorpromazine

Tablets: 25 mg. of the HCl

- 'Thorazine' controls psychotic symptoms
- Especially useful in agitated, violent or anxious schizophrenic patients
- Unsurpassed clinical experience
- 18 dosage forms and strengths

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudo-parkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only). Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg., in bottles of 50; in Single Unit Packages of 100 (intended for institutional use only). Injection, 25 mg./ml.; Syrup, 10 mg./5 ml.; Suppositories, 25 mg. and 100 mg.; Concentrate (intended for institutional use only), 30 mg./ml. and 100 mg./ml.

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Effective control of psychotic agitation

Suspicious and hostile?



Consider the advantages of starting her on HALDOL® (haloperidol)

Acts promptly to control hostility and suspiciousness

Several clinicians have cited the special value of HALDOL haloperidol in controlling hostility, suspiciousness, ideas of persecution and other delusions associated with psychoses.[1-6] Symptom control is achieved rapidly, with many patients showing distinct improvement in a few days to a week[1,6]—frequently within a few hours when the intramuscular form is used for initial control of acutely agitated psychotic states.[7,8]

Usually leaves patients relatively alert and responsive

Although some instances of drowsiness have been observed, marked sedation with HALDOL haloperidol is rare. In fact, this drug has been reported to increase activity in some underactive patients, while in hyperactive patients it usually reduces activity to a normal level without somnolence. HALDOL haloperidol has been found to "normalize" behavior and produce a sensitivity to the environment that allows more effective use of the social milieu and the therapeutic community.[4]

Reduces likelihood of certain adverse reactions

HALDOL haloperidol, a butyrophenone, avoids or minimizes many of the problems associated with the phenothiazines. The most commonly noted side effects of HALDOL haloperidol—extrapyramidal reactions—are usually dose-related and readily controlled with antiparkinson drugs.

Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. HALDOL haloperidol is also unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes. Ocular damage and pigmentation changes have not been reported.

References: **1.** Towler, M.L., and Wick, P.H.: Int. J. Neuropsychiat. 3: Suppl. 1,62 (Aug.) 1967. **2.** Crane, G.E.: Int. J. Neuropsychiat. 3: Suppl. 1,111 (Aug.) 1967. **3.** Ban, T.A., and Lehmann, H.E.: Int. J. Neuropsychiat. 3: Suppl. 1,79 (Aug.) 1967. **4.** Haward, L.R.C.: Clin. Trials J. 2:135 (May) 1965. **5.** Yun, B.S., et al.: Mich. Med. 67:1349 (Nov.) 1968. **6.** Rubin, R.: Alabama J. Med. Sci. 8:414 (Oct.) 1971. **7.** Reschke, R.W.: Dis. Nerv. Syst. 35:112 (Mar.) 1974. **8.** Palestine, M.L., and Alatorre, E.: Quart. J. Stud. Alcohol 34:185 (Mar.) 1973.

HALDOL®
(haloperidol)
tablets/concentrate/injection

highly specific control of disordered and disruptive behavior

For information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions, please turn page.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

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A Dosage Form for Every Need:



5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.



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Indications: HALDOL haloperidol is indicated for use in the management of manifestations of psychotic disorders.

It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9/74

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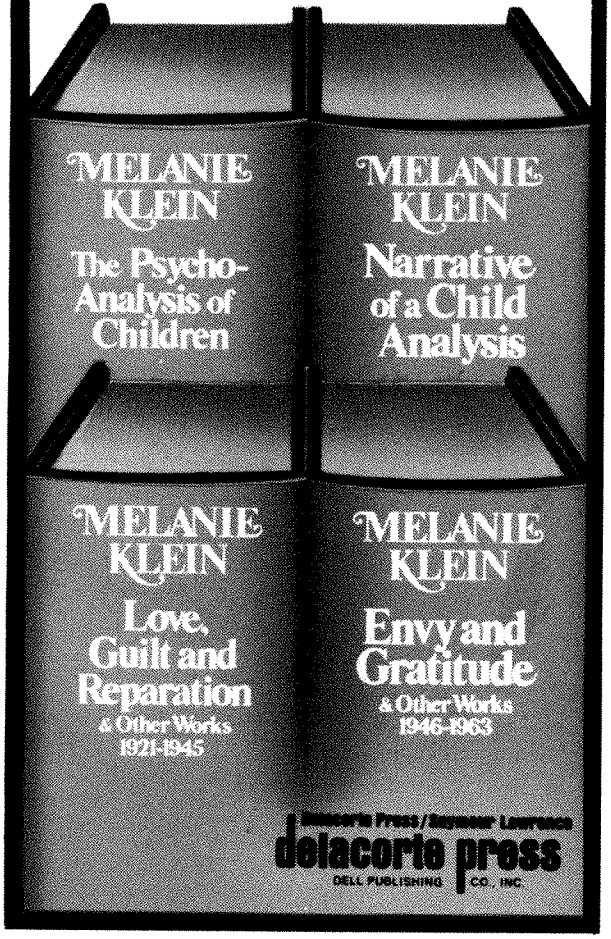
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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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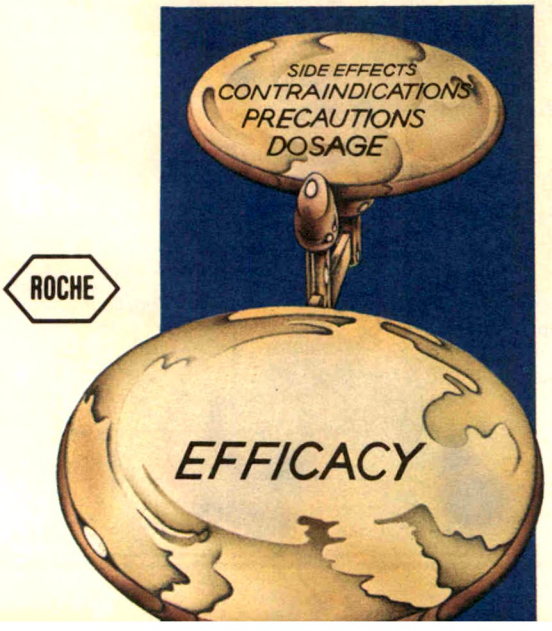
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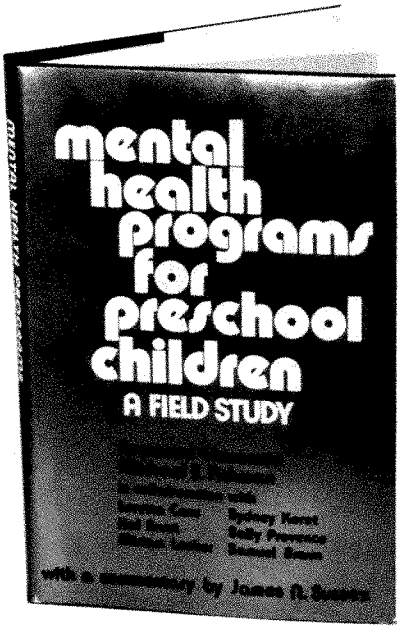
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EVALUATION OF RESPONSE TO LOXITANE BY THE BRIEF PSYCHIATRIC RATING SCALE (BPRS) IN 221 SCHIZOPHRENIC PATIENTS

(98 acute schizophrenic patients in 5 double-blind studies[1] treated for 3 to 6 weeks; 123 chronic schizophrenic patients in 6 double-blind studies[1] treated for 8 to 12 weeks)

SYMPTOM CLUSTERS (BPRS)		PERCENT MAXIMUM POSSIBLE IMPROVEMENT			Pretreatment Score	Posttreatment Score	Statistical significance of change from Base Line, p=
		Acute Schizophrenic Patients	Chronic Schizophrenic Patients				
DEPRESSION Somatic Concern Anxiety Guilt Feelings Depressive Mood		56%	27%	ACUTE CHRONIC	3.07 2.02	1.92 1.74	0.001 0.01
THOUGHT DISORDER Conceptual Disorganization Grandiosity Suspiciousness Hallucinatory Behavior Unusual Thought Content		61%	23%	ACUTE CHRONIC	3.88 3.30	2.11 2.78	0.001 0.05
ANERGIA Emotional Withdrawal Mannerisms and Posturing Motor Retardation Blunted Affect		47%	17%	ACUTE CHRONIC	3.75 3.75	2.46 3.27	0.001 0.05
EXCITEMENT-DISORIENTATION Excitement Disorientation		61%	21%	ACUTE CHRONIC	2.54 2.50	1.60 2.19	0.01 —

1. Data on file, Clinical Research Department, Lederle Laboratories.

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Exhibited certain favorable trends in side effects profile

IN 31 STUDIES INVOLVING 469 ACUTE AND CHRONIC SCHIZOPHRENIC PATIENTS, CERTAIN FAVORABLE TRENDS WERE EXHIBITED IN THE LOXITANE SIDE EFFECTS PROFILE; THESE REQUIRE FURTHER TESTS AND BROADER CLINICAL EXPERIENCE FOR CONFIRMATION.



While cardiac arrest, blood dyscrasias and renal toxicity have been reported with other antipsychotic agents, they have not been seen with LOXITANE. Hepatotoxicity manifested by jaundice or biliary stasis has not been observed. Transient liver enzyme changes, however, have been reported, but it has not been determined whether they are related to LOXITANE administration.

Although a few cases of changes in ECG have been reported, a causal relationship between this reaction and LOXITANE has not been established. Clinical experience with LOXITANE has not demonstrated ocular toxicity; however, the possibility of its occurrence cannot be ruled out at this time. Of the various endocrine abnormalities, only galactorrhea has been reported with LOXITANE and only in rare instances. Manifestations of adverse effects on the central nervous system other than extrapyramidal symptoms have been encountered infrequently, and drowsiness, when it occurs, is usually mild and subsides with continued therapy. Skin rashes of uncertain etiology have been observed in a few patients during the hot summer months; therefore the possibility of phototoxicity and/or photosensitivity cannot be excluded.

In general, LOXITANE presents a profile of extrapyramidal side effects similar to that of other agents used in the treatment of schizophrenia.

Cardiovascular effects such as hypotension, hypertension, lightheadedness and syncope have been reported. Anticholinergic effects seen with LOXITANE include dry mouth, nasal congestion, constipation and blurred vision.

arnings and precautions and for more detailed information concerning side effects.

FOR ORAL USE

PRODUCT INFORMATION

DESCRIPTION

LOXITANE *loxapine*, a dibenzoxazepine compound, represents a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines. Chemically, it is 2 - chloro - 11 - (4 - methyl - 1 - piperazinyl) dibenz [b,f] [1,4] oxazepine. It is present in capsules as the succinate salt. Each 1.36 mg of loxapine succinate is equivalent to 1 mg of loxapine.

ACTIONS

Pharmacologically, loxapine is a tranquilizer for which the exact mode of action has not been established. However, changes in the level of excitability of subcortical inhibitory areas have been observed in several animal species in association with such manifestations of tranquilization as calming effects and suppression of aggressive behavior.

In normal human volunteers, signs of sedation were seen within 20 to 30 minutes after administration, were most pronounced within 1½ to 3 hours, and lasted through 12 hours. Similar timing of primary pharmacologic effects was seen in animals.

Absorption of loxapine following oral or parenteral administration is virtually complete. The drug is removed rapidly from the plasma and distributed in tissues. Animal studies suggest an initial preferential distribution in lungs, brain, spleen, heart, and kidney. Loxapine is metabolized extensively and is excreted mainly in the first 24 hours. Metabolites are excreted in the urine in the form of conjugates and in the feces unconjugated.

INDICATIONS

LOXITANE *loxapine succinate* is indicated for the manifestations of schizophrenia.

CONTRAINDICATIONS

LOXITANE is contraindicated in comatose or severe drug-induced depressed states (alcohol, barbiturates, narcotics, etc.)

LOXITANE is contraindicated in individuals with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Safe use of LOXITANE *loxapine succinate* during pregnancy or lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the benefits of treatment be weighed against the possible risks to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from midpregnancy with doses of 0.6 and 1.8 mg/kg, doses which approximate the usual human dose but which are considerably below the maximum recommended human dose.

Usage in Children: Studies have not been performed in children; therefore this drug is not recommended for use in children below the age of 16.

LOXITANE, like other tranquilizers, may impair mental and/or physical abilities, especially during the first few days of therapy. Therefore, ambulatory patients should be warned about activities requiring alertness (eg, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

PRECAUTIONS

LOXITANE loxapine succinate should be used with extreme caution in patients with a history of convulsive disorders since it lowers the convulsive threshold. Seizures have been reported in epileptic patients receiving LOXITANE at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy.

Loxapine has an antiemetic effect in animals. Since this effect may also occur in man, loxapine may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

LOXITANE should be used with caution in patients with cardiovascular disease. Increased pulse rates have been reported in the majority of patients receiving antipsychotic doses; transient hypotension has been reported. In the presence of severe hypotension requiring vasopressor therapy, the preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine.

The possibility of ocular toxicity from loxapine cannot be excluded at this time. Therefore, careful observation should be made for pigmentary retinopathy and lenticular pigmentation since these have been observed in some patients receiving certain other antipsychotic drugs for prolonged periods.

Because of possible anticholinergic action, the drug should be used cautiously in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

ADVERSE REACTIONS

CNS Effects: Manifestations of adverse effects on the central nervous system, other than extrapyramidal effects, have been seen infrequently. Drowsiness, usually mild, may occur at the beginning of therapy or when dosage is increased.

It usually subsides with continued LOXITANE therapy. The incidence of sedation has been less than that of certain aliphatic phenothiazines and slightly more than the piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported.

Extrapyramidal Reactions—Neuromuscular (extrapyramidal) reactions during the administration of LOXITANE *loxapine succinate* have been reported frequently, often during the first few days of treatment. In most patients, these reactions involved Parkinson-like symptoms such as tremor, rigidity, excessive salivation, and masked facies. Akathisia (motor restlessness) also has been reported relatively frequently. These symptoms are usually not severe and can be controlled by reduction of LOXITANE dosage or by administration of antiparkinson drugs in usual dosage. Dystonic and dyskinetic reactions have occurred less frequently, but may be more severe. Dystonias include spasms of muscles of the neck and face, tongue protrusion, and oculogyric movement. Dyskinetic reaction has been described in the form of choreo-athetoid movements. These reactions sometimes require reduction or temporary withdrawal of LOXITANE dosage in addition to appropriate counteractive drugs.

Persistent Tardive Dyskinesia—As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movement of the tongue, face, mouth, or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia; antiparkinson agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Cardiovascular Effects: Tachycardia, hypotension, hypertension, light-headedness, and syncope have been reported.

A few cases of ECG changes similar to those seen with phenothiazines have been reported. It is not known whether these were related to loxapine administration.

Skin: Dermatitis, edema (puffiness of face), pruritus, and seborrhea have been reported with loxapine. The possibility of photosensitivity and/or phototoxicity occurring has not been excluded; skin rashes of uncertain etiology have been observed in a few patients during hot summer months.

Endocrine Effects: No endocrine abnormalities have been reported.

Anticholinergic Effects: Dry mouth, nasal congestion, constipation, and blurred vision have occurred; these are more likely to occur with concomitant use of antiparkinson agents.

Other Adverse Reactions: Nausea, vomiting, weight gain, weight loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, and polydipsia have been reported in some patients.

DOSAGE AND ADMINISTRATION

LOXITANE *loxapine succinate* is administered orally usually in divided doses two to four times a day. Daily dosage (in terms of base equivalents) should be adjusted to the individual patient's needs as assessed by the severity of symptoms and previous history of response to antipsychotic drugs. Initial dosage of 10 mg twice daily is recommended, although in severely disturbed patients initial dosage up to a total of 50 mg daily may be desirable. Dosage should then be increased fairly rapidly over the first seven to ten days until there is effective control of psychotic symptoms. The usual therapeutic and maintenance range is 60 mg to 100 mg daily. However, as with other antipsychotic drugs, some patients respond to lower dosage and others require higher dosage for optimal benefit. Daily dosage higher than 250 mg is not recommended. For maintenance therapy, dosage should be reduced to the lowest level compatible with symptom control; many patients have been maintained satisfactorily at dosages in the range of 20 mg to 60 mg daily.

HOW SUPPLIED

LOXITANE *loxapine succinate* is supplied in the following base equivalent strengths:

CAPSULES Hard Shell Printed "Lederle"

10 mg—Green and Yellow; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5360.

25 mg—Green Two Tone; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5361.

50 mg—Green and Blue; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5362.

REV. 3/75

For the manifestations of schizophrenia



Loxitane®
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Because the somatic and psychic complaints that accompany depressive neurosis can interfere with the treatment of the underlying psychopathology—rapid relief of these symptoms is often crucial to therapy.

In 14 double-blind studies of four weeks duration, 339 patients with depressive neurosis received Mellaril. In these studies, 55% of the overall improvement was observed by the end of the first week, and a total of 293 patients (86%) improved during the four weeks.*

Mellaril usually does not cause euphoria or undue sedation and is nonaddicting. Patients, therefore, generally remain alert and better able to respond to therapy. (The physician should, however, caution patients against participating in activities which require complete mental alertness, e.g., driving.)

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for the short-term treatment of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling. The following reactions have occurred with phenothiazines and should be considered: *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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THE AMERICAN JOURNAL OF PSYCHIATRY

The Current Status of Behavioral Psychotherapy: Theory and Practice

BY ISAAC M. MARKS, M.D., D.P.M.

Behavioral psychotherapy has become one of the definitive treatments available for the relief of psychiatric suffering. Although hardly a panacea for all ills, the behavioral approach is the treatment of choice for certain selected problems. Behavioral methods make their contribution in the context of general psychiatric management and often must be used in conjunction with other treatment. The theoretical and practical aspects of behavioral psychotherapy have changed a great deal over the past few years and continue to evolve rapidly. Although useful molecular theories are emerging to guide the discipline, no global theory is likely to be satisfactory in the foreseeable future.

THE PRINCIPLES OF behavioral psychotherapy reach back over the centuries. The ideas that govern the behavioral treatment of anxiety were enunciated clearly in the 1650s by John Locke. He recommended that if "your child shrieks and runs away at the sight of a frog, let another catch it, and lay it down at a good distance from him: at first accustom him to look upon it; when he can do that to come nearer to it, and see it leap without emotion; then to touch it lightly, when it is held fast in another's hand; and so on until he can come to handle it as confidently as a butterfly or a sparrow" (1, pp. 481–482). Today this principle is called exposure in vivo, and we could thus say that behavioral theory is Lockean in origin. It could also be said to be Freudian if one goes by Freud's comment in 1919: "One can hardly master a phobia if one waits till the patient lets the analysis influence him to give it up . . . one succeeds only when one can induce them by the influence of the analysis to . . . go [about alone] . . . and to struggle with their anxiety while they make the attempt" (2, pp. 165–166).

Another behavioral principle, that of satiation, is described in Shakespeare's opening lines of *Twelfth Night*: "If music be the food of love, play on; . . . that, surfeiting, the appetite may sicken, and so die." The concept of contingent reward dates back to the first parent who gave his child a candy or a kiss for good behavior.

The fact that the principles of behavioral treatment have been around so widely for so long indicates that they are rooted in common sense, which is as old as the human species. What, then, is new? The answer is that these commonsense principles have been welded together over the past two decades into a potent therapeutic technology that can now relieve several formerly untreatable conditions and holds promise for further advances. This development hinges on combining an experimental and a clinical approach. It enables treatment to be tested and refined so that redundant elements are gradually eliminated while effective ingredients are retained and extended.

This pragmatic approach reminds one of a legendary South African millionaire who was confronted with some awkward facts about his policies at an annual meeting of the board of his company. He faced his inquisitors squarely, thumped his fist on the table, and declared, "Gentlemen, those are my principles, and if you don't like them, I have many more."

Critics might call this theoretical promiscuity or gutless eclecticism. Progress, however, depends on keeping track of the main question facing clinicians: "What methods of treatment produce reliable improvements in problem X, and what principles govern such successful methods?" In short, what works and why? Advances can be rapid if we keep this question in mind and maintain a willingness to submit our ideas to ex-

Revised version of a special lecture presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Marks is Reader in Experimental Psychopathology, Institute of Psychiatry, the Maudsley Hospital, De Crespigny Park, Denmark Hill, London, SE 5, England.

perimental destruction. Inert therapeutic practices can thus be discarded and more potent methods introduced.

It is true that the behavioral field, like any other, has had its fill of dogmas. There has been no shortage of all-explanatory schools, each trimming the richness of clinical experience to its own Procrustean formula in the name of ideological purity. But the clash of rival parties becomes more muted as the area of tested knowledge expands. Behavioral treatment is beginning to take its place as one of several forms of psychotherapy available to the clinician for the relief of psychiatric suffering. Behavioral psychotherapy is not a panacea or an all-embracing theory or method. Like psychopharmacology, it is a therapeutic technology that can aid general clinical management and has its indications and contraindications.

INDICATIONS FOR BEHAVIORAL PSYCHOTHERAPY

There is no danger of a takeover bid by behaviorists for the whole of psychiatric practice. At the present time behavioral psychotherapy can help perhaps 10% of all *adult* psychiatric patients when used as the chief instrument of change. This means that 90% of all adult cases require other approaches as the main form of treatment. Behavioral psychotherapy is probably the *approach of choice* in phobic disorders, including social anxieties, and in obsessive-compulsive rituals. It is also helpful in sexual dysfunctions such as impotence and frigidity and in sexual deviations such as exhibitionism. In these areas behavioral methods have been found to be more useful than contrasting approaches.

In other adult disorders behavioral psychotherapy may be useful, but more controlled data about efficacy are needed. Examples include obsessive thoughts (as opposed to rituals); deficits and problems in social skills; disorders of habit or of impulse control such as stammering, hair pulling, self-mutilation, and compulsive gambling; appetitive disorders including obesity and anorexia; conversion hysteria; and some personality disorders. An operant approach can be helpful for the social rehabilitation of persons with certain chronic disorders such as schizophrenia and personality and organic deficits. In such cases the behavioral approach can be a worthwhile adjuvant to other treatment methods.

In several important adult disorders behavioral psychotherapy is *not of value*. Examples are acute schizophrenia, severe depression, and hypomania. This approach is also not useful in adult neurotic patients for whom clearly definable goals cannot be worked out.

In children behavioral psychotherapy might be the treatment of choice for nocturnal enuresis and phobias. It is also helpful for the educational rehabilitation of children with subnormal intelligence or learning problems. It has also been useful in institutional settings for the rehabilitation of delinquents, although backup facilities are a problem. Anecdotal reports suggest that behavioral therapies are sometimes useful in treating conduct disorders in children.

Until recently much of the evidence for behavioral psychotherapy fell far short of the ideal, i.e., controlled studies of groups of patients with clearly definable characteristics who were treated by either a behavioral or a contrasting method. Data were originally based primarily on uncontrolled case histories or on controlled trials of volunteers (3, 4), who may differ from patients in important ways. However, an increasing number of controlled trials of clinical patients is beginning to support the value of some behavioral methods.

Behavioral approaches are being applied increasingly not only for the relief of psychopathology, but also in educational, social, and other nonclinical settings. At certain points interventions with patients can be said to lie more in the domain of the educationalist and social psychologist than that of the clinician. However, this report is concerned only with behavioral psychotherapy in the field of clinical psychiatry and will not go into wider areas of behavior change.

PRINCIPAL FORMS OF BEHAVIORAL PSYCHOTHERAPY

Behavioral methods assume diverse forms, some of which bear little resemblance to one another. A cynic might argue that those methods which are found to be effective tend to be called behavioral and those which are not are disowned and given some other name. Often the same technique is practiced by adherents of different schools. A case in point comes from Viktor Frankl (5, 6), who attributed his ideas to existentialist logotherapy. In the treatment of an obsessive-compulsive ritualizer he described how he demonstrated to the patient that he could dirty his own hands by touching the floor and refrain from washing thereafter; then he asked the patient to imitate the same action. Behaviorists might call this modeling with exposure in vivo and response prevention. But we need not quibble about theoretical pedigrees. It is more important to describe our methods so that others can test and use them in a similar way.

Behavioral treatments can be administered to patients individually or in groups, depending on what is practical and desirable in a given case. Behavioral treatments can be arbitrarily divided into those which 1) reduce anxiety-linked behavior (e.g., phobias or compulsive rituals), 2) reduce appetitive behaviors (e.g., exhibitionism or obesity), and 3) develop new behavior (e.g., teaching social skills to the socially incompetent).

Anxiety reduction is usually effected by some variant of exposure treatment such as desensitization or flooding; self-regulation and modeling can also be useful. Appetitive behavior can be reduced by self-regulation, satiation, and aversive methods such as covert

sensitization. In covert sensitization, for example, an exhibitionist might be asked to imagine exposing his genitals to a pretty young girl in the street and then to imagine a policeman at his side arresting him. The fantasy of the policeman acts as an aversive stimulus. Equally, the exhibitionist could be asked to wear an elastic band on his wrist wherever he went, and when he felt an urge to expose himself, simply to snap the elastic band sharply against his wrist, repeatedly if necessary, until the temptation disappeared. This can be construed as a form of self-regulation as well as aversion. It could also be called thought-stopping. One patient I saw had morbidly jealous thoughts about his wife. After a few trials with the elastic band, he found it sufficient simply to stare at the band on his wrist and imagine the sensation of a sting on his wrist without actually snapping the band. Anticipation of the sting dispelled the jealous thoughts. He remained well at 6 months' follow-up after only 3 sessions of treatment.

These examples illustrate how the same technique can be described and used in several different ways. The elastic band can be an aversive stimulus, a thought-stopper, a self-regulator, or a response-disruptor.

A variety of methods is employed for the development of new behavior, including social skills training, educational programs, modeling, shaping, prompting, pacing, fading, self-regulation, contracting, and contingent reward. In social skills groups there is a great deal of overlap with techniques also found in psychodrama and encounter groups, e.g., warm-up games, role rehearsal, role reversal, and doubling. The same can occur in groups for family and marital therapy. These approaches are strictly behavioral only to the extent that they are directive and involve clearly specified goals of treatment and measurements of change.

GENERAL ISSUES

Certain general principles run across the arbitrary subdivision of behavioral methods. *Clear delineation of treatment goals* is essential. Often these are called target problems. If the behaviors to be changed by the end of successful treatment cannot be identified, then the patient is untreatable by behavioral means. Clear targets need to be set toward which treatment is directed. "Self-actualization" would be too vague to be a suitable target; so would "increased self-confidence." Examples of clear targets are "being able to ask a girl for a date without trembling" and "being able to shop in a crowded supermarket." Several targets are commonly needed for each patient. Targets must be specific and useful for a patient in terms of his life handicap.

Cooperation of the Patient

Behavioral treatment can only be given to adult neurotics if they are cooperative; successful treatment cannot be forced on patients against their will. Treatment often involves the patient in detailed, complex programs that require his daily cooperation for completion.

Family Involvement

Significant family members must participate in therapy when the patient's psychopathology is hindering family functioning. Treatment often needs to be continued away from the psychiatrist's office in the natural setting of the problem, which frequently includes the patient's home. Patients with social phobias may need to accompany a behavioral psychotherapist into a crowded restaurant and have a meal with him several times in order to overcome their anxiety. A compulsive handwasher may need to visit her home with her therapist and jointly contaminate the home with "dirty" material and refrain from washing thereafter. These unconventional roles may require some relearning experiences by therapists.

Backup Facilities in the Community

Backup facilities in the community are often required. Patients may need to be discharged to a residential treatment facility or other accommodation where treatment and supervision can gradually be "faded out." When the patient lives far from the treatment center, local personnel may be essential to provide necessary follow-up treatment after discharge.

Homework by the Patient Between Sessions

Another important issue concerns homework assignments and practice in self-regulation. Patients may be given set tasks to perform between sessions, and, indeed, the sessions may be contingent upon patients completing their homework first; e.g., a person with a social phobia may need to take his girlfriend out to a restaurant three times between two given sessions. Self-regulation may take the form of keeping a diary, monitoring and rating one's anxiety in phobic situations, practicing deep-breathing exercises during them, and learning to remain in the same spot until the anxiety has abated, which might take an hour or more.

It is usual practice to combine more than one therapeutic principle in the treatment of a given patient, as the occasion demands. The following example illustrates this.

Case 1. Jill was an 18-year-old girl with borderline intelligence and mild cerebral palsy. She had complicating chronic agoraphobia, compulsive handwashing, and frequent temper tantrums at home. The nurse-therapist treated Jill by first escorting her on walks outside the outpatient department for increasingly long distances, fading himself out of the situation, and persuading Jill to do the same exercises herself both during the session and at home between sessions. Between sessions Jill was asked to undertake increasingly long bus trips and to enter her achievements in a diary. The agoraphobia rapidly improved.

At the beginning the nurse-therapist had told Jill and her mother that he would leave the compulsive rituals for treatment at a later date when the agoraphobia had improved. The mother inquired about the nature of that treatment, and

the nurse-therapist explained the principles of exposure in vivo. By the time the agoraphobia had improved, the mother reported that she had spontaneously carried out the exposure in vivo treatment of her daughter's compulsions. She contaminated the house with dreaded material and asked Jill to do the same and refrain from washing thereafter. The patient's ritualistic behavior ceased when the mother acted as a cotherapist without the therapist's assistance.

The temper tantrums were dealt with by family contracting. The therapist jointly interviewed Jill, her mother, and her father and arranged an exchange of contracts whereby the parents agreed to lavishly praise their daughter for such behavior as helping at mealtimes to set and clear the table and wash the dishes. Jill promised to carry out such activities and not to engage in temper tantrums. Soon the temper tantrums ceased and the parents rewarded their daughter more often. At one-year follow-up Jill's improvement in her phobias, rituals, and temper tantrums continued and she was attending a special training course to develop work skills.

AN OPERATIONAL FRAMEWORK FOR THERAPEUTIC INFLUENCES

The many influences that bear on treatment can be viewed within a dual-process operational framework (see figure 1). First, a patient needs to be *motivated* to seek and complete treatment. Many influences will affect this process, including the patient's own commitment to change, social pressure on him to improve, that vague element called suggestion, and the credibility of the therapist. Of several hundred patients in my unit who were offered behavioral treatment, 23% refused it. This refusal rate is similar to that with many other treatments. In such cases treatment fails at the motivational stage because the patient fails to swallow his psychological pill, so to speak. Most behavioral treatments are, in fact, readily acceptable to patients.

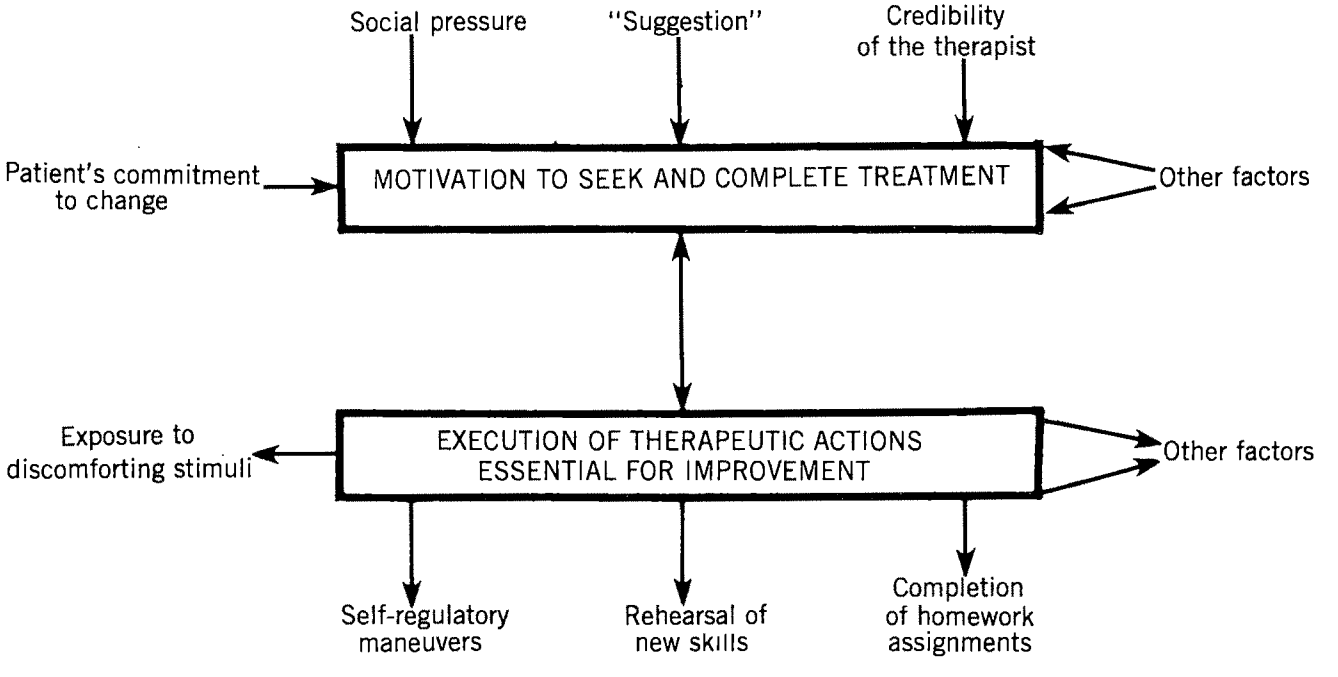
The second process involves the patient's *execution* of therapeutic actions essential for improvement, e.g., a few hours' exposure to a phobic stimulus or a complicated set of interactions with other people in a social training program. When execution of these actions is satisfactory, the failure rate is very low in phobic and obsessive-compulsive patients.

APPROACH TO PHOBIC AND OBSESSIVE-COMPULSIVE DISORDERS

Let us examine the principles and results of behavioral psychotherapy in two extensively studied syndromes—phobic disorders and compulsive rituals. Most behavioral approaches to their treatment employ a common principle called *exposure*. The concept of exposure holds that relief from phobias and compulsions comes from the individual's continued contact with the situations that evoke his discomfort. Clinicians need to search for those cues which trigger phobias and rituals and to confront the patient, given his agreement, with these cues. The principle of exposure does not explain why improvement occurs under these conditions.

Several controlled trials with phobic and obsessive-compulsive patients have shown that exposure treatments produce significant improvement in phobias or compulsions up to the latest 2- to 4-year follow-ups that are available. These results can be obtained with

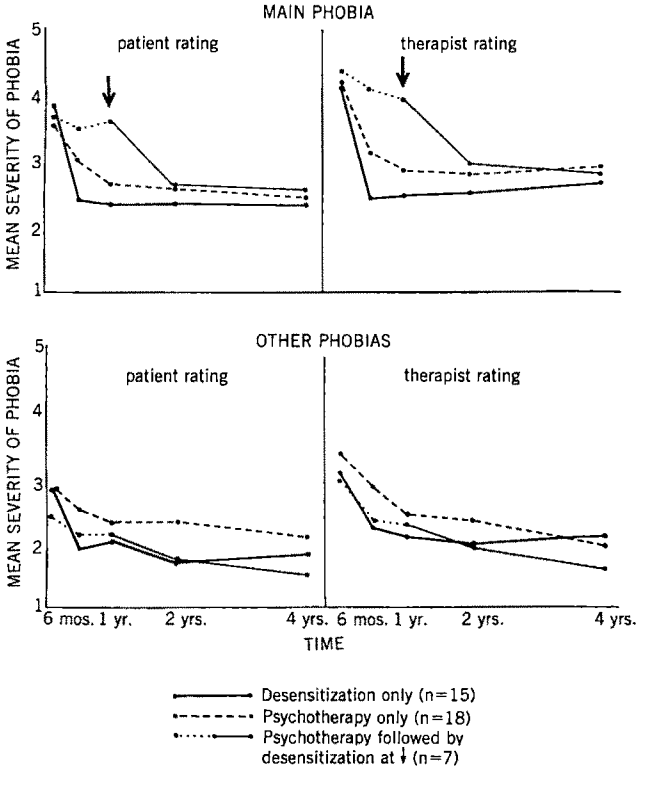
FIGURE 1
Therapeutic Influences: An Operational Framework



real-life exposure after 1 to 30 sessions. The last 100 patients treated in my unit required a mean of 11 treatment sessions. The more complex problems with wider ramifications require longer treatment. Chronicity of illness, however, is not important; even long-standing problems can be relieved in a few hours of treatment.

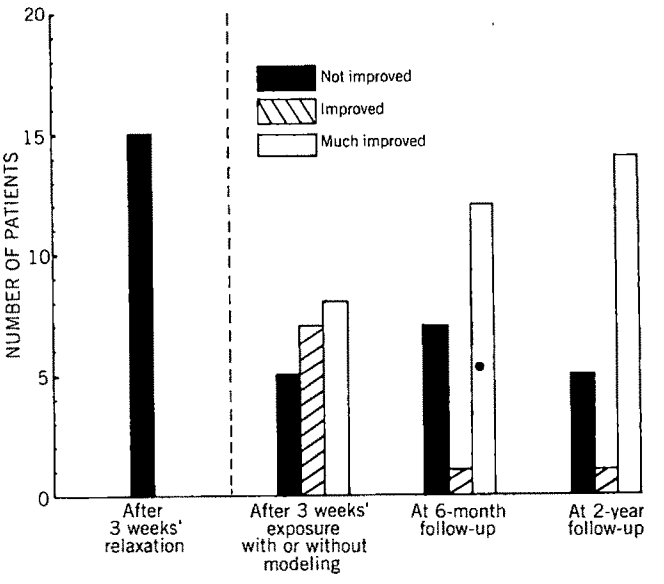
An early form of exposure treatment was Wolpe's (7) desensitization in fantasy, in which the patient is relaxed and asked to repeatedly imagine himself gradually approaching the object that causes him fear. Phobic images are visualized only for a few seconds at a time, and the subject is asked to relax between images. In an early series of trials by Gelder, Marks, and Wolff (8–10), desensitization in fantasy reduced phobias more than dynamic psychotherapy (see figure 2). Patients who did not improve with psychotherapy were given desensitization in fantasy, which reduced their phobias. The superiority of desensitization was gradually eroded as other patients similarly improved over the years, but subjects receiving desensitization improved earlier and with less treatment and maintained their improvement at 4-year follow-up (11) (see figure 2).

FIGURE 2
Improvement in Phobic Patients with Desensitization and Psychotherapy*



*Based on material from Gelder and associates (8) and Gelder and Marks (9). Reprinted by permission from "Phobic Disorders Four Years After Treatment: A Prospective Follow-Up," by I.M. Marks, *British Journal of Psychiatry*, volume 118, pages 683–688. Copyright 1971, the Royal College of Psychiatrists.

FIGURE 3
Improvement in Obsessive-Compulsive Patients with Exposure in Vivo and Relaxation*



*Reprinted by permission from "Treatment of Chronic Obsessive-Compulsive Neurosis by *in-vivo* Exposure: A Two-Year Follow-Up and Issues in Treatment," by I.M. Marks and associates, *British Journal of Psychiatry*, volume 127, pages 349–364, 1975. Copyright 1975, the Royal College of Psychiatrists.

These results were replicated by Gillan and Rachman (12) in a similar group of phobic patients. They found that desensitization in fantasy was superior to dynamic psychotherapy for the relief of phobias and that improvement continued to 3 months' follow-up. The amount of improvement with each treatment was comparable to that obtained in the earlier investigation.

More recently, emphasis has been on exposure in vivo. This gives quicker results by bringing the patient into contact with his discomforting situation without the relaxation exercises. A partially controlled experiment (13) showed that exposure in vivo improved obsessive-compulsive rituals significantly more than did relaxation treatment (see figure 3). In a subexperiment, patients who had exposure in vivo without preceding relaxation did just as well as those whose exposure followed relaxation. Improvement with exposure in vivo thus occurred whether it was given as the first or second treatment block (14). Improvement continued to 2-year follow-up (see figure 3).

EXPOSURE TREATMENT

Varieties

Exposure treatments come in many forms. To illustrate these, let the reader imagine a man with a height phobia quivering at the foot of a 20-foot ladder. His aim is to climb to the top of the ladder to retrieve a book titled *How To Overcome Your Fear of Heights*.

In this therapy many options are open. We can ask him to close his eyes, relax, and imagine himself slowly putting his foot on the first rung of the ladder and then relaxing again, and to imagine this several times until that inspires no anxiety, after which he can imagine himself on the second rung of the ladder, etc. That is *desensitization in fantasy*. If we asked the patient to carry out the same maneuvers in real life rather than in fantasy, that would be *desensitization in vivo*.

An alternative strategy would be to ask the patient to close his eyes and imagine himself standing right at the top of the ladder looking down, swaying, and feeling dizzy and scared at the same time, and to continue to imagine this until he feels better. This is *implosion* or *flooding in fantasy*. The *real-life flooding* variant would be (with his permission) to grab him by the scruff of his neck and thrust him to the top of the ladder and keep him there, sweating out his fear, until he becomes used to the situation. If for any of these procedures we first demonstrate to the patient what to do, e.g., if we precede him up the ladder, we would call this *modeling*. If we praise the patient each time he takes a step up the ladder this is *operant conditioning* or *shaping*. If we ask the patient to close his eyes and imagine himself persuading another patient with a height phobia that it is good for him to go up the ladder, this would be *cognitive rehearsal*. If the patient is asked to say to himself, "This is not so bad, I can really tolerate this fear"—that is *self-regulation*.

The Exposure Principle

These and many other techniques are called by a bewildering array of confusing terms. There is a natural tendency to seek a single explanatory principle behind diverse treatments, although reality is usually more complex than our constructs. A therapeutic element more pervasive than most in all the methods described for getting the height phobic up the ladder is that of exposure to a noxious stimulus until he becomes used to it. Alternative terms for "becomes used to it" are habituates, extinguishes, or adapts, each of these terms having overlapping implications. The noxious stimulus may be a troublesome fantasy, a feeling of uncertainty about the future, an airplane flight, or sexual intercourse. It is not my brief to discuss how such stimuli come to be perceived as noxious in the first place. However, once situations repeatedly produce discomfort, as in phobias and obsessions, then sufferers usually lose this discomfort by agreeing to remain exposed to those situations until they feel better about them.

Exposure is a similar concept to extinction. "Extinction" simply means that the response ceases to occur, without explaining why. Similarly, the concept of exposure simply holds that given enough contact with the provoking situation, the phobic or obsessive person ceases to respond with avoidance, distress, or rituals. Patients rarely become sensitized to a situation through clinical exposure. Unfortunately, we do not know what causes such sensitization, a reaction that has important theoretical implications. The problem is why 3% fail to improve at all, rather than a problem of relapse, which is a different but not a serious issue. The question is why exposure to a trauma sometimes produces phobias and at other times cures them. Which sets of conditions predict a traumatic or a curative outcome must still be delineated, but systematic research has begun to chart some dimensions of this problem.

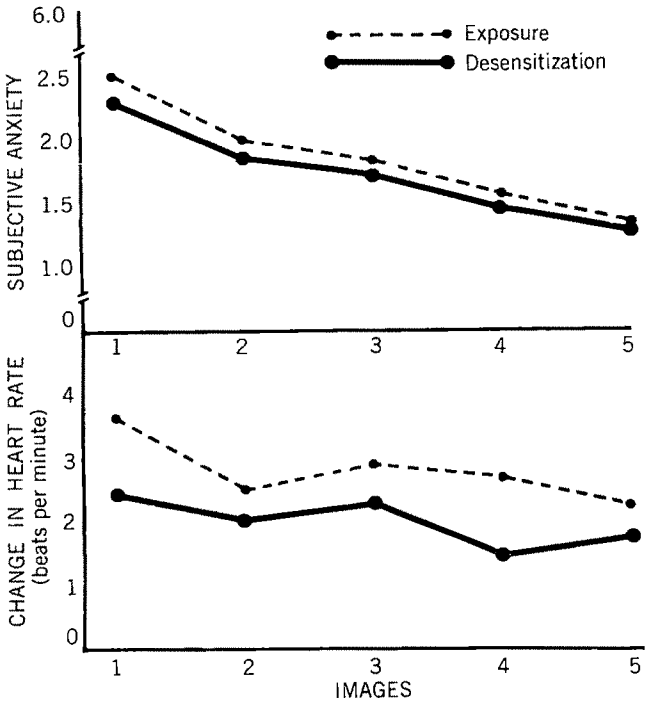
Low Anxiety

Wolpe has suggested that relaxation or other procedures are necessary to "reciprocally inhibit" anxiety during contact with the phobic stimulus, in order that improvement can follow. Many experimenters since then have found that the outcome to desensitization in fantasy is not impaired by omitting relaxation; the evidence points strongly to relaxation's being a redundant element in the therapeutic package (4). An example of an experiment that disconfirms the idea of reciprocal inhibition comes from Benjamin and associates' work (15) with chronic phobic patients. In a controlled study these patients imagined phobic images up a hierarchy while they were either relaxed or in a neutral affective state. The hypothesis of reciprocal inhibition predicts that relaxed subjects will show less anxiety to phobic images during treatment and have a superior outcome in reduction of fear.

The experimental manipulation was successful in producing two significantly differentiable treatment conditions. During treatment sessions relaxed patients had significantly less skin conductance activity between phobic images than did patients who had not been relaxed, i.e., they were less aroused. However, contrary to prediction from our reciprocal inhibition model, arousal between images during treatment did not correlate with decreased anxiety either during or after treatment. Relaxation did not increase the speed with which patients lost their fears during sessions and did not increase the extent of improvement at the end of treatment. During sessions, subjective anxiety and heart rate diminished at the same rate whether the phobic images were visualized during a state of relaxation (desensitization) or neutral affect (exposure) (see figure 4). After the end of each treatment condition the reduction in phobias was similar. This result was replicated by Gillan and Rachman (12), who found comparable outcomes in phobic patients after treatment by desensitization in fantasy with and without accompanying training in muscular relaxation. One can thus conclude that training in muscular relaxation exercises can be omitted from exposure treatment without impairing results.

Thus far I have dealt with relaxation accompanying exposure treatment. Relaxation without exposure is also not helpful. Rachman and associates (13, 14) found that compulsive rituals did not improve after 15 sessions with muscular relaxation but were reduced significantly after 15 sessions of exposure in vivo (see figure 3). These findings were subsequently replicated in a

FIGURE 4
Change in Heart Rate and Subjective Anxiety With Successive Presentations of Images During Desensitization in Fantasy With and Without Relaxation*



*Reprinted by permission from "Active Muscular Relaxation in Desensitization of Phobic Patients," by S. Benjamin and associates, *Psychological Medicine*, volume 2, pages 381–390. Copyright 1972, the British Medical Association.

study by Roper and associates (16). In fact, relaxation is such an inert factor in the treatment of phobias, compulsions, and even sexual deviations that it can safely be used as a control to contrast with more active treatments under investigation (17).

High Anxiety

A view opposite to that of reciprocal inhibition is the notion of implosion (18). The concept of implosion holds that for improvement to occur, anxiety must be maximally aroused during exposure until the patient is so exhausted that he cannot experience any more emotion. The evidence for this concept is based on uncontrolled clinical experience and can be paraphrased from Stampfl's comment about one obsessive-compulsive handwasher: "He who has lived in a cesspool for a few days [in his mind] will not worry later about a bit of dirt on his hands" (18).

The question is whether the deliberate evocation of anxiety adds to the therapeutic effect of exposure. Mathews and associates (19) found that high anxiety yielded no better results than low anxiety during fantasy exposure with phobics.

Whether high arousal facilitates in vivo exposure was tested by Hafner and Marks (20). Chronic agoraphobic patients were exposed continuously for 3 hours a day over 4 days to their real phobic situations, e.g., they were asked to shop in crowded supermarkets or to ride in subway trains until they felt better about being in these situations. In a high-anxiety condition the therapist commented on how bad the patients looked and mentioned all the catastrophes that might befall them in these situations. In a low-anxiety condition the therapist was reassuring, although he could not eliminate all anxiety. The experimental manipulation produced two significantly differentiable treatment conditions, with patients experiencing significantly more discomfort during exposure in the high-anxiety condition. However, this produced no difference in outcome on any measure. Low-anxiety patients improved at the same speed and to the same extent as high-anxiety patients. Thus anxiety did not facilitate improvement during exposure.

Further evidence that higher arousal is not especially helpful comes from a second controlled experiment by Hafner and Marks (20). In a double-blind study chronic agoraphobics were exposed as groups to their real phobic situations. Two groups of patients receiving diazepam reported less discomfort during exposure than patients receiving placebo, and they improved at the same rate as the placebo patients. Results from these studies of phobics are in line with findings from 3 earlier experiments that anxiety during exposure does not predict subsequent outcome in phobic patients (21–23).

In summary, phobias and obsessions improve with exposure treatment, but it does not matter whether patients are relaxed, neutral, or anxious during such exposure.

A more important variable is *duration of exposure*. In a Latin-square design, Stern and Marks (23) exposed chronic agoraphobics to flooding for long or short periods in fantasy and in vivo. Exposure in vivo gave significantly better results when carried out for 2 continuous hours rather than for 4 interrrupted half-hour periods in one afternoon. Long, continuous exposure was more effective in reducing heart rate during treatment sessions and in decreasing phobias at the end of treatment. Boulougouris and Rabavilas (24) replicated these results in obsessive-compulsive patients.

These experiments involved chronic patients, and the optimum time of exposure might well be less for those whose phobias are of more recent duration. Duration of exposure is presumably important because it gives certain unidentified processes more time to work while exposure is going on. For example, it might give people time to develop self-regulatory strategies to control their own emotions or to reach critical levels of habituation that may be necessary for lasting change to occur. The latter is implied in the question, "Is it best to end on a good note?" We have no answer as yet.

Fantasy Versus In Vivo Exposure

Another important variable is whether exposure is to the real or merely the fantasied situation. Re-

cent studies of volunteer phobics (4), agoraphobic patients (4), and obsessive-compulsives (24) have shown that phobias and rituals are reduced more efficiently by in vivo exposure. However, another study (25) failed to confirm this finding. The optimal strategy is probably to opt for in vivo exposure, using fantasy exposure only in special situations such as an unobtainable real phobic situation or a physical danger contraindicating marked anxiety, e.g., angina or severe asthma.

I have shown that what at first sight seem to be widely different forms of fear reduction—e.g., desensitization in fantasy, flooding in vivo, cognitive rehearsal, modeling, and operant conditioning—can all be subsumed under the rubric of exposure. The different names simply describe variations on the theme of exposure. The current treatment of choice for obsessive-compulsive and phobic disorders seems to be prolonged in vivo exposure of the patient to the noxious stimulus (with his consent). Response prevention may be yet another example of a treatment that finally works through exposure. In compulsive ritualizers, active prevention of the ritualistic behavior during exposure may be unnecessary except as a way of prolonging exposure, an idea supported in a pilot experiment by Lipsedge (26).

The principle of exposure to the stressful situation is important not only for phobias and obsessions but also for social deficits and sexual problems, although here it is bound up with training in interpersonal and other skills. In social skills training and in sexual retraining programs, of which Masters and Johnson pioneered one form, patients are required to rehearse appropriate behavior repeatedly in their problem situations until they lose their discomfort and acquire the requisite fluency of performance. In a controlled study of sexually dysfunctional patients, Mathews and Bancroft (27) found that one therapist was not significantly less therapeutic for a couple than a dual sex therapist team.

Shortcomings of the Exposure Model

The exposure hypothesis of fear reduction leaves several important facts unexplained:

1. A small minority of patients do not habituate during exposure despite their fulfillment of all criteria for predicted success, i.e., adequate motivation, absence of serious depression, no attempts to escape in fantasy or reality during exposure, and adequate duration of treatment. This is not a problem of relapse after improvement but rather the failure of any improvement at all. In these patients some crucial unknown factor for success is missing.
2. Some phobic, obsessive-compulsive, and other forms of anxiety improve with antidepressant drugs, without the need for any exposure.
3. Without any exposure to the specific phobic stimulus, anxiety sometimes remits after the abreaction of intense emotion, e.g., fear that is irrelevant to the phobic stimulus (22), anger (28), or other feelings.

A tempting explanation for the value of irrelevant fear might be that it teaches the patient a form of coping. This is a widening of the exposure hypothesis. It states that the patient benefits from exposure to irrelevant fear as he would to unpleasant emotions in general. This notion leads into muddy theoretical waters that are capable of experimental clarification.

The concept of coping with unpleasant emotions is related to that of stress immunization. It raises the possibility of preventing disorders by appropriate procedures in childhood and later periods. The idea was not new in ancient Sparta and amounts to the teaching of stoicism. Experiments with children (29) and students (30) indicate that this form of coping can be used in specific situations like visits to the dentist or pain in the forearm from ischemia induced by a pressure cuff on the arm. The question is to what extent stress immunization can be generalized, when it should be applied, in what way, for how long, and at what ages. We are only at the start of a long road of research into adaptive behavior.

DELIVERY OF BEHAVIORAL SERVICES

As behavioral treatments have been found to be effective for certain conditions, the demand for this therapy has greatly outstripped the supply of trained personnel. One possible solution is to produce new classes of therapists with shorter training than that required of doctors or psychologists. Operational research in this area has been successfully conducted in Britain. Given special training, psychiatric nurses working with patients with phobic, obsessive-compulsive, and sexual disorders achieved results as good as those of doctors and psychologists using comparable methods with similar patients (31).

The outcome of this work has been the development of an 18-month course, recognized throughout England and Wales, for the training of nurses in behavioral psychotherapy. These nurse-therapists act as the primary therapist in a therapeutic team while working with doctors or psychologists. This development greatly extends the number of patients who can be treated by a given team led by a doctor or psychologist. This is as true for private practice as for the public sector.

Many other classes of personnel have been involved in the administration of behavioral treatments for a wide variety of problems. More research is needed into possible models for the delivery of behavioral services in different countries. Such operational research might indicate improved ways of getting effective treatment to as many people as possible at a reasonable cost.

CONCLUSIONS

Behavioral psychotherapy is a vigorous growing point in psychiatric treatment. Several hitherto untreatable conditions now respond to behavioral treatment,

and it is at times a useful adjuvant for other problems as well. Behavioral psychotherapy is taking its place among the definitive treatments available for the relief of psychiatric suffering, and further advances in the field are appearing regularly. Although useful molecular theories are emerging to guide the discipline, no global theory is likely to be satisfactory in the foreseeable future. As always, as we learn to understand any area, the dialectic dance moves on to fresh ground.

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The Psychological Treatment of Personality Disorder

BY PAUL C. HORTON, M.D.

The author presents three case reports of individuals with personality disorder, none of whom demonstrated significant ability for transitional relatedness before therapy. In therapy, he conceptualized for the two adult patients the importance of their inability to experience in the transitional mode; he offered concrete potential transitional objects to the one child patient. The therapeutic results were good.

"PERSONALITY DISORDER" is rigorously defined in the second edition of APA's *Diagnostic and Statistical Manual of Mental Disorders (DSM-II)*.

> This group of disorders is characterized by deeply ingrained maladaptive patterns of behavior that are perceptibly different in quality from psychotic and neurotic symptoms. Generally, these are life-long patterns, often recognizable by the time of adolescence or earlier. (1, pp. 41–48)

The detection of personality disorder in adulthood requires clear evidence of significant and long-standing interference with the ability to love and work (2); psychological testing may be necessary to ensure the absence of covert neurotic or psychotic conflicts (3).

My colleagues and I have suggested that the central obstacle to the use of conventional psychotherapies for patients with personality disorder is the inability of these persons to experience in the transitional mode (3). This ability is nearly universal in children (4), well-functioning adults, and, in my experience, individuals in all diagnostic categories except personality disorder. Within the category of personality disorder there appears to be no difference between *DSM-II* subtypes in the ability to relate transitionally—if any competence in this area is observed, it is rudimentary and inconsistent at best.

Essentially, transitional relatedness consists of the soothing blending of internal and external reality. Winnicott (5) suggested that this "intermediate" or "third" area of human experience is where vital human illusions are formed. We stated that this ability

> takes its origin, in part, from the time a child is able to imbue a toy or other concrete object with the life he feels stirring inside of himself . . . . Even as he recognizes that the teddy bear is indeed cloth, stuffing and buttons, he suspends that aspect of reality just enough to permit himself to ascribe to the object those traits and qualities he needs to see in himself at the moment. In this way, *that* teddy bear becomes *his* teddy bear. Unique in the world and irreplaceable, it is the psychological area of the personal universe where the external physical reality of cloth, stuffing and buttons becomes inseparably interwoven with the internal life of its owner. (3, p. 621)

Maturation entails increasing subtlety and complexity of transitional relatedness (3, 6, 7), which is exemplified by an adult's ability to be psychologically soothed through contemplation of religious ideas, artistic expressions, or the virtues of another person. Unfortunately, those with personality disorders do not show this ability with such tangible, concrete objects as the teddy bear, let alone with the more abstract elements of the human condition.

I have evaluated and treated many people with personality disorders. I could do nothing for those who demanded drugs to service an addiction, who wished to avoid the consequences of crimes, or who came only because the parole board insisted. Many personality disordered individuals are in unremittingly serious straits and come to a psychiatrist only during a legal crisis. This article does not concern the treatment of out-of-control, impulsively aggressive individuals but describes a particular psychotherapeutic approach potentially useful in an outpatient setting. In the three cases with well-defined personality disorder that I present here, the focus is on the importance of the therapist's awareness of the normal role of transitional relatedness in human interaction and on the way in which this may be translated into ideas the patient can employ. The internal psychological significance of this intervention has been discussed elsewhere (8, 9).

CASE REPORTS

Case 1. A 34-year-old divorced mother of three presented with depression. Her paramour of many years was leaving her for a younger woman, and she feared that loss of her youthful beauty would deprive her of finding a more permanent partner. She emphasized her need for economic security.

She described having been brutalized by many men. Her father, an alcoholic, was often violent and sexually seductive with his daughters. Her eldest brother raped her when she was 12 years old. Her husband also raped her and compelled their daughter to perform fellatio on him.

Dr. Horton is in private practice, 41 Mattabasset Dr., Meriden, Conn. 06450; he is also Assistant Clinical Professor of Psychiatry, Yale University, New Haven, Conn.

Curiously, all of the many men she dated after her divorce proved to be manipulative, selfish, cruel, alcoholic, and sometimes sexually perverse. The relationship that brought her to treatment was one of convenience. The man with whom she was involved raised her standard of living by supplementing her welfare benefits. In turn she permitted him the companionship of herself and her children. He demanded little of her sexually; she was glad of this because she did not particularly enjoy sexual intercourse. There was little sense of emotional intimacy, sharing, or support, and he often complained of her coldness.

There was no evidence of transitional object usage. She had never been playful and had had no favorite toys or attachments to stuffed animals, blankets, pillows, pieces of silk, dolls, music boxes, powder puffs, songs, nursery rhymes, prayers, or other common and potential soothers of children. She felt only rage and resentment toward her promiscuous mother and icy disdain toward her father. When he died, she mentioned it only parenthetically and never showed any indication of grief or mourning.

I treated her with psychoanalytically oriented psychotherapy for 85 hours over 2 years. There was a 2-month interruption of treatment after the first year when her growing positive feelings toward me proved too unsettling for her. During the first few weeks of treatment she ventilated resentment toward her departed paramour and the emptiness of her life. She expressed curiosity about my questions pertaining to toys and other special objects but appeared to attach little significance to this line of inquiry. Later, as she described her arid relationships with men, I commented: "It's difficult for you to let others soothe you." She replied that men wished only to exploit her sexually. The basis of her inability to sustain a heterosexual relationship came to light in the evolving transference: she was subtly seductive and simultaneously hostilely suspicious. Hers had too often been a self-fulfilling prophecy. She consistently undermined the essential humanity of those who tried to be close to her by responding only to their frailties and ugliness. She tried hard to convince me that she had no failings and nothing to learn. She was suspicious of my discreetness with her, reasoning that I must be a "cleverer" seducer than most. She experienced anxiety and despondency as it became clear that I had no intention of seducing her. After many months I was able to tell her that she had many assets, but there were some things she needed to understand: there were men who could genuinely love and give to her; she had to learn how to recognize and accept the goodness in others; and she could be soothed but she would have to learn how to experience this. We spent many hours on this issue vis-à-vis her children and a new male friend. Insight followed:

> I've been thinking about what you asked a long time ago regarding toys and special things. My oldest child had a blanket that she carried all over the place when she was a baby. My 13-year-old still has her blanket and loads of stuffed pets. My boy has his stuffed animal. But it is true—I've never been able to find relief in anything. Some people can smoke, drink, bite their nails, use drugs, gamble—I haven't even been able to do things like that. It seems that all I have ever been able to do is to see bad things in everybody, including myself.

She made considerable gains in her ability to accept psychological soothing. This was accompanied by greater warmth and openness toward me and efforts to relate more soothingly to her children and others. At the conclusion of treatment she had even begun to show interest in and concern for her mother.

Case 2. A 62-year-old accountant and father of three presented complaining that his penis was too short. He was desirous of intercourse with his wife but stated that "she couldn't care less." In recent months it seemed that she had grown contemptuous of him. Moreover, he was not on speaking terms with two of his children. He had never been able to enjoy his work. He and his wife shared few friends; he was known derogatorily as a perfectionist. He stated, "When I do something, I do it with an intensity that is unbearable." He also suffered from long-standing mucous colitis.

The patient had been abandoned to his "Germanically strict" grandparents when he was 4 years old. The only remembrance of a toy was that of a wind-up locomotive with which he had been struck on the head by a playmate. There were no stuffed animals, favorite blankets, good luck charms, or other common soothers in early life. He grew up in a "state of anticipation—a big problem all [his] life." He was always worried and expected to be punished for unintended misdeeds. He became a tough, self-sufficient person; he stated, "I was going to be the next bank president. I used to dream of sitting back behind a big desk directing people."

The evolution of transitional relatedness, which normally begins in early childhood, did not manifest itself until he was in his early forties. He described his first memorable psychologically soothing inner experience as follows:

> I was hard at work as usual. Muzak was on. "Ebb Tide" was popular in those days and they were playing it. As swamped with work as I was, I stopped to listen. It was kind of peaceful. One of the girls even commented on my listening. After that I'd always stop to listen to that piece. That tune and a couple of others provided a few minutes of relaxation, an inner quiet, as if floating on a cloud.

His rudimentary transitional relatedness soon became submerged, however, as he sought relief in alcohol. A physical sedative was substituted for nascent psychological soothing.

> I drank to get that inner quiet. Music was qualitatively more soothing. But it was too short. You can't listen night after night to a few tunes. Alcohol loosened me up. I'd come home with my stomach churning. I'd have a few drinks and that waiting pile of work would get further and further away.

After several years the patient joined Alcoholics Anonymous because, "Whatever soothing I got from alcohol had stopped. Drink was killing me. I was sick and getting sicker." Subsequently, he took a brief interest in religion but was not able to find emotional sustenance. Like the previous patient, he had no compensations—nothing to protect him from his growing sense of loneliness. As his wife became less sexually accessible, he turned to psychotherapy for the first time.

A thorough history of potential transitional object usage was elicited early in treatment. Significantly, he could not recall having experienced anything like an "inner quiet" vis-à-vis another human being, even during sex. I summarized this: "You seem to have trouble letting others soothe you." I also said that I believed he had been struggling to grow in this way since his early forties when he had first experienced an inner quiet with music.

> This focus proved useful. The following week he said: Last week's hour was the best yet. That damn teddy bear thing had never entered my mind. I used to think it was the other way around. In fact, there's something I'd better tell you about. My little granddaughter carries a blanket all over the place. I'm afraid to say that from time to time I've tried to take it away from her. My

daughter has been getting mad at me about this. I thought there was something wrong with her having it. Now it looks like it's me who's got something to learn.

The remaining sessions revolved around his need for mutually soothing relationships with other human beings. That this theme was beneficial and even itself soothing was confirmed by his parting remark: "Since coming here I've gone without Lomotil for the longest time in 10 years."

Case 3. A 9-year-old boy, the second of three siblings, was brought by his mother for psychiatric evaluation with the chief complaint, "We can't get him to go along with what we want." The incident resulting in referral was his threatening his older brother with a knife. His mother reported that "he always seemed to have a mean streak, was easily upset, and would have to get even for anything that [was] done to him." He had no history of transitional relatedness.

From the outset he sought to structure the hour's activities as completely and rigidly as possible. After many sessions during which he verbalized very little about himself, his family, school, or friends, I introduced, with some initial difficulty, a teddy bear and two dolls for play. He named the teddy bear Bruce and the two dolls Sam and Blackie. The patient spoke for Bruce, and I spoke for the dolls.

Bruce hit the dolls often. When they protested the hurt, he replied, "I have no feelings. I'm super. See, I can fly and twist my arms and legs in all directions." He challenged the dolls to hit him to prove that he had no feelings of hurt. They refused, stating that they had no desire to hit him. He persisted: "Come on! Hit me! I'll show you. Come on, I don't mind. Hit me." Sam demurred, asking him what it was like to be without feelings of pain. He said it was good, that without such feelings he could be super. He denigrated human beings, especially women, as weak because they had feelings. Sam and Blackie stated that they liked being filled with feelings of all kinds, that feelings made life rich. Bruce replied anxiously, "But, I'm stronger—see, I can turn my head all the way around." Blackie said, "Its good that you are strong. But I wouldn't trade my feelings just to be strong." Bruce then revealed that he was a "robot bear from outer space" and that it was impossible for him to have feelings.

In a subsequent session, Sam offered to hug Bruce to help him learn about human feelings. The patient reacted with acute agitation, put Bruce down, and said, "I don't like that hugging stuff." Returning to play, he mercilessly pummeled the dolls until they had to retreat from reach. They resumed play only on the condition that he not hit them. He broke his promise, however, and again forced the dolls to retreat under a barrage of blows. Finally, after several broken promises, the dolls withdrew and stated that they would not return that day. Bruce begged them to come back. They hesitated. He offered, "If you come back, I'll even hug you."

During the ensuing week, the patient suffered for several days from a headache that reached its apex on the day of the session. At the beginning of the therapy hour *he* eagerly initiated play with Bruce and the dolls. Bruce informed the dolls that he was the "old Bruce's twin brother." He explained that he had come in Bruce's place because Bruce was away seeing a "computer doctor." The doctor would fix Bruce so that "he [would] have feelings too." After a while the old Bruce reappeared and offered to hug the dolls as proof of his cure. The headache remitted but the "fixed" Bruce was worried. In order to be cured, he had had to give up his super strength. He was now merely strong. At home, improvements were noticed: he had begun snuggling up to his parents on the couch; he had discovered his siblings and had begun playing with them. His mother—an emotionally timid woman—was bewildered by his accusation, "You don't love me." The social worker explored ways in which the parents, especially the mother, might show more emotional accessibility and initiative. She was encouraged, for example, to kiss the patient goodnight even if he did not ask. After 6 months of therapy the attacks on the dolls had become infrequent. Bruce now protected the dolls from the school principal, creatures from outer space, and other menacing figures. The patient's growing fondness for Bruce and the dolls was paralleled by substantial improvement in his relationships at home, at school, with peers, and with me.

DISCUSSION

Even though they had not experienced other than rudimentary transitional relatedness, the adult patients had witnessed transitional object usage by others. The first patient saw that all of her children used blankets and stuffed animals, and the second patient had been troubled by his granddaughter's use of a blanket. My inquiries regarding their own experience in this mode made them aware of an important psychological difference between themselves and others. Therapeutically useful associations and insights followed. The first patient contemplated her lack of psychological compensations; the second patient recognized that he had yet to learn about a mode of relating that children exhibit spontaneously.

The child patient required a more concrete introduction to transitional relatedness. After a period of time, a vehicle for play and dramatization of conflicts (the teddy bear) became invested with fond regard. Correlative treatment of the parents was also attempted, and I suggested that the mother, in particular, become more soothing to the patient.

This evolving treatment technique has several steps: 1) the therapist must identify the inability to relate transitionally as a core ego deficit; 2) he must interpret this to the patient, calling on the latter's observation of transitional object usage by others and explain in nontechnical language its relevance to the patient's relationships; and 3) he must then help the patient to contemplate various interactions, the outcome of which depends on his ability to experience transitionally or to permit others to do so. The therapist thus *orients* the patient to perhaps the most basic ingredient in human interaction.

This educative approach is attempted in tandem with the therapist's efforts to create a soothing atmosphere. I did not, for example, discuss the adult patients' difficulties without acknowledging their assets. The therapist must constantly scrutinize the transference to eliminate painful, frightening, or hostile distortions of present reality; for example, I eventually acknowledged the first patient's unspoken expectation that I would ultimately sexually exploit her.

In conclusion, it is encouraging to see that transitional relatedness may be an ability whose acquisition by personality disordered individuals does not have a

terminal critical period as, for example, does language development. This observation has positive implications for the treatment of individuals with these disorders.

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Psychosurgery: Some Current Observations

BY STEWART A. SHEVITZ, M.D.

The term "psychosurgery" encompasses a wide variety of different neurosurgical procedures applied in the treatment of behavioral and psychiatric disorders. Arguments about the effectiveness and ethicality of psychosurgery are often based on studies using outdated procedures or inappropriate patient populations. The debate over psychosurgery is also obscured by the frequent confusion between its use in classical psychiatric syndromes and in such controversial areas as aggression or violent behavior associated with temporal lobe epilepsy. The author believes that such factors need to be clarified so that practitioners can choose a personal position based on sound medical fact.

PSYCHOSURGERY IS one of the most complex, controversial, and misunderstood areas of psychiatry today. Even the name is controversial; some authors prefer the terms "psychiatric surgery" (1), "mental surgery" (2), "functional neurosurgery" (3), or "the surgery of the emotions" (4). Arguments on the merits and ethics of psychosurgery tend to be colored with emotionalism, offering antithetical viewpoints substantiated by references to a voluminous but contradictory literature. One can easily become bewildered by articles that range from severe condemnation of all forms of psychosurgery (5–7) to others espousing considerable enthusiasm (4, 8–13). This state of affairs, in combination with the notoriety that psychosurgery has gained from reports in the lay press, contributes to the rejection of psychosurgery out of hand by many psychiatrists.

The purpose of this paper is to offer the reader some guidelines with which to evaluate and put into perspective current efforts in the field.

I will focus primarily on the use of psychosurgery in the treatment of traditional psychiatric illness. This admittedly is discussing psychosurgery in a very restricted context, but one that may be most important to the practicing clinician. It is also a context frequently obscured by the debate over the use of psychosurgery in other behavioral disorders. The use of psychosurgery in more controversial areas such as aggressive behavior and violent behavior in association with temporal lobe epilepsy will be briefly touched upon.

One of the major difficulties in this area is that articles discussing different psychosurgical procedures, with different side effects, for various indications, and with various results are often lumped together indiscriminately when the merits of psychosurgery are debated. I will attempt to tease out some of these important variables that deserve individual attention.

TYPE OF PROCEDURE

In the 40 years since Moniz' first prefrontal lobotomy in 1936, a large variety of neurosurgical procedures have been employed in attempts to treat abnormal behavior. These include the standard lobotomy, transorbital lobotomy, cingulectomy, topectomy, gyrectomy, and bimedial leucotomy, among others (14). The frontal lobes have been assaulted by everything from scalpels to icepicks, from thermocoagulation to cryogenic probes, from radiation to ultrasound.

As Knight (15) has said, one can see in retrospect that procedures such as the standard lobotomy of Freeman and Watts were developed by clinicians who were perhaps over-eager following Moniz' demonstration that patients occasionally improved because lobotomy lessened the intensity of their emotional reaction to delusions or hallucinations. This was unfortunate because adequate physiological and anatomical studies of areas involved had not been performed. Hence, large areas of white matter were sacrificed in attempts to divide the unknown pathways involved. It was not possible to find specific areas in the prefrontal region responsible for clinical change following isolation or ablation. Clinical improvement and personality change appeared to correlate quantitatively with the amount of prefrontal cortex separated from its white matter. Marked blunting of the individual's personality and frequent surgical complications were undesirable side effects that occurred all too often with the early procedures (14, 15).

Recent evidence suggests that the beneficial effects seen following standard lobotomy might largely be at-

When this work was done, Dr. Shevitz was a resident in the Department of Psychiatry, Dartmouth Medical School, Hanover, N.H. He is now Chief of Psychiatry, Public Health Service Indian Hospital, Tuba City, Ariz. 86045.

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tributable to the disconnection of relatively discrete, functional areas of the orbital and medial frontal cortex from intermediate limbic circuits that lead to autonomic outflow pathways via the hypothalamus and brainstem (4, 15, 16). Efforts are now being made to attack these areas selectively to achieve therapeutic benefit without the adverse effects of inadvertent cutting of other fibers (4, 10–12, 17, 18). One example is the work described by Sykes and Tredgold (10). A form of orbital undercutting first developed by Scoville and Wilk (19) and modified by Knight (10, 20) is employed. By direct vision through burr holes, the orbital cortex (the ventral surface of the brain overlying the bony roof of the orbit and encompassing Walker's areas 13 and 14) is undercut at the junction of the white and gray matter. Such a procedure has a mortality rate of 1.5% and a postoperative epilepsy rate of 16% (well controlled by medication in most cases). Most importantly, only 5% of the 177 patients intensively studied by Sykes and Tredgold (10) were considered by them to have developed adverse behavioral effects following the operation, a marked improvement over the results of older procedures.

A further modification using radioactive yttrium implants stereotactically placed has also been developed by Knight (4). This procedure was originally thought to involve the substantia innominata; it is now termed a "stereotactic subcaudal tractotomy." In addition to a mortality rate of less than 1% from the operation, two reports reveal a postoperative epilepsy rate of only 1% to 2.2% (8, 9). Behavioral and psychological disturbances were lasting and troublesome in only 3% and 7% of the patients; 86% and 93% showed no undesirable symptoms at all.

These are but two examples of "modified" procedures. A number of other modified procedures involving cingulate and orbital regions have been described in the literature (11–13, 17, 18).

The important point to realize is that one must make distinctions between the various psychosurgical procedures before one can evaluate them. The standard open lobotomy that isolated 160 square centimeters of frontal cortex is markedly different from a stereotactic tractotomy in which only 8 square centimeters of orbital cortex (and possibly some fibers from the anterior cingulate gyrus) may be affected (4). Lesions produced in the cingulate or orbital regions do not appear to cause the severe emotional blunting or impairment of intellectual function that the older, more extensive procedures often did (4, 10, 18). Most practitioners concur that the detrimental effects of the standard lobotomy and its variants certainly outweighed the benefits (of which there were some) and have discarded these techniques. These techniques should be considered of historical interest only and not as evidence that subsequently developed procedures must invariably result in the same adverse effects. The difficulties with human heart transplantation did not invalidate all surgical approaches to the treatment of coronary heart disease. Similarly, the evaluation of current psychosurgical procedures should rest on their own individual merits and should not be judged by the adverse effects of their predecessors.

DIAGNOSTIC CATEGORIES

Much prefrontal surgery was originally aimed at schizophrenic patients before major tranquilizers were available (14, 21). Today schizophrenia should no longer be considered a prime indication for such procedures (8–10). Except when depression or severe anxiety are prominent, lobotomy has little to offer in acute or chronic schizophrenia and certainly cannot reverse chronic deterioration (14).

Psychiatrists often fail to recognize that many proponents of psychosurgery do not advocate this treatment method for schizophrenia. The best follow-up studies on psychosurgery from a psychiatric point of view are found in the British literature. Table 1 summarizes results from three such reports (8–10) mentioned previously. The table shows the number of patients out of the total patient population by diagnostic category who were either fully recovered or had only minimal symptoms following leucotomy. Follow-up occurred over a period of years following the operations. The best results were obtained with patients previously suffering from intractable affective illness. A number of patients with severe obsessional or anxiety neuroses also improved, although not as frequently as depressive patients. Results are also shown for the few schizophrenic patients operated on. When one considers the relatively intractable illnesses in this patient population, the results are noteworthy.

It is evident that although psychosurgery was originally employed extensively in treating schizophrenia, its effectiveness is now better evaluated with intractable depression, anxiety states, and obsessional neuroses. One is reminded of the history of imipramine, which was originally developed in the search for a better antipsychotic compound. Although it was found to

TABLE 1
Proportion of Postleucotomy Patients Who Were Recovered or Had Minimal Symptoms at Follow-Up

Study	Diagnostic Category			
	Depression (all types)	Obsessional States	Anxiety	Schizophrenia
Sykes and Tredgold (10)	68 (98)*	6 (20)	11 (25)	4 (13)
Ström-Olsen and Carlisle (8)	42 (75)	10 (20)	19 (46)	0 (5)
Göktepe and associates (9)	53 (78)	9 (18)	15 (24)	0 (4)

* Numbers in parentheses represent the total population of patients who received leucotomies.

be ineffective for psychosis, its mood-elevating properties in certain depressed patients were noted to be remarkable, and it has developed into one of our most effective antidepressant agents. The value of imipramine in treating depression is not invalidated by its ineffectiveness in treating schizophrenia. Similarly, psychosurgery's unsuitability for the treatment of schizophrenia today does not negate the possibility that it may be of value in other psychiatric disorders.

It is important to consider such factors when one is appraising a particular study from the literature on psychosurgery. Often, studies quoted as evidence against the efficacy of lobotomy (22, 23) have used a modification of the standard lobotomy procedure of Freeman and Watts or similar extensive procedures that cause broad lesions of variable extent and placement. Also, as pointed out by Post and associates (24), the subjects in many of these studies were hospitalized chronic schizophrenic patients. Such studies substantiate the fact that the older procedures are not of great value when used with schizophrenic patients, but these procedures and that patient population are not the areas in which psychosurgery is currently being investigated.

USE WITH BEHAVIOR DISORDERS

Psychosurgery, like many new treatment techniques, has been advocated for a wide variety of disorders. These have ranged from ulcerative colitis to drug addiction to hypochondriasis (14). Much of the current controversy regarding psychosurgery relates to its use with aggressive and violent behavior (6), particularly the treatment of temporal lobe epilepsy by amygdalotomy. This area has been well publicized in the lay press, especially the efforts of Mark and associates (25–27) to relate some forms of violent behavior to epileptic discharges in the amygdala. Such work is highly controversial and certainly has to be considered experimental at this time. The term "psychosurgery" is often applied to the lesions made in the amygdala to control the epilepsy that presumably is causing the violent behavior. Whether the behavior in question is actually secondary to the epileptic discharges is a subject of intense debate, as is the issue of side effects from this particular operation. Similarly, psychosurgical procedures are sometimes used to control behaviors that may or may not be considered "illness," for example, criminal behavior (6) and sexual deviations (28).

It is important to distinguish between the use of psychosurgery in these controversial areas and its use for such classical, well-defined psychiatric illnesses as involutional melancholia or severe obsessional neuroses. The controversy over a treatment modality such as psychosurgery should not be confused with the controversy over whether or not certain disorders for which it might be advocated are valid illnesses. The validity of psychosurgery should be considered with respect to its use in classical, clear-cut psychiatric syndromes accepted as disease entities by the medical community. One must have established the validity of the illness being treated before the efficacy of treatment becomes a meaningful question.

ABUSE OF PSYCHOSURGERY BY SURGEONS

A review of the world literature on psychosurgery quickly makes one realize that there is a wide variation in the indications and criteria that psychosurgeons use in choosing their cases (5). It would appear that not all employ the rigid standards of patient selection that would be demanded by many psychiatrists or by other surgeons. This should not, however, invalidate studies in which higher standards are met.

CRITERIA FOR PATIENT SELECTION

The current consensus seems to be that psychosurgery is not the treatment of choice for any psychiatric illness (14). The continuing debate centers on the fact that some practitioners do consider it a treatment of last resort in certain very specific cases. To be even considered for psychosurgery a patient should have proven to be unresponsive to all other appropriate psychiatric treatment modalities and he should be disabled to the point that any potential hazards from the operation are felt to be equal to or less than the disability suffered as a result of his illness. The probability of future remission without further treatment should also be considered nil (2).

Lehmann (29) has described his personal criteria for referring a patient for a psychosurgical procedure. He included the following criteria:

1. The disease should be a chronic tension state, anxiety, depression, or obsessive-compulsive disorder.
2. The patient should have a good premorbid personality.
3. The patient must have been disabled for at least two years.
4. Treatment by any of the following treatment modalities that were considered appropriate proved unsuccessful: a) psychotherapy by an experienced therapist for six months; b) antianxiety drugs such as chlordiazepoxide hydrochloride in doses up to 100 mg per day for at least two months; c) imipramine, 250 mg per day for six weeks; d) chlorpromazine, 1000 mg per day for two to three months; e) one or two courses of ECT (10 to 15 treatments per course); and f) a trial of lithium for one year if there is recurrent depression or bipolar illness.

Certainly every psychiatrist and surgeon would have his own modifications and additions for such a list; it is offered as an example of one person's opinion. It should also be recognized that there are many physicians who would not recommend psychosurgery under any circumstance.

ETHICS

The other major area of controversy regarding psychosurgery concerns the ethics and morality of such procedures. I will not address this very important issue at length because it is such an individual matter for each practitioner. The range of opinion is extremely diverse. One of the most prolific critics of psychosurgery, Breggin, considers such operations to be a horrible mutilation of man's highest function (5–7, 30). He abhors the idea of the frontal lobes being attacked in such a fashion because he feels they represent the seat of man's humanity. Others feel that an intractably ill individual who is terribly disabled by illness but can still voluntarily consent to such a procedure should not be denied the possibility of therapeutic benefit. In any event, one's position on this matter is probably not determined by rationality and deductive reasoning but rather by one's inner values and ethical interpretation of one's role as a medical practitioner under the Hippocratic oath.

CONCLUSIONS

One reason for the precipitous decline in the popularity of psychosurgery that followed the wave of enthusiasm it inspired in the late 1940s was the frequent complications—especially those involving personality changes—found with the more extensive procedures of that era. Another reason was the negative results of such comprehensive investigations as those of the Columbia-Greystone associates (31) concerning psychosurgery that involved selected partial ablation of the frontal cortex. Perhaps the most important factor in psychosurgery's fall from popularity was the advent of modern chemotherapy (major tranquilizers). It was the schizophrenic population—at which psychosurgery had been originally aimed—for which these new drugs were most effective.

Today the possible merits of certain psychosurgical procedures for some disorders have once again brought the area under intense public and professional scrutiny. Along with the formidable difficulties of clinical investigation, the problems of adequate control and criteria for patient selection are particularly difficult. In addition, it is known that adverse effects as judged by psychological testing may not become evident until 10 to 12 years after the surgery (32). The need for definitive studies to confirm the benefits reported and to answer the questions raised by critics is evident. Certainly such irreversible procedures deserve the severest scrutiny.

I have attempted to isolate some of the important factors that need to be weighed in evaluating the merits of this therapeutic modality. The purpose of this paper is not to advocate psychosurgery. It is a plea to every physician concerned with the subject to base his position on sound medical fact and practice. Only in this manner can we do proper justice to those working in the field. More important, only in this manner can the best interests of our patients be served.

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Prompt Publication Policy

The *Journal* would like to remind authors who are particularly interested in early publication (whether to establish priority of an idea, reveal innovative results of new research, or for other pressing reasons) of its prompt publication policy. Under this policy, short (maximum of six double-spaced pages) manuscripts will be considered for publication in the Brief Communications section of the first available issue. These manuscripts must still undergo expert scrutiny before being accepted, and two months must be allowed for the printing process. However, the time required for publication of these short articles can be lessened considerably.

Authors who would like to have their manuscripts considered under this policy should indicate this desire, as well as the reasons for it, when they submit their manuscripts.

Services for the Aged in Community Mental Health Centers

BY ROBERT D. PATTERSON, M.D.

The author studied the services provided for the elderly at eight community mental health centers. He describes discrimination against the elderly, the reasons why relatively few elderly persons seek care, and innovations in treatment. He discovered that high-quality care depends more on staff awareness of the unique problems of the elderly than on specialized services. The author recommends a more public-health-oriented approach that would set priorities on the basis of community needs.

THE PURPOSE of this paper is to aid the program planning of mental health services for the elderly. The needs of the elderly have often been neglected by mental health programs. There has been little clinical research and very little research or discussion about program planning to meet their needs (1, 2). Two reasons for this neglect are negative attitudes toward the elderly and a false belief that most psychiatric conditions in the elderly are untreatable. The purpose of the survey reported here was to determine how community mental health centers are responding to the needs of the elderly.

Since the survey was conducted Congress has revised the community mental health center legislation (Public Law 94-63) to require a full range of "specialized" diagnostic, treatment, liaison, and follow-up services for the elderly in all federally assisted centers. Providing the required specialized services will, in most instances, not require segregating the elderly from patients of other ages. The legislation also provides a mechanism for consultation and education grants. If adequately funded, these grants can be especially beneficial to the elderly.

THE SURVEY

My associates and I visited 8 federally funded community mental health centers as part of a contract study for the National Institute of Mental Health. The centers were selected to provide a wide range of geographic locations and degrees of urbanization as well as a broad representation of communities with different ethnic, social, and age compositions. The sample may not be representative because centers with major organizational problems were excluded.

Each center was visited by 2 mental health professionals who spent 3 days in the area. Two days were devoted to obtaining information from persons not employed by the mental health center, such as welfare department and nursing home personnel and others familiar with the needs of the community. Elderly patients were interviewed, and in each area there was a meeting with at least one senior citizens' club or similar group. One day was devoted to interviews with staff members of the mental health center.

RESULTS OF THE SURVEY

Priorities

The primary orientation of the centers was to serve those who requested help. Since only a small number of the elderly asked for help, the amount of service given to them was small. There were few instances of a public health approach that set priorities on the basis of community needs. Thus the elderly were not given high priority.

Other factors also influenced the priority given to the elderly. In one case the community board asked the director of the center to give higher priority to the elderly. This request seems to have resulted from the experience of some of the board members with their own aged parents and from their awareness of the high proportion of elderly in the community. The possibility of earmarked federal funds to serve the mental health needs of the elderly had already stimulated some preliminary planning for improved programs.

Three of the centers gave special attention to the mental health needs of the aged. One had a full-time geriatric service coordinator (a social worker) and a small staff that provided direct service and consultation. Another center had a social worker with extensive experience in community development who devoted half of his time to planning, organizing, and coordinating services for the elderly. The third center provided staffing for a psychiatric unit in a nursing home. However, the unit was not prepared to provide intensive psychiatric care.

Having a specialized staff was not an indicator of the

Dr. Patterson is Lecturer on Psychiatry, Laboratory of Community Psychiatry, Harvard Medical School, 58 Fenwood Rd., Boston, Mass. 02115.

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priority given to the needs of the elderly, since some programs without specialized staff gave them appropriate attention.

Services

The centers differed widely in the policies and practices of their clinical services.

Inpatient services. All of the inpatient units admitted elderly persons. Most patients completed their treatment without transfer to a state hospital; direct admission to a state hospital was uncommon.

One inpatient unit admitted persons from nursing homes for brief treatment, most often for behavior problems that overwhelmed the nursing home staff. Behavior modification techniques were often used, and they seemed well suited to help many of these patients. Nurse consultants were available before admission and for follow-up.

In all, the patients we interviewed felt that they had received good care. Several who had experienced relapses after earlier hospitalizations praised the aftercare programs of the mental health centers.

It was evident that in many cases elderly patients received less adequate care than other adult patients. For example, one center, which often had a waiting list, required that the inpatient unit director approve the admission of all persons over age 65 who sought admission. Another center with a shortage of beds often sent elderly patients to a general hospital's extended-care facility rather than sending them to the psychiatric unit.

Although most state hospitals have screening procedures to reduce the number of inappropriate admissions, there were instances of patients being routed to bypass these procedures. Thus patients were admitted to state hospitals when admission to a mental health center would have been more appropriate. The staff of a state-operated geriatric screening unit, which interacted with several centers, reported that they had great difficulty in obtaining admission for geriatric patients.

When priorities for admission had to be set, it appeared that the elderly were likely to be given low priority.

Partial hospitalization. Among the centers visited, elderly persons were seldom referred to day care, although day-hospital programs have proved valuable for elderly patients (3).

Staff members of several day hospitals felt that their programs were not appropriate for elderly patients. They showed a lack of awareness that substantial numbers of elderly patients can benefit from programs designed to include them.

Outpatient care. Only one of the centers had a specialized staff to treat geriatric outpatients. Two had psychotherapy groups for the elderly, which were organized to meet the needs unique to their stage in the life cycle.

Two centers provided nurses who made frequent home visits to the elderly. Usually, an elderly person readily accepted a nurse's care because of her easily identified medical role. Sometimes only after establishing trust would the nurse tell the patient that she was from the mental health center. Another advantage of using nurses with elderly patients is that they are capable of responding to situations where both physical and emotional needs must be met.

Emergency services. Few elderly patients asked for emergency services, and no center had separate emergency facilities for them. A suicide prevention and crisis hotline in an area with a high suicide rate and high proportion of elderly was rarely used by old people.

One mental health center used the general hospital emergency room to receive psychiatric emergencies when the center's outpatient department was closed. The emergency service of a general hospital has the advantage of providing facilities for the physical diagnostic studies frequently needed by the elderly.

Consultation. The centers provided little consultation to the organizations that serve large numbers of the elderly, e.g., nursing homes, welfare departments, visiting nurses, "meals on wheels," and home health care programs.

Consultation to nursing homes was usually conducted on a case-by-case basis, although experienced consultants believe that greater long-range impact results from in-service education and policy-oriented consultation (2).

Nurses provided most of the consultation in nursing homes and were given considerable autonomy and responsibility in this area. They found such work rewarding, and their initiative built strong consultation programs. Backup by a psychiatrist who knew the nurse's capability was important, as when a nurse recommended a change in medication or hospitalization.

In one innovative program, the mental health center supervised staffs for 2 homes for the aged in conducting group therapy sessions for a large proportion of the residents.

Comment

The centers we studied devoted little effort to organizing prevention or treatment services based on an assessment of the greatest need for the population as a whole; rather, they focused on the elderly who came to them for help.

It is clear that elderly patients are not using inpatient, day-treatment, or outpatient services in proportion to their numbers. The site visits and a previous study (2) indicate several possible reasons for the low utilization rate.

1. *Lack of belief in appropriateness of mental health assistance.* The elderly are less likely than the young to believe that their problems are appropriate for mental health assistance. Normal elderly people outside the centers reacted to questions about mental health problems with anxiety, often expressed by nervous joking. Many thought that mental health services were only for "crazy people"; they associated mental prob-

lems with long hospitalization. Some described problems of depression, anxiety, and psychosomatic illnesses, but they had not considered it appropriate to take these problems to mental health professionals.

The majority of the centers were not devoting appreciable effort to educating the elderly about mental health. Many non-mental health care givers knew little about the services of the centers, and some believed that the centers did not wish to treat elderly patients.

One exception was a center that held public educational meetings at which topics relating to mental health and community organization were discussed. One of the purposes of these meetings was to increase community awareness of the individual's psychological needs. The sessions were highly regarded by other agency personnel. It seems likely that these efforts were successful because numerous care givers were remarkably alert to their clients' psychological needs and were familiar with the mental health services available.

Needed services can sometimes be provided without requiring elderly persons to accept the stigma of being mentally ill. Centers can cooperate with other organizations to provide mental health services as part of larger programs that carry no stigma. For example, two centers participated in the operation of multiagency sponsored groups that met away from the mental health centers and had goals that included socialization, education, and community organization as well as goals associated with traditional group psychotherapy. Another center participated in health screening clinics conducted by a health department for people over age 40.

2. *Transportation*. Problems in getting to a treatment center can be a serious obstacle for the elderly. Satellite facilities and home visits may help to overcome the transportation problem.

3. *Cost of service*. The ability of the elderly to meet the cost of mental health services is limited by low retirement income and the limitations of Medicare coverage.

One center that received little government financial support depended heavily on patient fees. As a result, it had a reputation among the elderly for being expensive. Another center, forced to reduce its day-treatment program because of reductions in Medicaid benefits, created several socialization groups in alliance with the local welfare department. The groups used a small paraprofessional staff. The change probably increased the overall effectiveness of the center because it served more elderly people and did not label them as mentally ill.

DISCUSSION

We believe that every mental health center should have a staff member designated as the advocate for the elderly. This advocate should be involved in policy making, clinical and organizational consultation to the center staff, in-service education, and community development.

We believe that services for the elderly should be integrated with the clinical services for other adults. Specialized services are likely to be inferior because the elderly are given low status in our society.

The future of population-oriented mental health services for the elderly is threatened. Increasingly, funds for health care come from third-party reimbursement, which requires a fee for service to a designated patient. Medicare is designed this way, and national health insurance, which is likely to be established in a few years, will probably follow the same plan.

How, then, are mental health centers to support population-oriented programs? NIMH funds, which in the past have supported some of these efforts, are being systematically reduced. Local and state funds could be used for these purposes when they are less needed to pay for direct patient services. However, after the establishment of national health insurance, community mental health centers are likely to be closely scrutinized about their need for continuing support from local and state governments. The centers will be required to convince such potential supporters that population-oriented services are valuable; yet most centers have not invested great resources or imagination in these programs, much less documented their effectiveness.

There is a grave danger that many centers may give up the struggle for population-oriented programs and choose the fee-for-service mode of providing care. This would be especially deleterious for the mental health care of the elderly.

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Therapy Groups for Women

BY CAROL S. WOLMAN, M.D.

The author describes her experiences with two all-female psychotherapy groups, comparing these groups with both mixed-sex and consciousness-raising groups. Problems included a tendency of group members to drop out when dependency on the group became too intense and a lack of referrals to replace these dropouts, with resulting decreases in group size. The special strengths of such groups include the opportunity to concentrate on issues relevant to women, the possibility for development of a support system, the lack of sex-role divisions of labor, and the increased respect of members for other women and for themselves.

THIS PAPER describes my experiences with two outpatient psychotherapy groups for women only. Such groups are relatively rare in spite of the women's movement and have not received much attention in the psychiatric literature. They differ from both mixed-sex therapy groups and women's consciousness-raising (CR) groups and offer advantages over both for many women.

COMPARISON WITH MIXED THERAPY GROUPS

Since Broverman and associates documented the unconscious sexism of most therapists 5 years ago (1), very few consciousness-raising articles have appeared in the psychiatric literature. If what therapists read and write bears any relation to the way they practice, it seems that many therapists are still unaware of their own sexism. I define sexism as a belief system that includes the assumptions that 1) men are superior to women in various ways, 2) men should dominate women, and 3) women should be defined chiefly by their relationships with men.

A few therapists have written about sexism. Miller and Mothner (2) argued convincingly that women's oppression is real, and their striving for liberation is valid. They insist that the conflicts inherent in this striving should be examined in the therapeutic situation. Rice and Rice (3) also advocated active examination of the psychically crippling effects of sex-role conflicts in therapy, and called for more women therapists and use of the women's movement as a resource. Mintz (4) listed four sexist assumptions often made by therapists and therapy groups: 1) a woman who does not want to marry is abnormal, 2) a woman who does not want children is abnormal, 3) it is normal for a woman to lack ambition and 4) men should take the initiative in heterosexual courtship.

It is difficult enough to challenge sexist assumptions in an all-female setting; it is far harder in a mixed group, where the tendency of many women to seek approval and even self-definition from men is much more likely to be covertly reinforced than overtly challenged.

None of the classical literature on group therapy (5–7) even addresses the issue of the male-female power relations within therapy groups. There continue to be articles in which women are defined by their male connections (8, 9). Schonbar (10) and Fried (11) both discussed the power of a female therapist to act as a positive role model in mixed therapy groups, but they did not write about covert male dominance of women and how it could be handled.

A therapy group only for women eliminates most of the here-and-now sexism that goes on unconsciously in any mixed setting. It provides patients with the opportunity to explore the ways in which sexism affects their individual lives. Moreover, it conveys several covert messages that mixed groups do not convey: 1) women exist apart from men and are persons within themselves, 2) it is permissible and even desirable for women to bond strongly to each other and not to be chiefly defined by their bonds to men, 3) women are worthwhile and have valuable gifts to offer each other, and 4) men have no monopoly on power and goodness.

The patients in both groups I will describe were permitted, in fact encouraged, to become friends and to give each other support outside the group meetings. Therapists in most mixed-sex groups discourage outside contacts, their rationale being that such socializing would divert energy from the therapeutic endeavor. They may also fear sexual pairing. Mixed groups are usually expected to be time- and space-limited events where patients learn to change as individuals, transferring their growth to their home social situation.

My cotherapists and I had a somewhat different philosophy. Most of the women we saw were socially isolated and especially lacking in female friends. As Betty Friedan (12) explained, this restriction and isolation of

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Dr. Wolman is in private practice at 4348 26th St., San Francisco, Calif. 94131.

women is a post-World War II phenomenon. Smith-Rosenberg (13) documented the fact that nineteenth century American women maintained extensive female support systems. We believe that in addition to exploring their individual problems in a sympathetic group setting, women need to learn anew to depend on and value each other. Such interdependence eases problems of isolation and anomie and also teaches people to be dependable and to value themselves. We encouraged our patients to call each other for support between sessions, and at times helped to organize phone relays when a group member was in a crisis situation.

All-female groups obviously eliminate heterosexual interaction, and there was no homosexual bonding in our groups, although one patient was homosexual. It could be argued that these groups robbed patients of a chance to work through their oedipal conflicts. Like most people who seek psychiatric help, however, almost all of our patients had severe problems with basic trust and/or self-control and needed to concentrate on these problems. The absence of sexual attractions and games within the groups was a helpful simplification.

It might be feared that men would readily be scapegoated in all-female groups. This rarely happened. In fact, the patients tended to err on the side of self-blame, and the therapists often pointed out that a patient's grievance against a man might be legitimate and that she might have a right to be angry. For the most part, however, the patients concentrated on themselves and each other.

COMPARISON WITH CR GROUPS

These all-female therapy groups differ from CR groups (14–17), which form within the women's movement, in a number of important ways. The guding principle of most CR groups is that "the personal is political." As each woman shares her individual unhappiness, the group struggles to understand how her story illustrates sexism and to reduce her feeling of personal responsibility for her suffering. It is considered axiomatic that the problem is society-wide. Individual solutions are therefore impossible—women must fight sexism collectively. Paradoxically, many reports of CR groups (18–20) have emphasized the increased strength and personal mobilization that women gain from the CR experience.

The women who joined our therapy groups differed from women who seek CR groups in that they had defined themselves as psychiatric patients and were seeking professional help. They were referred to us by male therapists or male-dominated institutions and had not sought a women's group. They saw themselves as passive and helpless neurotic or dysfunctional individuals; they did not relate their problems primarily to their gender. Women who join CR groups may also be troubled, but they are seeking confirmation of the idea that at least some of their troubles are socially induced and occur because they are women. They have already opened themselves to the ideas of feminism and are functioning well enough to seek actively the specific type of group they desire.

CR groups are leaderless and opposed to expertise, although a woman with previous CR experience may help to get a new group going. No fee is involved, and the place, frequency, and duration of meetings are agreed on by the members. In contrast, the therapy groups I will describe had two leaders, both of whom had expertise as psychotherapists and as group facilitators. Patients paid $10 per session and met once a week for 90 minutes at a place chosen by the therapists. Structurally, these groups had more in common with mixed therapy groups than they did with CR groups.

DESCRIPTION OF THE GROUPS

The two groups developed quite similarly in some ways, and the differences between them were often instructive. H. Kathryn Ratner, M.S.W., and I worked with group A from November 1972 to June 1974. The group met as a service of the psychiatric outpatient clinic of a university hospital and residency training program. Most of the women in group A had never been hospitalized and received diagnoses of depression or psychoneurosis in their clinic intake interviews. Jeannette Black, R.N., and I ran group B as part of our private practices. We depended on both the community mental health system and on private psychiatrists for referrals. The group began in September 1973 and terminated in December 1974. All of the members had either been hospitalized or had spent several nights at a crisis unit. All had individual therapists. Their diagnoses ranged from schizophrenia to severe depression. (We excluded women who were actively hallucinating or delusional from group membership.)

In both settings we had trouble gathering enough referrals to begin the group and to replace patients who dropped out. This may be a general problem with group therapy. Our referral sources (most of whom were men) assured us that they thought a women's therapy group was a good idea, but we sometimes wondered if they had reservations they did not share with us.

Our intake procedure in both groups consisted of a 30-minute interview with each prospective patient in which both therapists asked her what she wanted from a women's group and what she needed to know about us in order to decide whether to make the required commitment to participate for 6 months. Since very few of these women had initially requested a women's group, they had no ready reply. Responses ranged from "I feel more comfortable with women than with men" to "I feel very uncomfortable with women, and I want to work on that." If a woman had no special desire to be in a women's group, we suggested that she

consider joining a mixed group instead. About 60% of the women we interviewed joined one of our groups.

Most of the prospective patients were quite taken aback by the thought of asking us questions about ourselves. They lacked the necessary assertiveness and saw us as authority figures whose credentials were not open to question. We found this part of the interview to be a good tool to demystify ourselves from the start and to make it clear that as consumers of mental health services, our patients had the right to "shop around."

The size of both groups fluctuated between 3 and 8 members. The patients ranged in age from 19 to 40. Most were single, divorced, or intermittently living with a man. Twelve women moved through group A and 8 through group B. The group A women were lower and middle class and most worked or went to school. All of the group B women were middle class and most did not work. None of the women in either group considered themselves feminists, although several had thought about the issues involved and were sympathetic toward them. In general, they complained of maternal deprivation and fear of their fathers. Three women in group A and 2 in group B had children.

Initial Phase

The initial phase of the groups was characterized by airing of problems, with frequent crises in group B. The women competed for the therapists' attention and help and offered each other little support. The desire to depend on the identified leader at first is strong in all groups, but it may have been heightened in our groups by the fact that the therapists were seen as maternal figures. The group members may have initially seen each other as deficient females.

It was remarkable how little overt talk there was about the lack of men in the group or about the special experience of being only with women. Instead, patients introduced male "ghosts," often talking about their husbands and lovers. As this was pointed out, they shifted the focus to themselves, their own strengths and weaknesses, and their bonds to each other. According to several observers, the initial phase seemed to be briefer for both these groups than for mixed groups.

Middle Phase

Both groups evolved into a well-functioning support system in the middle phase. This happened spontaneously in group A; it was actively encouraged in group B. The women carpooled to and from meetings, shared babysitting arrangements, and often had a meal together after the sessions. When a group member had a crisis, the others would support her with visits and phone calls during the week. There were parties and outings that the therapists did not attend. In group A, one member got a new job and bequeathed her old one to another member. In group B, one member was hesitant to return to school and the others escorted her to registration.

Group time was often used to explore problems in giving, receiving, and asking for help. Disagreements and conflicts among group members, as well as hard-to-express feelings of warmth and tenderness, were often worked through. The women learned to trust and care for each other and to respect their ability to offer friendship.

During this middle phase, group members tended to see one of the therapists as "male"—cold, nongiving, authoritarian, and punitive—and the other as "female"—warm, nurturing, available, and forgiving. Usually, I received the "father" transference, probably because I have an M.D., a higher status degree than an M.S.W. or an R.N. My personal style was also more nondirective that either of my cotherapists'. Thus the patients tended to divide their transference according to prevailing male and female stereotypes, despite the reality of an all-female group.

Several members reported that the men close to them (including some male therapists) seemed to feel threatened by their participation in an all-female group and even made it difficult for them to attend meetings. These women seemed to be feeling a conflict between their role as wife or girlfriend and their wish to be more self-assertive, with the group representing the latter possibility.

The members became extremely dependent on the group. They often said it was the most important event of their week, and many made financial sacrifices to attend. It would be inaccurate to say that these were entirely transference feelings, since the group was in reality fulfilling some of the members' needs. Of course, it could not fulfill all of their needs, and several members of both groups manifested extreme rage and disappointment at times. The rage tended to focus on the therapists, who had a policy of not offering support outside of the meetings. In group A, 2 of the women were used to expressing anger and taught this skill to the others. In group B, rage was an unaccustomed and explosive phenomenon. Some of the women in both groups were able to work through their rage successfully and move on to new levels of autonomy. In each group there were 2 or 3 members who ventilated their rage and then fled the group. In some cases, women developed a sudden new dependence on a man when they were too frightened by dependence on the group. Unfortunately, the women in group B were unable to cope with the frequent losses and the group eventually terminated because of attrition.

This middle phase was thus notable for dropouts, for active transference, and for increasing intimacy among the women who remained. The frequent losses gave the remaining group members an opportunity to practice mourning and to learn the realistic limits of their responsibility to other people.

Final Phase

In the late or "ripe" phase both groups were quite small (3 to 5 members). They dealt extensively with being female, covering such issues as sexuality, mothering, sexism, and dating patterns. Most of them

showed increased self-esteem, and several began planning new occupational or educational endeavors. Some of their ambivalent bonds with men dissolved; others matured and deepened. They discovered that masturbation was helpful, and several became orgasmic for the first time. The women with children seemed more at ease with mothering and reported improved relations with their children.

In group A, the remaining women thought of each other as sisters. In group B, where flight overwhelmed intimacy, the 3 members who remained were not very close outside the group. In both groups, however, the therapists were demystified by this time. We were no longer seen as omnipotent and as withholding love but rather as professional women with interesting lives who could serve as role models in some ways. The patients also began to see their own parents more realistically and to be less afraid of their fathers.

Group A terminated when I moved to another city 2 years ago. The members have kept in touch with each other and with me since then and have helped each other through hard times, even though they now live hundreds of miles apart. One of them died of leukemia; the others visited her frequently and gave her husband support after the funeral. The members of group B have not kept in touch with me.

DISCUSSION

The groups I have described were similar in many ways to mixed therapy groups. The patients worked on the same types of issues that patients in mixed groups work on, and the therapists used the same skills and techniques that other group therapists use. However, these two groups had certain inherent problems that mixed groups do not have. First, there was no heterosexual bonding to hold members in the group when they wanted to flee. I can only surmise from the tendency of group members to find a new boyfriend when they quit the group that the presence of men might have prevented some dropouts. Second, the overwhelming dependence on the group that most of the patients developed might have been intensified by the equation our culture makes between "female" and "nurturing," as well as by the therapists' policy of fostering a support system. In mixed groups, dependency may be mitigated by the need to maintain sexual distance among the members.

Women's therapy groups also have special strengths to recommend them. There is less covert sexism and it can be more easily confronted, and the group can concentrate on issues relevant to women, particularly sexism. In group B, the therapists shared their perceptions of sexism, and gave advice on how to fight it. Such intrapsychic wounds of sexism as fear of assertiveness, the tendency to define oneself as wife, mother, girlfriend, or sex object rather than as a person, masochism and deference to men, hatred of other women and concomitant self-hatred, and repression of one's ability to think can be put in context, shared in the group, and healed. Group members feel safer and more comfortable than they would in mixed groups. Because they tend to let themselves depend so heavily on the group, they have the opportunity to work through issues of rage about not receiving enough nurturance and to learn true autonomy in this setting. The women experience women leaders functioning in authority roles, which teaches them that women can have power and deserve respect and provides them with role models. There are no sex-role divisions of labor, despite the tendency of the members to perceive such divisions. Both therapists are women, and both carry out nurturing and reality-testing functions, teaching that women are strong and complete. There is also much less need for inhibitory incest taboos. True intimacy among group members can develop more readily than in a mixed group, and there is less need for prohibitions on outside contact; thus a true support system can form. Such a support system is extremely valuable to its members, whose lives are often anomic and lonely. Moreover, the bonds among members can be scrutinized clinically in the group meetings, and the therapists can help the patients learn better skills of sharing and caring.

All-female therapy groups can be very helpful to women who seek psychiatric assistance. They do many of the things that consciousness-raising groups do, teaching women to respect other women and themselves, and providing an opportunity to identify common problems. At the same time, women in therapy groups can avail themselves of the skilled help of professional therapists.

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Journal Policy on Multiple Authorship

It has come to our attention that there has been some misunderstanding regarding the *Journal*'s policy on polyauthorship. The number of authors listed on a paper is not a factor in the review and acceptance/rejection procedure. However, once a decision has been made to publish a paper, we do apply *Journal* policy regarding the number of authors in the by-line: only principal researchers and writers of articles are listed in the by-line; collaborators who have provided technical and other assistance are acknowledged in a footnote. In papers that list more than 4 people as principal authors, we query the first author to verify that those individuals were substantial contributors to the research or ideas reported. Since certain types of psychiatric research may require multiple workers, we will of course include all of the names in these cases.

Experiences of Psychiatry Board Exam Casualties: A Survey Report

BY MARTIN R. LIPP, M.D.

The author collected completed questionnaires from 52 psychiatrists who had failed to pass the examinations of the American Board of Psychiatry and Neurology (ABPN) on their first try. He quotes from the responses of 6 of these psychiatrists, showing the profound emotional impact of their failure. He recommends that the ABPN explain its low pass rate, supply feedback on the exam results, provide the specific criteria by which competence is judged, allow candidates to take the written exam on a trial basis, and drop the oral exams if their reliability cannot be documented. He also recommends that the profession of psychiatry take responsibility for the certification procedure.

FOR THOSE WHO PASS the American Board of Psychiatry and Neurology (ABPN) examinations on their first try, certification is probably not a major event in life. The certificate gets framed and hung on the wall or placed in a file or safety deposit box with the dozens of other pieces of paper that chronicle our lives and our professional achievements. The ordeal of studying gradually fades and may even assume a certain luster in our memories along with other battles well fought.

However, for those who do not pass the examinations on the first attempt (or, for some, ever), the experience assumes very different proportions. The intent of this paper is to record and discuss the subjective experiences of colleagues who have become "Board exam casualties."

The statements cited here have been extracted from the returned questionnaires of psychiatrists answering advertisements in *Psychiatric News* and the *Northern California Psychiatric Society Newsletter*, as well as those of colleagues contacted personally and those who responded at the 1975 annual meeting of APA. The questionnaire asked the respondent to detail his or her subjective experiences as a Board exam casualty. A total of 52 completed questionnaires was collected.

I make no pretense that this sample is representative of the entire population of Board exam casualties or that the results are in any way statistically significant. This report's value depends on the heuristic impact of fragments of personal testimony on readers who have shared or will share the Board exam experience—and on our profession as a whole.

The 52 respondents ranged in age from 32 to 65; they had completed their residencies in years ranging from 1950 to 1975. All but 4 of the 52 respondents stated that English was their native language. Their experiences with the exam varied—from some who had taken their written exams once, failed them, and decided not to try again, to 1 psychiatrist who took various parts of the exam six times before completing the whole exam successfully. Twenty-three of the 52 respondents were certified by the ABPN at the time these data were gathered.

The following quotations from the answers of 6 of the respondents were chosen to illustrate the range of subjective experiences and because these respondents related their perspectives in a particularly articulate fashion. The remarks are essentially verbatim, although abbreviated, with stylistic changes for readability.

QUOTATIONS FROM RESPONSES

Colleague 1 (Age 35)

The exams were a significant professional landmark for me because I could not remain on the faculty of the university where I teach without Board exams and would not be able to assume the administrative position which I sought. I studied an average of six hours per week for six months preceding the written exams, as well as studying for about ten days prior to the writtens. For the oral exams [1974], I put myself through a series of mock oral examinations in both psychiatry and neurology.

When I learned I had conditioned psychiatry, I responded with shock, panic, severe embarrassment—wanted to get away—called a friend of many years and spent an hour talking with him in the park. Was quite depressed, felt stupid, worthless, a failure, fell asleep obsessing over each examination, wondering what I did wrong, woke up early in the morning thinking of the exam. No therapy—wished afterwards I had. Had a hiatus in work and writing, seemed to mark time, drain on all my energies, withdrew from colleagues, and

Revised version of a paper presented at a forum at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

At the time this paper was written, Dr. Lipp was Chief, Psychiatry Consultation Service, San Francisco Veterans Administration Hospital, Calif. He is now with the Permanente Medical Group at Kaiser Hospital, Hayward, Calif., and in private practice in San Francisco, Calif. Address reprint requests to him at P.O. Box 26393, San Francisco, Calif. 94126.

avoided teaching activities. More irritable with family, lost sense of humor. Did not want to be in social setting with colleagues. Considered leaving psychiatry and taking up general practice or neurology and just getting away.

This was my first real failure, and I felt helpless as a consequence. The experience of failure was lonely and isolating. Able to tell only three colleagues and my wife. Responded to the experience by requesting information from the Board and received a nonspecific reply which was not helpful. Felt at a loss to know what the next examiner would want, complained about the subjectivity of the experience, and was bitter because the comments returned by the ABPN in no way reflected three years of evaluation by supervisors in residency training. Ultimately I retook the oral exam in 1975 successfully and am now a Board-certified psychiatrist.

Colleague 2 (Age 41)

I put a great deal of time, money, effort, and ego into the taking of my exams. When I learned that I conditioned my psychiatry orals [1972], I was shocked and stunned. Not clinically depressed or suicidal. It was my first such failure. I felt it was a fluke, something completely out of line and inappropriate, as much the ABPN's fault as mine. No effect upon my work, social life, family life, except for the necessity to do more studying. My only previous failure had been a divorce with which I coped by going into a seven-and-a-half-month-long clinical depression, which required therapy. Failing Boards scores 10 on a scale of 100. The divorce scores 90.

Another response to my failure was to write the Board. The ABPN was very gracious in indicating to me the sources of my poor showing. This was the key, incidentally, to my passing the next time around. My exam performances both times were at a lower level than my usual performance. Certification is like a Phi Beta Kappa key, not everyone who has one *really* deserves it. Many who do not have one are of equal or higher caliber than those who do. Yet the Board certification has opened doors to me otherwise unavailable.

Colleague 3 (Age 53)

I put a great deal into Boards. Studied 20 hours per week for one year. Took a mail-order course, went through a neurology review book, and so forth. Forsook nearly all social and family life; the experience prolonged my training analysis about one year. Upon receiving notification of failure [1971], was disappointed, very angry, and bitter. Mildly and transiently depressed, but my training analyst helped me work that through.

There was little effect on my private practice, but I lost about $280 per month at my part-time government job. At times, when put on the spot, I feel like a second-class citizen: the well-trained child psychiatrist I am, forever excluded from certification in my field because of my lack of certification in the adult Boards. Apologetic explanations are often necessary for why I am not Board certified.

I never really failed at anything important in the past. In this case, at my age, I was not willing to try again—wouldn't know where to start. A number of questions are unanswered. Did I fail by a little or a lot? Where was I weak, neurology or psychiatry? Did *I* goof or was the test unfair? I did not get any feedback about these questions.

I've been able to talk about my experience with only a very few select colleagues. Although I do not believe my performance on the exam was representative of my performance generally, I blame myself to a large degree. I am not really sharp on that type of test. My clinical style is more slow and deliberate. In some ways I believe I have *too much* knowledge, and I am aware of too many "ifs" to do well on a multiple-choice exam. In that sense the ABPN is very much at fault.

There is one additional factor you should know about. During my prolonged studying for the exams and the prolonged emotional tension associated with it, I neglected many physical symptoms because I regarded them as part of the stress I was under. Because of that neglect and the additive effect of stress, I ended up requiring major abdominal surgery. I have no intention of ever subjecting myself to that ordeal again.

Colleague 4 (Age 43)

In my entire life this was my only failure, although in the long run I may be a better person for it. I put a great deal of ego involvement, time, and effort into studying and taking the exams [1962]. Afterwards, I was mostly very angry, with intermittent feelings of depression and shame. I had thought that I did well in neurology and psychiatry. After being informed that I failed both parts of the exam, I was shocked. I contacted my examiners in neurology personally, all of whom told me that I had done satisfactorily and that they had passed me. My attempts to find out how I could have failed both parts were in vain. I could never find out why I failed. My best *guess* is that somehow I rubbed one of the psychiatric examiners wrong—so badly that I was therefore flunked on everything.

At the time I took the exam, I felt that my level of training and my skills were excellent. I had gotten recognition as an excellent resident at my highly regarded residency, been director of a military mental hygiene clinic, written several clinical papers, taught psychiatry to medical students, and been a successful clinician in private practice. Since failing the Boards, I've gone on to graduate from a nearby psychoanalytic institute, been actively involved in training at a prestigious local neuropsychiatric institute, been consultant to many agencies, presented papers at various APA meetings, and so forth. We seem to be entering an era of medicine where it would be "good" to have one's Boards. I would even be willing to restudy the psychiatric part, but it's just too huge and unpleasant an undertaking to have to restudy neurology.

Colleague 5 (Age 65)

I put a considerable amount of energy into preparing for the Boards [1959], and I did not expect any particular trouble in passing. Afterwards, I felt disappointed and disillusioned. I got the feeling that the Board was not interested in helping me pass. My residency training, all in the military, did not stress or prepare me for Boards. I felt that a good overall knowledge of psychiatry would be sufficient, but it proved not to be. I had had other experiences with failure in the past and believe them to be my fault; in this instance, I feel the Board itself was unfair.

What little feedback I did get was essentially that I was military trained, that "the Board had to" fail its quota, and that people like me had to expect to fail at least for a time or two. On both occasions when I took the exams, first in New Orleans and then again in Chicago, I felt the examiners (mostly quite young) were openly antimilitary, and I was a captain on active duty when I took the Boards. I have been a physician for 38 years and in psychiatry since 1949. I have been chief

of psychiatry in three hospitals and chief psychiatrist at a state prison. I believe that my experience and accomplishments deserve some recognition from my profession. I have been a member of the APA since 1951, and as far as I can see, among "the brethren" I am a nobody.

Colleague 6 (Age-51)

In studying for the orals, I spent six months of daily intensive study, as well as one month of full-time six-hours-per-day studying. Much ego involvement, family interest, and investment of money. When I heard of my "failure," I responded with surprise, disbelief, anger, frustration, indignation. The exam has had no effect on my practice; I was appointed superintendent of a large state psychiatric hospital after taking and failing my first oral exams. My family was disappointed. Colleagues were supportive.

The problem with Board examinations lies in examiners' not attempting to test you on *what you know* but on what they know best and their structuring the exam on a professor-student level rather than as colleagues. The ABPN is too rigid and is controlled by men who are too far removed from real psychiatry to know the difference.

In October 1974, test conditions were awful in New York, crowded and hurried, with many long journeys across town by taxi. Although I am a superintendent of a state hospital, I was given two adolescents during the psychiatry hours and never asked about administration, forensic psychiatry (which I know well), or community psychiatry. One patient was a 15-year-old girl with anorexia nervosa, and the examiner was a chairman and professor of child psychiatry; she was the first such case I had seen in 21 years of medicine. You can imagine the odds. When we finished, he said, "You certainly did a marvelous interview and handled the patient well"—and then he failed me.

On reexamination, I passed psychiatry and conditioned neurology, therefore failing the exam as a whole. The neurology examiner was culturally and socially not atuned to me, and he dominated the other examiner, who seemed more on my wavelength. It was extremely hard for me to appear to be a junior medical student when I have been a person in authority for so long, although I tried my best after New York. Although I frequently read EEGs, the neurologist marked me up for not knowing EEGs, which I do not remember a question about.

DISCUSSION

This discussion will be brief so as not to detract from the impact of the first-person narratives and because it is hard to know what to say that has not been articulately and sometimes poignantly illustrated above. However, I will try to emphasize some of the more widely held or recurring themes.

First, it is apparent that being a Board exam casualty has a profound emotional impact on those who share the experience. All of the respondents experienced depression, some of considerable magnitude, although none reported suicidal ideation or abuse of alcohol or drugs. Overwhelmingly it was a lonely experience, to be shared openly with very few others. For many, shame, bitterness, anger, bewilderment, helplessness, and frustration were important themes that dominated their lives for months or years after learning their exam results. The personal and professional cost of these often painful emotional states is impossible to assess, but it would be fatuous to assume that the cost is negligible. Several casualties blamed their Board experience for major repercussions in their private life. Although the merits of any given case are certainly debatable, the painfulness of the experience for many can hardly be argued.

Second, the absence of spontaneously given feedback was a source of frustration for practically all of the respondents. The recently developed Board policy of giving feedback on oral exams for those who request it was clearly regarded as a positive step, although many of the respondents took issue with the specifics of the criticism rendered. Others were aggrieved that the availability of feedback was officially a secret and not mentioned explicitly in the ABPN brochures. To deprive a duly trained and experienced psychiatrist of confirmation of his or her professional identity without saying why seems unfair. No other single aspect of the examination procedure was the source of so much resentment.

Third, there was widespread criticism of logistic hurdles encountered in the exams. These both increased the cost of the experience for the candidate and at the same time made it more traumatic. Again, it is impossible to say whether the stories told by the respondents are apocryphal, whether these problems contributed to the casualties' exam performances, or even whether logistical problems occur more frequently with casualties than with noncasualties. However, anecdotes abound: candidates in one location told of having an unexplained 70-minute delay before beginning the written exam in 1972; exam sites and schedules were changed at the last minute one year; a proctor in a written exam gave a running discourse about local restaurants in 1975; one year a candidate from Philadelphia was assigned to take written exams in Washington, D.C., at the same time that a colleague from Washington journeyed to Philadelphia to take the exam; both candidates and examiners were flown from the same California city to go through the oral exam ritual together in New York City in 1974.

Fourth, a concern relating to the ambiguity of the criteria by which a candidate's performance is judged was often noted. The weight of this criticism fell on the oral exam, but the written exams were not immune. One example in a written exam was a question that could be answered one way if the respondent used Bleuler's criteria for schizophrenia but another way if Schneiderian criteria were used. The respondent who told about this question decided that the more widely used Bleulerian criteria were probably called for, but he felt that this question was typical of an exam that sought simple answers, penalized sophistication of knowledge, and lacked clarity concerning which authorities were being used as a standard for what is "correct."

The comments were particularly biting on the topic

of the oral exams, in which subjectivity clearly can play a larger role. Candidates frequently complained of being judged according to an unknown yardstick by examiners whose unknown biases, theoretical preferences, and emotional states dominated the exam's results at the expense of any objective assessment of candidates' expertise in representative clinical settings. Reference was often made to the American Board of Internal Medicine, which abandoned oral exams after finding that exam scores correlated more with the identity of the examiner than with the identity of the candidate. One respondent commented, "At a time [1973] when the American Psychiatric Association had just formed the Task Force To Define Mental Illness and What Is a Psychiatrist, it seemed ludicrous to ask candidates to answer oral exam questions in the absence of explicitly stated criteria on which we will be judged."

Fifth, a number of older practitioners argued persuasively and poignantly that the examination procedure discriminates against psychiatrists who have been out of school for many years. Taking multiple-choice exams, they pointed out, is an acquired skill that they never had an opportunity to develop because it was not in vogue when they were in training. Even if they had had such experience, it was so far in the past that their talents in taking this kind of exam had long since atrophied. The oral exams are also more difficult for older practitioners, both because they have had less recent experience with question-and-answer repartee and because being scrutinized by young examiners with a fourth of their clinical experience has its humiliating aspects.

Sixth, many casualties believed that the responsibility for their failures remained an unresolved issue. Some blamed themselves, others blamed their residencies, many blamed the ABPN. Most of the respondents implicated more than one factor. No one specifically blamed the profession of psychiatry or the quality of psychiatric knowledge per se, although many felt that they had been unfairly penalized by the absence of agreed upon, reliable criteria for judging professional competence. Several felt that residency training programs should share the pain when their graduates do poorly on a Board exam. Although the thinking here was not clearly stated, two factors seem to be involved: 1) to punish and restrict programs that consistently fail to assist their graduates in the process of certification and 2) to give the residencies a greater impetus to confront and help modify a Board that is generally seen as unresponsive to input by individuals.

A final issue relates to the reluctance of the respondents to undergo the same ordeal again. Even those who were preparing for a repeat examination clearly were doing so with reluctance and even dread. With the prospect that mandatory recertification is becoming an increasingly likely reality, a number of respondents voiced the hope that recertification will be applied equally to all. One respondent stated, "Who will examine the examiners? If they have to go through the same procedure as I do on a regular basis, at least that will tend to reduce the arrogance and insensitivity which I personally encountered. No grandfather clauses, please!" In the absence of such safeguards, recertification procedures will be met with profound cynicism, as a rote performance demanded by the haves for the purpose of excluding the have-nots.

RECOMMENDATIONS

The suffering inherent in our current system of Board examination demands that something be done. Almost two-thirds of all psychiatrists (our noncertified brethren) are disenfranchised by current practices. I am embarrassed and ashamed that I can advance the following suggestions only now that I have successfully completed this official rite of passage:

1. The ABPN must be held accountable for the extraordinarily low rate of passes that characterizes its procedures. The pass rate for psychiatrists on part I of the exam (the written portion) was 63% in April 1974. Recent pass rates for graduates of U.S. medical schools for part II (the oral portion) ranged from 58.4% (Los Angeles, April 1974) to 68.7% (Atlanta, February 1975), resulting in a successful completion rate for both parts of the exam ranging from 36.8% to 43.3% (1). Because these figures place our certification procedures at the most stringent extreme of all specialty boards (2), the ABPN must be asked to explain and justify their punitive and arbitrary standards.

2. Feedback must be included as an integral part of the examination procedure. All candidates, successful or not, should be told as a matter of course where their perceived strengths and weaknesses lie. Further, this information should automatically be transmitted to each candidate's residency training program. In addition, success-failure ratios for all residency programs should be published in the *American Journal of Psychiatry* along with the published names of successful candidates.

3. The ABPN must provide specific criteria to the candidates, advising them precisely by what standards they will be judged. If specific, measurable criteria and scales of professional competency exist, there is no reason for keeping them a secret. If they do not exist, we must recognize the caprice and subjective bias with which professional competence can currently be determined and therefore grant certification automatically to anyone who completes an approved residency.

4. All candidates should be allowed to take the written exams on a trial basis without penalty and without exhorbitant cost. This certainly should be the case for older candidates who have only remote experience with multiple-choice exams; in fairness, however, everyone should have this opportunity.

5. Unless the oral exams can clearly be demonstrated to be free of the subjective bias with which they are so frequently charged, they should be dropped from the examination format. At present, a

good case can be made that the oral exams provide only a facade of examining clinical competence.

6. Our profession as a whole must take responsibility for the certification procedure that helps to define us all. Although scapegoating the ABPN often seems an attractive alternative, it is sheer sophistry to do so. The task belongs to all of us. Is not the practice of psychiatry difficult enough without adding this additional burden to the role we have chosen for ourselves? In short, what are we doing to ourselves and to one another?

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Change in 1976 Annual Meeting Site

The APA Executive Committee decided to move the May 10-14, 1976 APA Annual Meeting from Atlantic City to Miami, Florida, after hearing the APA Meetings Management Department report that it had ascertained that a major Atlantic City hotel had inadvertently committed several hundred rooms to another organization during the week the APA meeting was scheduled. This would have left APA with the unacceptable alternative of using a myriad of scattered small hotels in the Atlantic City area. It has been confirmed that Miami Beach can accommodate the meeting.

Further arrangements and developments will be reported in future issues of *Psychiatric News*.

Effect of Drug Ritual Changes on Schizophrenic Patients

BY RODNEY W. BURGOYNE, M.D.

The author evaluated the charts of 115 chronic schizophrenic patients who regularly attended a follow-up group to determine whether changes in long-standing medication regimens produced an increased dropout rate, decreased attendance, or psychotic decompensation. He found that although there were nonsignificant trends showing a relationship between medication change and group attendance for individual patients, there were no significant differences between group attendance and change or no change in medication routine for the group of patients as a whole. He concludes that necessary changes in medication routines for chronic schizophrenic patients are safe.

THIS PAPER REPORTS the effect on outpatient attendance of changing the drug routines of stabilized chronic schizophrenic patients. In contrast to studies that have assessed the effect of antipsychotic medication, this study measures the effect of factors surrounding the use of maintenance drugs per se. It was designed to determine whether changes in long-standing medication regimens produce an increased dropout rate, decreased attendance, or psychotic decompensation in previously well-stabilized patients who were regular attenders of follow-up groups.

BACKGROUND

The establishment of community mental health centers in the 1960s promoted the use of maintenance antipsychotic medication, the less routine use of long-term hospitalization, and an increased interest in post-hospitalization outpatient aftercare for schizophrenic patients. Outpatient psychotherapy was first modeled on modifications of psychoanalytic concepts. Special "Tuesday afternoon groups" (1) or "Wednesday afternoon clinics" (2) denoted that a nonintrusive, supportive, nonthreatening form of psychotherapy should be used. Research studies noted that group therapy was more effective than individual therapy (3, 4) and that aftercare coupled with short hospitalization (5) or day care (6) was as good as or better than prolonged hospitalization.

Although some think that an adequate assessment of the effect of using or discontinuing antipsychotic drugs, particularly when they are given in low doses, has not yet been made (7, 8), there is general consensus that maintenance antipsychotic medication is useful in the treatment of many, if not most, schizophrenic patients.

Similarly, although a proper assessment of the effect of psychotherapy on schizophrenic patients has yet to be made (9), it remains an almost ubiquitous clinical impression that the resocializing, supportive "active" psychotherapies are helpful. Many different psychotherapy techniques are used with schizophrenic patients; there is little agreement on which is most helpful or why it may be so. Therapeutic interventions may hinder the recovery of some patients or even drive them away from treatment. Sometimes patients are considered improved after moving from one setting to another (10). Simply talking with patients about their behavior since they were last seen correlates with a decrease in rehospitalization rates (11). Perhaps aftercare groups are effective because they provide modeling of how others have coped with their problems (12). The effect of any given approach on chronic schizophrenic patients as a group remains questionable.

In spite of the importance of social and supportive therapy for these patients, the major initial factor in successful treatment is antipsychotic medication. Patients who do not come for outpatient aftercare may discontinue their medication. They are also deprived of social therapy at a time when it has been demonstrated to do the most good—after 18 months (13–15). In an effort to prevent unnecessary stress, it is widespread practice for therapists to allow questionable drug regimens to continue unaltered for months, thereby avoiding conflict with patients over their medication.

The Follow-Up Clinic

At the Los Angeles County-University of Southern California (LAC-USC) Medical Center, years of experience with large numbers of patients who require continuing care have resulted in a program similar to that recently described by Isenberg and associates (16). At the LAC-USC Center, psychotropic medication is considered primary, but social and supportive psychotherapy is stressed.

Presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Burgoyne is Assistant Professor of Psychiatry, University of Southern California School of Medicine, and Associate Director, Adult Psychiatric Outpatient Clinic, Los Angeles County-University of Southern California Medical Center, 1237 North Mission Rd., Los Angeles, Calif. 90033.

Twenty groups meet for 45 minutes once a week. Five groups meet at one time in the follow-up clinic. Each group may have 40 to 60 patients enrolled as members, but the weekly attendance averages about 18 patients. Some patients come each week, others every other week, and some less often. Many of the patients in these groups have been attending for years. For some of them the clinic has become a family substitute, and the patients have formed an allegiance to the institution rather than to specific doctors. Before the group meets, many of the patients gain gratifying input through socialization over coffee in the large waiting area (17). Afterwards, medications are refilled for each patient individually.

Because it is less effective for chronic schizophrenic patients (18), individual therapy is discouraged, but it is used when necessary to interrupt a crisis. Movement therapy (19–21), vocational rehabilitation (22), and help with social welfare agencies are readily available to appropriately selected patients (23). Most group therapy work is done through metaphor and latent language communication (24). Restrictive solutions (25) to group conflicts are used more often than other solutions. Interaction is almost always supportive and goal directed and only occasionally intrusive (26). To avoid augmenting any disappointment the patients may have encountered with outside-world relationships, interaction within the group is kept empathetic but firmly reality oriented (27).

One early goal of the follow-up clinic is to enable the patient to recognize his narcissistic and anaclitic needs (28) and to focus on abilities that are either already in the patient's behavioral repertoire or that can be developed to appropriately meet these needs. The difficulty this task imposes on the usual schizophrenic patient is recognized and stressed (29), but hope is fostered and successes are actively acknowledged (30). Severe symptoms, such as hallucinations, are interpreted as evidence of the need for medication and as a communication of the patient's dependency needs, but they are not solicitously emphasized (31).

Remaining out of the hospital is recognized as a significant success. As others have also noted (14, 32), there are surprisingly few patients participating in these groups who are hospitalized.

A few patients come to the clinic only for medication and do not attend the group session. These patients give various excuses, but apparently they do not attend because either they are more paranoid and isolated or they are not limited in social contacts and feel they do not need the group (33).

When therapist replacements are necessary, they are made relatively smoothly and are structured to facilitate allegiance to the group and to the institution as a whole rather than to the therapist.

Clinical Observations on Attendance

When a therapist of one of these groups becomes ambitious and presses for introspection or acknowledgment of feelings or sets higher goals for social and productive functioning, the anxiety level of the group rises and attendance dwindles. Anxiety is seldom acknowledged by the patients as a reason for poor attendance. This is usual for schizophrenic patients, who have great anxiety and have to withdraw from the activities of life to keep it under control while presenting a blunted exterior to the world (34).

Changing drug routines appears to place significant stress on these patients. Sometimes they are not allowed to maintain the social safety they need by isolating themselves when interaction becomes focused on a change in their long untouched medications. This change requires increased monitoring and attention. Other times this change may provoke open disagreement. Patients who have maintained the same medication pattern for years and have not suffered acute exacerbation of their psychoses are particularly likely to resist any change.

The risk of tardive dyskinesia and other side effects of long-term antipsychotic maintenance drugs necessitates periodic medication review, drug holidays, and lowered doses. Therapist anxiety sometimes leads to unnecessarily extreme or poorly timed medication manipulations. This is particularly true if a patient has been on a mixture of several drugs. Occasionally the former drug routine is denigrated when changes are administered, thus undermining the patient's trust in the clinic and in his doctors. Because the expectation of help and a feeling of success in gaining it are necessary for treatment to continue (30), poor attendance after a change in medication seems understandable.

Many patients strongly believe that their medication regimen keeps them compensated. Evidence supports their belief that they tolerate more stress when receiving antipsychotic medication (35), although at times their allegiance to the drugs appears magical. Unfortunately, these patients may appear merely overly dependent on their medication, a view that allows the doctor to abruptly change the prescription. Patients who challenge such changes by an insecure doctor sometimes cause him to defensively assert his will and further undermine rapport.

Because of the long washout period of most antipsychotic drugs, even discontinuance produces little pharmacological effect for a while. In properly medicated, stabilized patients, changes in routine have significant effects (36). It is often assumed that major medication manipulations will therefore produce significant unwanted stress, possibly leading to diminished or ceased attendance at aftercare programs altogether.

METHOD

Patient Selection

Patients included in this study had a firm diagnosis of schizophrenia, had shown regular (twice a month or more) attendance at a follow-up group for the six months preceding the study, were receiving major antipsychotic drugs, and had been receiving a stable and

unchanged dose for four months before the study.

In an effort to refine the diagnosis of schizophrenia, the clinic therapists made a judgment based on several months of experience with the patient as to the presence of first-rank Schneiderian symptoms (37). Patients who did not currently exhibit these symptoms were diagnosed as schizophrenic if they had recently exhibited them and if they had a history indicative of chronic schizophrenia. Of over 600 patients who attended the follow-up clinic, 115 met these criteria and attended the clinic during the five-month study period.

Of the 115 patients, 59 were men and 56 were women. Their ages ranged between 19 and 62. Their median age was 38, and most of the patients fell into the 28–52-year-old range. Sixty-one patients (53%) were white, 35 (30%) were black, and 19 (17%) were Mexican-American. Fifty-eight were married or lived with someone considered to be supportive, and 57 claimed to live totally alone.

Measurement of Variables

Patient attendance over the five study months was divided into those whose attendance was unaltered (continued two times or more a month), was diminished but regular (averaged at least one time a month), was interrupted (for at least one month), or ceased.

The medication regimen changes that occurred during the five study months were deemed necessary in the clinical judgment of therapists blind to the study. The medication regimens are divided into the categories of no change, minor change, and major change. A major change was defined as a substantial change in dose of a major antipsychotic drug and/or of any concomitantly given tricyclic antidepressant. A minor change was defined as any other medication change. More specific criteria are as follows:

Major medication change criteria. 1) Substantial increase or decrease of dose of an antipsychotic drug (plus or minus 5 mg of trifluoperazine or thiothixene or plus or minus 50 mg of chlorpromazine, thioridazine, or their equivalent); 2) addition to, discontinuance of, or substantial change in dose of concomitantly given tricyclic antidepressant (50 mg of imipramine or its equivalent).

Minor medication change criteria. 1) Addition to, discontinuance of, or change in dose of hypnotics, antiparkinsonian drugs, or benzodiazepine drugs; 2) changes in antipsychotic drugs within the same group to another of approximately the same potency dose or involving time of day or division of dose; 3) any other medication addition or change deemed of possible importance in my judgment. (I reviewed the changes because I had not been the therapist for any of these patients.)

RESULTS

The charts of the 115 patients were reviewed for five months. The attendance patterns were unchanged in 61 patients (53%), regular but diminished in 23 patients (20%), interrupted for one month or more in 26 patients (23%), and totally discontinued (not seen at all in the last two months of the study period) in 5 patients (4%).

Sixty-one of the patients (53%) had no medication changes during the study period. Seventeen patients (15%) had minor changes in medication, and 37 (32%)

TABLE 1
Analysis of Effects of Medication Changes on Attendance Patterns of 115 Schizophrenic Patients

Item	No Medication Change		Medication Change							
			Minor Change		Major Change		Total			
	N	%	N	%	N	%	N	%	χ^2	p Value
Comparison of all medication categories versus attendance										
Attendance interrupted or ceased	13	21	4	23	14	38	—	—	—	—
Attendance diminished	13	21	3	18	7	19	—	—	—	—
No change in attendance	35	58	10	59	16	43	—	—	—	—
Analysis	—	—	—	—	—	—	—	—	3.53	p>.10
Comparison of no medication change and any medication change versus attendance										
Any change in attendance	26	43	—	—	—	—	28	52	—	—
No change in attendance	35	57	—	—	—	—	26	48	—	—
Analysis	—	—	—	—	—	—	—	—	0.979	p>.10
Comparison of no medication change and major medication change versus attendance										
Attendance interrupted or ceased	13	27	—	—	14	47	—	—	—	—
No change in attendance	35	73	—	—	16	53	—	—	—	—
Analysis	—	—	—	—	—	—	—	—	3.23	p>.10

TABLE 2

Comparison of Demographic Characteristics and Type of Medication Versus Attendance Patterns of 115 Schizophrenic Patients After Change in Medication Schedule

Item	Number of Patients, by Attendance Pattern		
	No Change	Diminished but Regular	Interrupted or Ceased
Sex			
Male (N=59)	33	10	16
Female (N=56)	28	13	15
Living arrangements			
Living with supportive person (N=58)	30	13	15
Not living with supportive person (N=57)	31	10	16
Type of medication			
High-dose antipsychotic (N=57)	28	12	17
Low-dose antipsychotic (N=58)	33	11	14
Concomitant tricyclic (N=15)	9	2	4
Concomitant anxiolytic (N=24)	11	7	6
Ethnic group*			
White (N=61)	39	11	11
Black (N=35)	15	7	13
Mexican-American (N=19)	7	5	7

* There was a significant difference between the white patients and the other patients in attendance patterns ($\chi^2=6.365$, $p<.05$).

had major changes. Of the 54 patients whose medication was changed, two-thirds had a major change according to the study criteria.

No significant differences were found between attendance patterns and changes in medication (see table 1). There were nonsignificant trends suggesting that a change in a long-established medication regimen might be related to attendance in some specific patients, but these trends were not significant for the groups as a whole (see table 2). No matter what comparison was made, attendance and medication change were independent of one another. There was no evidence that medication regimen manipulation has, on the average, any significant effect on the follow-up clinic attendance of stabilized chronic schizophrenic patients.

Table 2 lists some other variables thought to possibly relate to attendance patterns. Analysis revealed no significant differences between gender, living alone, or dose level of antipsychotic medication and attendance patterns.

When only the extreme groups (no change in attendance and interrupted or ceased attendance) were compared with age (above the median and below the median age), there was a strong trend for younger patients to change attendance more often, but the difference was not statistically significant ($\chi^2=3.374$, $p<.10$). Clinical experience confirms that younger patients have more difficulty identifying themselves as members of groups of chronic mental patients. Perhaps that is fortunate for them.

Patients of minority ethnic groups changed attendance patterns significantly more often than white patients (see table 2). This has been observed at our clinic and elsewhere (38, 39). One of the causes of this difference in attendance patterns of minority patients might be their feeling that they do not belong. One hopes this will change as the number of therapists from minority groups increase.

According to these data, changes in medication rituals do not produce a measurable alteration in attendance patterns of chronic schizophrenic patients. A trend suggests that there may be a relationship between medication change and attendance for some patients, but a recheck of charts for evidence of this relationship produced no definite examples. It was noted that even extreme disagreements over medication seldom upset an attendance pattern.

CASE REPORTS

Case 1. A 39-year-old Mexican-American housewife had been on a stable drug routine (50 mg of chlorpromazine three times a day and 100 mg at bedtime) for many months. Her physician made a change without warning her (he substituted 5 mg of thiothixene three times a day and then increased it to four times a day after four weeks). She objected to this change often; after two and a half months she demanded that her medication be changed back again. Her doctor talked her out of this, noting that he "convinced her to try one more week." He recorded no evidence of impending decompensation.

The next notation on this chart, made three weeks later, revealed that she had been in the state hospital for 14 days and was then on a third regimen (200 mg of thioridazine at bedtime and 50 mg of doxepin twice a day). This regimen was continued at the clinic with little discussion. Follow-up at one year showed her to be improved in the judgment of the therapist.

It would be highly speculative to posit that the decompensating stress for this woman was the disagreement she had over her medication. Clinically it appears possible. However, the only time she did not attend her group was during her hospitalization.

Case 2. A 33-year-old single black woman had a fairly marked ongoing disagreement over her medication and yet continued to come to her group regularly. After many months of the same regimen her doctor discontinued her medication (100 mg of thioridazine daily) with notation, "No clear evidence of psychosis."

Two weeks later she demanded her medication and was given a small dose, discontinued again later because her doctor felt it was not indicated. At six weeks she requested a new doctor. At two months she claimed to be "desperate and in great need of her medication." At three months she claimed olfactory hallucinations. At four months a neurological workup was done; the results were normal. At five months she described fantasies of people following her and demanded that the doctor tell her what the problem was and treat her for it. He began medication (10 mg of thiothixene three times a day), and her clinical course smoothed out.

This case demonstrates evidence of a substantial stress related to medication changes as well as a decompensation, possibly as a result of loss of maintenance drug; yet this patient's attendance pattern remained unchanged.

The changes in attendance patterns of these two patients appeared to be related to a variety of factors. Other charts revealed that some patients were told to come less frequently and complied. Most of the upsets noted appeared to be related to outside events that were described when the patient returned to the group.

CONCLUSIONS

Upsetting long-standing medication rituals has been shown to have little effect on the attendance of stabilized chronic schizophrenic patients as a group. Clinicians should exercise judgment in changing medications, but it certainly appears safe to do so. Patients often see medication reviews as evidence of therapist enthusiasm and interest and as cause for new hope. Therapists who make no changes in medication routines because of patient fragility should reconsider this policy to be sure they are not rationalizing their own wishes to keep their interaction with patients unmolested and unbothersome. Certainly, because of the occasionally permanent side effects of antipsychotic medications, long-standing drug regimens must be reevaluated periodically. This study shows that medication changes are tolerated by schizophrenic patients. To paraphrase Bergin's comment on psychotherapy as a whole (40), manipulation of the medication routines of chronic schizophrenic patients makes them better as well as worse, producing little change in the group when viewed as a whole.

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A Psychiatric Study of Patients Referred with a Diagnosis of Hypoglycemia

BY CHARLES V. FORD, M.D., GEORGE A. BRAY, M.D., AND RONALD S. SWERDLOFF, M.D.

Five-hour oral glucose tolerance tests (GTTs) differentiated 30 volunteer patients who considered themselves hypoglycemic into three major groups: those who had reactive hypoglycemia, those who were normal, and those who had diabetes. Clinical psychiatric evaluation and Minnesota Multiphasic Personality Inventory testing revealed that half of the 30 patients were experiencing a current psychiatric disorder, usually depression. Hysterical personality traits were also noted in many of the patients. The idea that reactive hypoglycemic patients have specific personality characteristics was not substantiated by the authors' data. They hypothesize that some patients with psychiatric illness may have their symptoms erroneously attributed to incidental GTT findings.

IN RECENT YEARS there has been considerable popular interest in "hypoglycemia." Stimulated by books that implicate hypoglycemia as the cause of such diverse diseases as asthma and alcoholism, many people have become preoccupied with glucose tolerance tests, special diets, and unproven therapeutic modalities. Popular medical books (1–3) purport that any psychological discomfort may be secondary to metabolic problems that are treatable by diet.

Opposed to the view that psychological symptoms may result from hypoglycemia is the work of some early investigators (4–6) suggesting that hypoglycemia itself may be an effect of tension and anxiety. Other writers, while not offering opinions as to etiology, have commented that hypoglycemic patients are often intense, driving, and conscientious (7), tense and emotionally labile (8), and nervous and apprehensive (9) and that they experience exacerbations of symptoms during periods of emotional tension (10). There are few systematic psychiatric descriptions of hypoglycemic patients in the literature, although some case histories have been reported (4, 5). Most communications have made use of such general terms as "emotional tension" and "strain." There is considerable cross referencing among these descriptions.

In a 1973 study, Anthony and associates (11) administered the Minnesota Multiphasic Personality Inventory (MMPI) to a number of patients who were considered hypoglycemic according to glucose tolerance tests. Their findings indicated that, regardless of the cause of the hypoglycemia, the patients had MMPI scores compatible with significant hypochondriacal and hysterical personality features. They suggested the possibility that reaction to the hypoglycemic symptoms could cause this personality pattern, although they acknowledged that a definitive statement as to causality could not be made on the basis of these data.

Because of the widespread belief that emotional distress in many people is causally related to low blood sugar, we conducted a pilot study to define the psychological status of people purported to have reactive hypoglycemia.

METHOD

We advised local practicing physicians that patients diagnosed as hypoglycemic could be hospitalized at no cost to the patient in the Clinical Study Center of Harbor General Hospital for a detailed medical workup. Although we told potential patients that an assessment of their psychological status would be made during their admission, we made no effort to recruit patients with psychiatric symptoms. Thirty people volunteered to take part in the study. They were instructed to maintain a high-carbohydrate diet for several days before admission to the hospital, where they stayed for an average of five days.

A five-hour oral glucose tolerance test (GTT) was

The authors are all with the Harbor General Hospital Campus, Torrance, University of California, Los Angeles, Calif. Dr. Ford is Head Physician, Psychiatric Liaison Service, and Associate Professor of Psychiatry; Dr. Bray is Director, Clinical Research Center, and Professor of Medicine; and Dr. Swerdloff is Chief, Division of Endocrinology, and Associate Professor of Medicine. Address reprint requests to Dr. Ford at Harbor General Hospital, Box 69, 1000 West Carson St., Torrance, Calif. 90509

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obtained on the second day of hospitalization. Venous blood samples were obtained 30 and 15 minutes before the test, at the start of the test, and at 10, 20, 30, 40, 50, 60, 75, 90, 105, 120, 135, 150, 165, 180, 210, 240, 270, and 300 minutes during the test. Blood samples were frozen, and analyses were not made until after the psychiatric and psychological opinions were obtained. Thus the psychiatric interviewer had no knowledge of the presence, type, or extent of each patient's hypoglycemic response. Serum glucose was determined by the glucose oxidase method. (Because numerous factors influence the glucose tolerance test [12], we arbitrarily used the GTT under standardized conditions to define the presence or absence of a hypoglycemic response.)

Multiple biochemical determinations of hormones and other metabolic substances involved in glucose homeostasis were also made; the results of these studies will be reported elsewhere. This paper will be concerned with the psychological evaluations of 30 patients who referred themselves to our clinical center because they thought they had hypoglycemia and the relationship of their psychological findings to their glucose tolerance curves.

The clinical sample was composed of 22 women and 8 men. The age range was 20 to 67 years, with an average of 37.6 years. All of the patients were Caucasian and of middle-class socioeconomic status. Most were employed or functioning as homemakers. Four had never married, 4 were divorced, and 6 had been married two or more times. All of the patients regarded themselves as medical patients and considered their distress to be caused by hypoglycemia. Ten patients (33%) had received some type of psychiatric treatment in the past.

The patients were seen for a nonstructured interview that lasted from one to one and a half hours. They also completed the MMPI and a symptom questionnaire that asked about symptoms commonly attributed to hypoglycemia. From these data, using standard nomenclature and diagnostic descriptions, the patient was assigned a current reactive psychiatric diagnosis and a personality diagnosis when applicable. The psychiatric and psychological data were then grouped and compared according to the patients' GTT diagnoses.

There is recognized difficulty in defining the dividing line between normal and hypoglycemic glucose tolerance curves. Different investigators may choose different nadirs on the GTT to define hypoglycemia. Consistent with values used by local practicing physicians, we arbitrarily used a nadir of below 65 mg/100 ml to define hypoglycemic. However, because controversy exists as to what constitutes a hypoglycemic level, the MMPI data were also analyzed using Spearman's rank correlation coefficient to relate the severity of the hypoglycemia to the degree of MMPI abnormality on three clinical scales (depression, hysteria, and psychasthenia). We hypothesized that by this means any relationship between symptoms and blood sugar concentration would be demonstrated irrespective of the value used to define hypoglycemia.

RESULTS

Glucose Tolerance Tests

Despite the fact that all of the patients reported that they had had glucose tolerance tests in the past that diagnosed hypoglycemia, not all of the patients demonstrated hypoglycemia during our standardized test. Patients were placed into the four following different groups on the basis of their GTTs: patients with reactive hypoglycemia (normal GTT, except for one or more values below 65 mg/100 ml during the test); those who were diabetic (elevations of glucose that met the definition of diabetes of the U.S. Public Health Service [13]); those who were normal; and those who were atypical. Eighteen patients were diagnosed as having reactive hypoglycemia, 7 patients had a normal GTT, 4 were diabetic, and 1 patient had fasting hypoglycemia (later diagnosed as secondary to an insulinoma; this patient was considered atypical). The nadirs on the GTTs of the reactive hypoglycemic patients occurred between 75 and 270 minutes of the five-hour test. The mode at which their nadirs occurred was 210 minutes. Two of the diabetic patients and the patient with an insulinoma also had at least one hypoglycemic value. The distributions of the times and the extent of the glucose nadirs during the GTTs are indicated in table 1.

Reported Symptoms

The patients usually reported histories of multiple symptoms. Symptoms reported at least 50% of the time by the entire group included emotional lability, depression, headaches, diaphoresis, tremor, tachycardia, weakness, dizziness, and difficulty thinking. Other

TABLE 1
Distributions of Glucose Nadirs and Times of Glucose Nadirs of 30 Patients During a Five-Hour GTT

Item	Number of Patients, by GTT Diagnosis		
	Reactive Hypoglycemia (N=18)	Normal (N=7)	Diabetic and Atypical (N=5)
Glucose nadir (mg/100 ml)			
36–40	1	0	0
41–45	1	0	0
46–50	2	0	0
51–55	5	0	1
56–60	6	0	2
61–65	3	0	0
66–70	0	2	0
71–75	0	1	0
Above 75	0	4	2
Time of test (minutes)			
0– 30	0	0	2
40– 60	0	0	0
60– 90	2	0	0
105–120	0	0	0
135–150	1	2	0
165–180	3	1	0
210–240	8	2	0
270–300	4	2	3

symptoms reported by 25% or more of the group included nausea, hunger, fatigue, visual disturbances, transient neurological symptoms, and syncope. Each patient complained of an average of 8.6 symptoms. There was no correlation between degree of hypoglycemia and number of symptoms reported (r=.059).

Psychiatric Interview Data

Many of the patients appeared to be experiencing acute emotional distress. However, they denied this distress or attributed it to somatic problems; they displaced their psychological discomfort to concerns and preoccupation with physiological processes. Their personal histories revealed a close relationship between the development of the symptoms and external precipitating stress, e.g., marital discord, separation, divorce, job change, and the death of a close relative.

Some of the symptoms reported, such as sleep disturbance, were compatible with a primary psychiatric disorder. Fifteen of the patients (50%) were judged sufficiently symptomatic (in the interview and according to self-reports) to establish an acute psychiatric diagnosis. Latent problems were suspected in patients who were not formally diagnosed (diagnostic criteria were conservative). Ten patients were considered to have a depressive neurosis, 3 an anxiety neurosis, 1 schizophrenia, and 1 a psychophysiologic reaction (headaches). The distribution of the clinical diagnoses according to the patients' GTT diagnoses is shown in table 2. The numbers are too small for meaningful statistical analysis, but it is interesting to note that all three diagnoses of anxiety neurosis occurred in the reactive hypoglycemia group.

In addition to establishing acute psychiatric diagnoses, we made an effort to document the patients' personality structures on the basis of lifelong characteristics, not just the manner in which the patient responded to his or her current ailment. An unhealthy personality pattern was identified in 19 of the 30 patients. The other 11 were regarded as having an essentially normal or healthy personality. Hysterical personality (12 women, 2 men) was the predominant personality type diagnosed. There was no demonstrable relationship between personality type and GTT diagnosis (see table 2).

Characteristics used in diagnosing a hysterical personality included dramatization of life events, conflicts in sexual identity, narcissism, and immature dependence on others. Characteristics used to define an obsessive-compulsive personality included emotional rigidity, excessive worry and attention to detail, inhibition, and marked conscientiousness.

MMPI Data

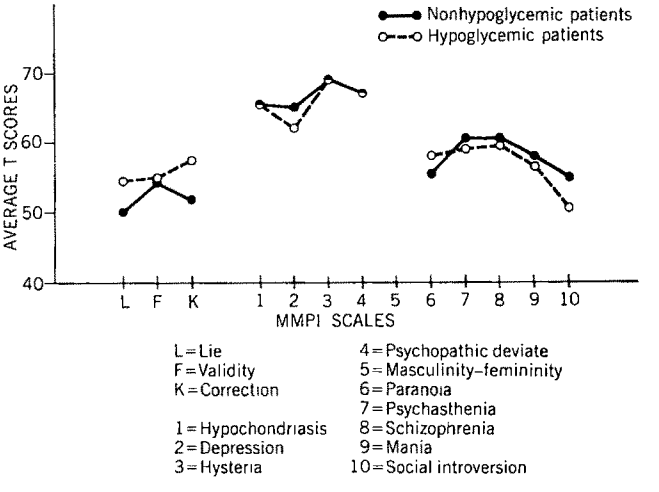
The MMPIs of 18 of the 30 patients were interpreted as abnormal, i.e., their T scores exceeded 70 (2 standard deviations above the norm). Another 9 patients had an elevation greater than 65 on at least one T score, placing them in a borderline abnormal group. The most typical profile was one that contained the "conversion V," in which the scores for scales 1 and 3 (hypochondriasis and hysteria) were at least one-half a standard deviation higher than the score for scale 2 (depression). This MMPI configuration is often associated with hysterical complaints (14, pp. 86–116). Ten of the 21 hypoglycemic patients showed the "conversion V," and 2 of the 9 nonhypoglycemic patients demonstrated this profile (the difference was not statistically significant).

Averaged MMPI T scores failed to distinguish the hypoglycemic group from the nonhypoglycemic group (see figure 1). Average MMPI T scores for the reactive hypoglycemic patients compared with those of patients with normal GTTs also did not show significant differences (see figure 2).

TABLE 2
Distribution of Psychiatric Diagnoses and Personality Patterns of 30 Patients, by GTT Diagnosis

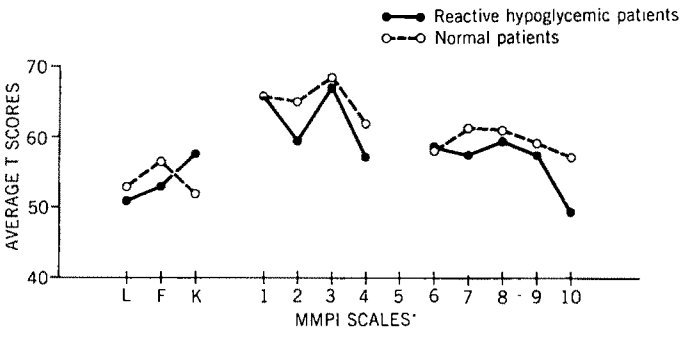
Item	Number of Patients, by GTT Diagnosis		
	Reactive Hypoglycemia (N=18)	Normal (N=7)	Diabetic and Atypical (N=5)
Psychiatric diagnoses			
Anxiety neurosis	3	0	0
Depression	6	2	2
Schizophrenia	0	1	0
Psychophysiologic reaction	1	0	0
Personality patterns			
Hysterical	10	2	2
Healthy	5	3	3
Obsessive-compulsive	3	1	0
Inadequate	0	1	0

FIGURE 1
Comparison of Average MMPI Scores of All Hypoglycemic Patients (N=21)* and All Nonhypoglycemic Patients (N=9)**



* The glucose nadirs of these patients were below 65 mg/100 ml.
** The glucose nadirs of these patients were above 65 mg/100 ml.

FIGURE 2
Comparison of Average MMPI T Scores of Reactive Hypoglycemic Patients (N=18) and Normal Patients (N=7)



* See figure 1 for MMPI scales.

The type of average profile obtained for our 30 patients is consistent with that for people having hysterical personality traits with conversion symptoms. Denial, repression, and somatization have been suggested as defenses against underlying depression and/or anxiety (14, pp. 158–214).

Comparison of the degree of GTT abnormality (as measured by the serum glucose nadir) with the individual MMPI scales of depression, hysteria, and psychasthenia yielded no correlation. All values, as computed by Spearman's rank correlation coefficient, were close to 0.

DISCUSSION

The terms "reactive hypoglycemia" and "functional hypoglycemia" are generally used to describe patients with postprandial hypoglycemia in whom an abnormality of one of the organs of glucose homeostasis is not demonstrable. There are many causes of fasting and postprandial hypoglycemia (15), including such potentially serious disorders as islet cell tumors, adrenal and pituitary insufficiency, severe liver disease, and alcohol-induced hypoglycemia. Other recognized causes of postprandial hypoglycemia include those related to altered gastrointestinal structure and physiology (alimentary hypoglycemia) and the hypoglycemia of early adult-onset diabetes.

Of the many categories of hypoglycemia, functional hypoglycemia has preoccupied the interest of the popular writers. Many physicians advise their patients that an alteration of dietary intake and habits can have a favorable effect upon symptoms. There is a rationale for attributing psychologic symptoms to hypoglycemia (15).

The symptoms of hypoglycemia can be divided into two general pathophysiologic causes. The first, cerebral glycopenia, can cause neurological symptoms that include obtundation of consciousness proceeding to coma, dysarthria, visual disturbances, paresthesia, and other symptoms of central or peripheral nervous system dysfunction.

A second set of symptoms is referrable to a different physiologic process (hyperepinephrinemia). Following heavy intake of sugar or carbohydrate there may be excessive release of insulin. Insulin results in increased glucose utilization and a falling blood sugar. In response to decreasing blood sugar concentrations, a number of counterregulatory hormones (epinephrine, cortisol, glucagon, and growth hormone) are secreted; these act to return blood sugar concentrations toward baseline levels. Hyperepinephrinemia produces generalized physiological effects that include flushing, diaphoresis, tachycardia, tremor, pallor, and a subjective sense of weakness and anxiety. Patients may also complain of nonspecific symptoms, e.g., headaches, nausea, abdominal pain, hunger, fatigue, and dyspnea.

Given these symptoms and their relationship to carbohydrate intake, there is a possibility that a person suffering from what appears to be emotional distress may have a metabolic disturbance treatable by adherence to a low-carbohydrate diet. Logically, the severity of symptoms should be related to the degree of reactive hypoglycemia.

CONCLUSIONS

Our data do not support the proposition that the emotional distress experienced by hypoglycemic patients is due to their hypoglycemia. Neither the number of symptoms nor the severity of psychological distress (as measured objectively by the MMPI) was related to the degree of hypoglycemia demonstrated in our sample of 30 patients.

What is remarkable in our findings in these patients, who all believed that they suffered from reactive hypoglycemia whether they did or did not, was the similarity in their reported symptoms, psychiatric diagnoses, and MMPI scores. Our MMPI data for the hypoglycemic patients are in agreement with those obtained by Anthony and associates (11), but these authors found differences between hypoglycemic patients and a control group (hospitalized patients with endocrine abnormalities). Their conclusion that hypoglycemia may specifically lead to the development of hysterical characteristics is intriguing, but it cannot be substantiated by our data. We found no difference between patients who were hypoglycemic and those who thought they were hypoglycemic.

While suggesting that many hypoglycemic patients are hysterical, our data also indicate that some hypoglycemic patients are obsessive and that others have no discernible psychiatric disorder. We also found that some people who believe themselves to be hypoglycemic are hysterical but have no demonstrable hypoglycemia. Landmann and Sutherland (16) studied a group of psychosomatic patients and also found a high percentage of hypoglycemic patients but no relation-

ship between symptoms or personality characteristics and hypoglycemia.

What may be in question here is not so much whether hypoglycemia is specifically related to certain psychiatric syndromes but what type of patient is more likely to be referred for study of hypoglycemia. Both hysterical and obsessive patients are often concerned with somatic complaints. In the search for the etiology of their symptoms many laboratory procedures, including a GTT, may be performed. In an effort to establish a diagnosis, all of the patient's complaints may be mistakenly attributed to hypoglycemia, although, in essence, what brought the patient to the physician were symptoms of depression or anxiety. The finding of a reactive hypoglycemic glucose tolerance curve may be more of an incidental finding than the basis for etiology of the patient's symptoms (17).

This formulation does not negate the concept of hypoglycemia as a real entity that can express itself in multiple ways and cause genuine somatic discomfort. Rather, we would emphasize that the numerous nonspecific somatic complaints of hysterical and obsessive patients may be overdiagnosed as hypoglycemia, while more psychologically normal individuals may tolerate the symptoms of hypoglycemia with less anxiety and therefore be less likely to seek medical attention.

This hypothesis is consistent with reports that it is not uncommon for a nonsymptomatic volunteer subject to have a GTT with some markedly low values (18, 19). In the report of Park and associates (19), 23% of an apparently normal population had blood sugar levels below 50 mg/100 ml during a standard five-hour GTT. Another article (20) also stressed the epidemic nature of "nonhypoglycemia" and the number of people who now regard themselves as hypoglycemic without demonstrable GTT abnormality. Some of the patients in our sample of 30 appeared to fall into this category.

Another consideration is whether the anxiety and depression experienced by a patient with a psychiatric disorder affects glucose tolerance. However, our data demonstrate no relationship between depression and anxiety (as measured by the MMPI) and degree of hypoglycemia. Herzberg and associates (21) were also unable to demonstrate a relationship between depression and glucose tolerance.

Fabrykant (22) noted that the hyperglycemic effect of low-carbohydrate diets is limited to the first hours of the GTT and that the magnitude of the hypoglycemic response after carbohydrate restriction may exceed the response in controlled tests after standard high-carbohydrate diets. Therefore, people who believe themselves to be hypoglycemic may treat themselves with low-carbohydrate diets and then have their self-diagnosis "confirmed" by a five-hour GTT. Fabrykant recommended that one not be too quick to ascribe symptoms to hypoglycemia because of the danger of overlooking a more serious illness. The findings of our study suggest that one serious illness that can be easily overlooked is depression.

This pilot study, which used volunteer subjects who believed themselves to have reactive hypoglycemia, uncovered significant psychopathology but no intersubject relationship between the severity of psychiatric symptoms and the degree of hypoglycemia. Because of the popular interest in hypoglycemia and the potential of misdiagnosis of psychiatric illness, further research is indicated. Use of large numbers of randomly selected subjects not already identified as patients would be desirable but methodologically difficult because of such factors as the need for informed consent.

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Family and Character Change at Buffalo Creek

BY JAMES L. TITCHENER, M.D., AND FREDERIC T. KAPP, M.D.

Psychiatric evaluation teams used observations of family interaction and psychoanalytically oriented individual interviews to study the psychological aftereffects of the 1972 Buffalo Creek disaster, a tidal wave of sludge and black water released by the collapse of a slag waste dam. Traumatic neurotic reactions were found in 80% of the survivors. Underlying the clinical picture were unresolved grief, survivor shame, and feelings of impotent rage and hopelessness. These clinical findings had persisted for the two years since the flood, and a definite symptom complex labeled the "Buffalo Creek syndrome" was pervasive. The methods used by the survivors to cope with the overwhelming impact of the disaster—first-order defenses, undoing, psychological conservatism, and dehumanization—actually preserved their symptoms and caused disabling character changes.

On February 26, 1972, an enormous slag dam gave way and unleashed thousands of tons of water and black mud on the Buffalo Creek valley in southern West Virginia. This Appalachian tidal wave destroyed everything in its path, killing 125 people and leaving 4,000 homeless and carrying away human bodies, houses, trailers, cars, and other debris. It expended its force in no more than 15 minutes at any one point in the 18-mile-long valley.

Just below the dam and the tipple of the Buffalo Mining Company stood the town of Saunders; there was no trace of this town minutes after the black water broke through the dam. The sides of the valley are steep at this point, and the wall of water and mud caromed from side to side, miraculously sparing some homes but destroying many others as it slammed down the valley.

The wall of water sped through 14 mining hamlets with names like Crites, Becco, Lundale, and Pardee, hitting their schools, churches, taverns, stores, and homes, leaving no trace of some and damaging nearly all. The sides of the valley become less steep and it spreads out, so the black sludge and water became more of a "flash flood" at Amherstdale and just an overflow at Man, where it reached the Guyandotte River.

None of the settlements in Buffalo Creek, which had a total population of 4,000–5,000 inhabitants, were incorporated. There was no governmental organization beyond the commercial structures provided by post offices, schools, and churches. There are five deep mines in operation and evidence of stripmining is everywhere. In spite of the stripping, the ugly tipples, the dozen or so huge black heaps of waste, the railroad and highway construction, it is still a beautiful valley, and young adults there will tell you it was once much more beautiful, with pleasant homes and gardens where there are now primarily mobile homes. It was and is a middle-class area. Nearly all families are supported by employment in the coal mines or in the supporting industries and services. There is an accepted (but not documented) belief that this valley had not had the degree of emigration of young people that typified others like it since the Depression.

There had been rumors for years that the dam would give way, but hundreds of people reported they did not believe it had really happened until a few moments after the fearsome sight and sound of the advancing water. All the survivors know that the time of the dam break (8:00 a.m. on a Saturday morning) was fortunate. Few people were down in the road, and the children were not in or waiting for the school buses. Nevertheless, 125 were killed, and most lost their homes and possessions.

Subsequently, a group of 654 survivors of this disaster from 160 families began a legal action against the

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Drs. Titchener and Kapp are Professors, Department of Psychiatry, University of Cincinnati Medical Center, 234 Goodman St., Cincinnati, Ohio 45229.

company that owned the dam. This group contacted the law firm of Arnold & Porter in Washington, D.C., and a legal team headed by Mr. Gerald Stern traveled to the area to interview survivors. His observations of the psychological effects of the disaster and a summary of the litigation are presented in "From Chaos to Responsibility" in this section. The law firm first contacted Robert J. Lifton, M.D., who assessed "The Human Meaning of Total Disaster" (1), and Kai Erikson, Ph.D., whose observations of the situation in the community are presented in "Loss of Communality at Buffalo Creek." The suit was settled in July 1974 for $13.5 million of which $6 million was for psychological damages.

The legal team then retained one of us (J.L.T.) to organize a group of experts to interview the survivors and assess for the court the psychological impairment they had suffered as a result of the flood. This paper presents our findings on the severity and duration of these psychological effects, a symptom complex we have labeled the "Buffalo Creek syndrome." The size and composition of the evaluation teams varied with the nature of the families assigned to them. A full-sized team consisted of a general psychiatrist, a child psychiatrist, and two psychologists or case workers. These teams did their work in the valley itself, visiting the respondents' mobile homes and those houses that were still standing.

We conducted a pilot study consisting of interviews of 50 survivors in June of 1973. The court then directed that all of the survivor-plaintiffs be interviewed, as all were bringing suit separately. These evaluations were carried out on several long weekends in the spring of 1974.

We began each evaluation with a family interview in which we asked the survivors to talk about their experiences on the "day of the black water" and during the weeks and months that followed. As they talked, we were able to see beyond the immediate clinical phenomena to these people's underlying feelings and their ways of coping with them. The family sessions were followed by psychoanalytically oriented individual interviews with each family member, conducted in backyards, living rooms, or on porches.

EVALUATION FINDINGS

Disabling psychiatric symptoms such as anxiety, depression, changes in character and lifestyle, and maladjustments and developmental problems in children were evident more than 2 years after the disaster in over 90% of the individuals we interviewed. We asked ourselves whether we were examining people who were presenting major symptomatology and character problems that resulted from basically weak ego structures and who were using the disaster in order to win a large settlement from the mining company. Our answer was and is "no." In our evaluations, we witnessed difficult and prolonged struggles with powerful feelings and ideas aroused by the traumatic experience of the disaster and the very uneven attempts of the survivors to reorganize themselves and redevelop shattered coping and adaptive mechanisms. The affects associated with the catastrophe and its aftermath, as well as the psychological and social ways these people chose to deal with them, must be seen against the background of the universal crises of human development. The attendant threats of separation, abandonment, castration, and death—residuals of the developmental crises of separation and individuation—provided the context for the meaning of the catastrophe to the survivors (2, 3).

We found a definite clinical syndrome in the survivors of the Buffalo Creek disaster that arose from both the immediate impact of the catastrophe on each individual and the subsequent disruption of the community and that affected everyone living there. We are all predisposed by previous experiences to be traumatized by pathogenic forces as destructive and awesome as the Buffalo Creek catastrophe. Variations in the clinical picture resulted from individual differences in modes of processing and reorganizing the traumatic experience (4).

A clear pattern emerged from our evaluations and analyses. A traumatic neurotic syndrome was diagnosed in more than 80% of the survivor-plaintiffs, and changes in character structure were equally widespread. These changes, although they were attempts at readjustment, occasionally resulted in maladjustment in the social sense and always went in the direction of psychologically disabling limitations.

Character changes represent the stabilizing neurosis, the psychologically hardening and fixating consequences of the catastrophe. We found conscious and latent meanings and understandings and misunderstandings of the disaster and its aftermath, all of which were associated with the feelings and conflicts aroused by the trauma. The result of this was changes in object relations and attitudes toward the self. We delineated various processes of reorganization—attempts at putting personality functions back together—that were directed toward reintegration and resumption of a nontraumatized life.

We shall indicate below how this personality reorganization, which was so aimed at prevention of recurring experience of the traumatic state, actually *interfered* with flexible and effective recovery and thus preserved symptom patterns and forced changes in the way of life.

SYMPTOMS AND CHARACTER CHANGES

During the first days and on into the weeks and months after the disaster, the survivors reported disorganization and sluggishness in thinking and decision making. They complained of having difficulty controlling their emotions. These problems ranged from emotional outbursts to the simple inability to feel any-

thing. Some described transient hallucinations and delusions. Almost all reported anxiety, grief, and despair, with severe sleep disturbances and nightmares. Later, the anxiety was manifested in obsessions and phobias about water, wind, rain, and any other reminder that the disaster could recur. Occasionally these obsessive disturbances coalesced and became a group phenomenon. For instance, the wife of a community leader never slept when he was asleep so that one of them would always be on the alert. On rainy nights, this man received phone calls regarding rumors that another dam was about to give way. He would then take his rifle and spend the night sitting on the supposedly weakened dam, guarded by others to protect him from attack.

Grief over the loss of relatives, friends, possessions, and mementos such as family Bibles, as well as the loss of the feeling of communality discussed by Dr. Erikson, was widespread. For many, unresolved grief turned into depressive symptoms, ideation, and behavior, and some developed a depressive lifestyle (5). In some individuals, depression was channeled into a wide range of somatic complaints, with probable increases in the incidence of duodenal ulcer and hypertension.

Many of these people have become listless, apathetic, and less social since the disaster. They cling to their families, lack ambition, and are disinterested in former hobbies and sports. These changes have led to an overall limitation of essential expression, a lack of zest for work and recreation, and despair about ever again resuming the lifestyle they once had.

THOUGHTS, FANTASIES, AND FEELINGS AROUSED BY THE DISASTER

The survivors referred to the disaster as "the end of time" or "the end of everything," and noted that "No one who was not there could ever really know what happened." They were haunted by visual memories and emotions associated with the drownings of relatives and friends and of blackened bodies and parts of bodies that were uncovered for weeks after the flood.

All of the survivors had to confront the loss of a sense of personal invulnerability. The former feeling of comfort and assurance about the continuity of life had depended partly on magical beliefs that horrible things like this disaster do not happen to one; that they could not occur in nice sane communities in this country. Then the impossible happened. The shock was overwhelming and a new outlook took form that reflected a swing from the former sense of invulnerability to pessimism, emptiness, and hopelessness. We heard such comments as "Nothing counts anymore"; "What's the use now?"; and "Since we lost everything, what's to be gained by trying?" The disaster took on the meaning of chaos, helplessness, and death, giving rise to feelings of personal insignificance.

Three other reactions contributed to depressive symptoms and lifestyles. The first was a feeling of impotent rage over the destruction to life, property, and a way of life. This rage is an explosion of feeling against the attack on the self. The victim has little outlet for his anger or hope of satisfaction. This feeling had special intensity because the destruction in Buffalo Creek was man-made; it was caused by the inexplicable inhumanity of a powerful corporation that gave terrible evidence of not caring about its employees or their community. The survivors' guilt was expressed in a wide variety of derivative feelings about the self, in symptoms, in character change, and in behavior through self-denial and lack of hope. These conflicts were not resolved, and their persistence took form in identification with the dead in dreams, actions, and attitude toward life (6).

No one behaves exactly as he thinks he should in a hazardous situation, particularly in a situation he is powerless to influence. Memory becomes clouded and feelings of helplessness influence the way one looks back on the traumatic event. Many people in Buffalo Creek manifested "survivor shame." One of the actual heroes of Buffalo Creek, who had been extraordinarily effective in mobilizing and leading rescue efforts, was able to fend off depression and anxiety in the first four weeks after the flood while he worked relentlessly to help others. When he attempted to return to his former work, he was overwhelmed by anxiety and depression connected with feelings of inadequacy. He developed a phobia connected with his job, began drinking heavily, and became clinically depressed.

We noted in many people a sense of isolation and feelings of alienation combined with an increased need for vigilance and a tightening of the ring around the family. Former feelings of self-assurance, sociability, trust in neighbors, and enjoyment of community activities disappeared. The isolation we observed clinically can be explained by the depressive reactions, the chronic anger, the loss of a way of life, and the dissolution of self-confidence and basic trust.

It has been hypothesized that the emotional disturbances aroused in the victims of disaster quickly disappear after the stress has subsided. Our work at Buffalo Creek suggests that this is rarely the case; the manifestations of a traumatic neurosis do not subside with the receding flood waters. The effects may seem to disappear quickly if one is not alert to the subtle covering-up behavior of the victims of a psychic trauma.

Lifton and Olson (1) explain the persistence of traumatic effects on the basis of an analysis of the nature of the disaster itself and the special psychological effects of such an experience. Our study complements their work by showing how the effects of a traumatic event are preserved by the modes of adaptation to overwhelming fears and hopelessness. The very attempts to protect self, family, and community from a recurrence of helplessness and loss are responsible for the individual and societal neurosis and restrictive character change. Our combined approach has been to show what occurred, the nature of its impact on the psyche,

and why its effects became chronic.

One can analyze the sequential formation of the "Buffalo Creek syndrome" as follows. The disaster activated intense affects, including fear, rage, and helplessness. These waves of external and internal overstimulation overran the stimulus barrier and the ego's capacity to integrate the traumatic experience and control and discharge the affects. There was temporary ego collapse and the ego was damaged. We estimate that reorganization of the ego in whole or in part required 6 to 24 months. The course of the reorganization and the way individuals processed these affects, memories, and the associated conflicts made the ultimate difference in outcome. The survivors' course of ego reorganization and their manner of processing the disaster experience were reflected in their symptoms and character change (2, 4, 7, 8). The variables in the reconstitution of the personalities of the survivors we studied can be divided into four categories.

PERSONALITY RECONSTRUCTION

First-Order Defenses

There was a continuous and steady deployment of a coordinated system of character-shaping first-order defenses (9), i.e., projection, externalization, and denial. Projection defended against feelings of guilt and shame aroused by the disaster. The constructors of the dam, state and federal agency representatives, and intrusions from the society outside of the valley became objects of increasing anger and fear. Externalization blocked awareness of this anger and fear as well as feelings of helplessness. Individuals became sensitive to and acutely observant of the anxiety and unrest in their families, coworkers, and the social group. Denial defended against recognition that the self had been changed in any way; it disavowed the feeling of helplessness and the awareness of psychological scarring (10). Denial enabled people to believe that while much had happened to them and to those around them, they had not been affected in an *essential* way, and that they were the same people they had been before the flood. This defensive complex protected against emotions that would have otherwise reactivated memories and feelings of fright and helplessness. It was oriented to the present and functioned continuously, preventing the gradual recollection and discharge of the feeling of helplessness and blocking recognition of the irrationality of shame and guilt. Although one can never be the same after an experience with disaster, this defensive system provides a desperate sort of status quo that substitutes for personal regrowth.

Efforts to "Undo" the Disaster Experience

Undoing consisted of efforts to change the past by reliving the disaster in dreams and other ways, giving it a different outcome. Survivors' memories of the early postdisaster period contained fantasies of magical reliving of childhood stresses. Attempts at undoing also appeared in strange, symbolic reenactments of the trauma, sometimes leading to violence to the self or others. Freudian repetition compulsion was often replaced by the mechanism of undoing, which is a defense against facing the anxiety associated with the trauma.

The dreams of the survivors during the period of initial shock and (in many cases) for months thereafter were fantasied attempts to relive the disaster, but with a less painful outcome. At first, such dreams were unsuccessful and people awoke from them in terror. As time passed, the dreams were modified. Although the affects remained frightening, the subject matter shifted from the flood to previous, often long-past, images of chaos and threats of annihilation. The dreams no longer involved direct reliving of the disaster but instead depicted stressful episodes that represented repetitions of normal developmental crises such as separation, abandonment, castration, and guilt (1, 8).

The regressive process in these traumatic neuroses differs from that in other psychoneuroses. The goal is not gratification or mastery of infantile conflicts, but rather an attempt to work through recent traumatic anxiety. The anxieties of infantile and childhood phases of personality development become the focus of undoing because these problems had been successfully contained or overcome; dreams of long-past stresses that had been mastered provided reassurance to the survivors that they could overcome the recent trauma. Just as "examination dreams" attempt to deal with anticipatory anxiety by fantasying a past stress that had been overcome, dreams that are characteristic of traumatic neuroses attempt to neutralize the overwhelming anxiety of the traumatic event by recalling successful past adaptations to difficult situations. Each of these phases of dealing with normal stress, reproduced in the survivors' dreams, is common to all persons as part of human epigenesis. Each past crisis included not only a deprivation of instinctual demand but also a threat to the continuity of life. The latter aspect is what makes them particularly suitable for undoing the threat of annihilation experienced in a trauma like the Buffalo Creek disaster.

Because undoing relies on omnipotence and magic, it prevents recognition of the influence of guilty, shameful attitudes toward the self. The undoing process—aimed at fending off fearful anticipation of a recurrence of the traumatic experience—is a continuing obstacle to the relatively nonanxious acceptance of human vulnerability that is necessary for readaptation.

The Psychological Emphasis on Survival

Psychological conservatism consisted of avoidance of situations that might raise the level of excitation either internally or externally. It is the defensive and ego psychological counterpart of the psychic numbing described by Lifton and Olson (1). We perceive psychic conservation as mental activity designed to control behavior by banking energies, surrendering ambition, reducing enthusiasm, dampening socializing and love-

making, and discouraging novel experience (11). Psychological conservatism accepts survival as the only goal of existence. It is a trade-off: the individual accepts hopelessness in the present to prevent helplessness in the future, as if to say, "Better to live without hope than not to live at all." Psychological conservatism functions as if the disaster will recur tomorrow, thus totally distorting an individual's view of the future. If you live as though the dreaded uncertainty is certain to occur, you become a psychic conservative.

Dehumanization

Dehumanization affects one's view of life and human relationships and has a direct toxic effect on personality function. Every disaster places man at the mercy of forces beyond his control. The feeling of being a pawn of fate is dehumanizing—people feel without appeal, beyond empathy, and cannot be persuaded or assuaged. When the catastrophe is man-made, dehumanization is magnified. In Buffalo Creek, there was the terrible realization that other human beings had planned, built, and maintained an unsound dam and then acted irresponsibly and uncaringly after the resulting disaster. The defense of dehumanization is an example of identification with the aggressor. It destroyed pride and joy in being human.

Dehumanization may be mitigated by corrective experiences with empathic people in the helping professions and private and public institutions. Collaboration with other sufferers in a law suit against the dehumanizing aggressor may also be useful in that it can ensure that it will be more difficult for such organizations to risk human life in the future.

CONCLUSIONS

It is our belief that the reactions we have described are not those of individuals with weak egos who were exaggerating their complaints in order to win a law suit. These people, by and large, did not exaggerate their complaints; the majority minimized or denied them. If their reactions were merely exacerbations of old neurotic symptoms and problems, we would have encountered a wider range of psychoneurotic reactions. Although there were differences in modes of response, the uniformity of the psychological reactions comprising the Buffalo Creek syndrome was striking. Our analyses of dreams and early memories, reported elsewhere (12, 13), support the consistency and severity of this syndrome.

We found a definable clinical entity characterized by a well-delineated group of clinical symptoms and changes in character and lifestyle that were related to clear-cut psychopathogenic factors precipitated by the disaster. All of us have in our unconscious memory systems encounters with the various forms of dread that a disaster reawakens. There need not be any preexisting neurosis for the Buffalo Creek syndrome to become disabling and chronic. All of us are susceptible to traumatic neurosis and the "death imprint."

To be successful in treating these traumatic neuroses, we must substitute active recall and working through of the painful memories of helplessness and separation for counterphobic behavior, passive reproduction of the experience in dreams, and magical ways of living out and reenacting the trauma. The change from passive to active experience, from reproduction to re-creation is the essential thing. By linking long-past and previously worked-through childhood anxieties with the overwhelming anxieties aroused by the recent disaster, we may be able to strengthen the ego of the individual with a traumatic neurosis. Through his relationship with helping and capable persons and institutions, the disaster survivor is given an opportunity for regrowth, much like the ego development that came about as the individual met and dealt with the normal crises of growing up.

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From Chaos to Responsibility

BY GERALD M. STERN, L.L.B.

The litigation initiated by the 625 survivors of the Buffalo Creek flood who refused to settle with the coal company claims office was a landmark case. For the first time, individuals who were not present at the scene of a disaster were allowed to recover for mental injuries. Psychic impairment, the term coined for these injuries, was found in virtually all of the survivor-plaintiffs. In an out of court settlement, the survivors were awarded $13.5 million, $6 million of which was distributed on the basis of a point system as compensation for the psychological damages.

THE DESTRUCTION of the Buffalo Creek community gave rise almost immediately to the creation of a new kind of group—a community of 625 survivors from 160 families who joined together to sue the coal company that owned the dam. These individuals, unlike the majority of the survivors, refused to settle their cases at the coal company claims office. Instead, they sought legal help outside the state of West Virginia.

This group contacted Arnold & Porter, a law firm in Washington, D.C., and we agreed to represent them.[1] We immediately went to Buffalo Creek and spent many days interviewing survivors at Charlie Cowan's gas station, one of the few buildings remaining in the Buffalo Creek valley. Mr. Cowan was the leader of the citizens' committee that called to ask for our legal help. The survivors' legal right to sue for traditional damages was clear; they could sue for lost property, for their homes and all their possessions, for physical injuries, or for the death of family members. However, it soon became apparent that they also had significant mental injuries, and it was not so clear whether the law would permit recovery for these damages.

The magnitude and significance of these mental and emotional damages hit me personally when I interviewed a coal miner who had lost his 22-month-old son and his pregnant wife in the disaster. The flood waters caught this family asleep in their home. As the wife disappeared in the black water, she cried out to her husband to save their son. He held the child tightly and tried to struggle to safety, but houses and debris battered him and the child as they were washed miles down the valley. Somewhere in this maelstrom, he lost his grip on his son, who disappeared forever into the black waters. Eventually, this man was able to struggle to safety, although his body was badly lacerated by the jagged wood in the water. At the time I interviewed him, my own son was exactly 22 months old. I was terribly upset by his story and decided to try to expand the lawsuit to recover for his mental agony and for the mental suffering of others like him.

We contacted Robert J. Lifton, M.D., who had studied the survivors of Hiroshima. He agreed to interview a number of our clients and to help us explain to the court in lay terms the common psychiatric injuries of these survivors. He also suggested that we ask Dr. Kai Erikson, whose findings are reported in this section, to study the sociological aspects of this disaster. With these two men as our principal experts, we articulated for the court and for the coal company defendant what we called the "psychic impairment" damages suffered by every one of our survivor-clients.

We coined the term "psychic impairment" to include both the psychiatric damages identified by Dr. Lifton and the loss of communality found by Dr. Erikson. We wanted to avoid alleging that the survivors suffered mental illness and felt that the phrase "psychic impairment" had a less negative connotation.

Eventually we also employed a team of psychiatrists from the University of Cincinnati, some of whose findings are also presented in this section, to interview each of our clients. The coal company also retained a psychiatrist—actually, a physician whose primary field was neurosurgery—and a young psychologist in training, who also examined each of the 625 men, women, and children involved in the lawsuit.

Our psychiatric studies indicated that almost all of the survivors were suffering from psychiatric damage of varying degrees as a result of this disaster. In contrast, the physician retained by the coal company determined that the survivors generally suffered only transient situational disturbances that he felt should have abated soon after the disaster. The fact that the survivors still had disturbances when he examined them some 18 months after the disaster led him to presume almost invariably that these people were suffering primarily from preexisting mental conditions.

Under traditional legal principles, if the survivors had been physically injured by the flood waters and, as a result, had suffered psychiatric damages, they could recover full monetary damages unless their current

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Mr. Stern is a partner in the law firm of Arnold & Porter, 1229 19th St., N.W., Washington, D.C. 20036.

[1]A much more detailed report of my observations and involvement in this case will be presented in a forthcoming work (1).

mental conditions were merely the result of an aggravation of preexisting mental conditions. Our physicians indicated that the survivors' psychiatric damages were caused *solely* by the disaster. The coal company physician disagreed. This is a dispute juries must often resolve in cases involving psychiatric damages.

The more interesting and more difficult legal question presented by this case was whether the survivors could recover monetary damages at all, even if the jury found that all of the survivors' present psychiatric injuries were caused by the disaster. Traditionally the law does not permit recovery for psychiatric injury on the sole ground that the injury can be proven to have been caused by another person or persons. For example, a mother who sees a truck run over and kill her child may suffer severe psychiatric trauma, but the law traditionally has denied the mother recovery for her own suffering, terming her a mere bystander. Needless to say, an individual who sees a friend killed has even less chance in the courts of recovering for mental suffering.

In this case, most of the survivors were not seriously injured physically. Many of them had run up the side of the valley just ahead of the flood waters, and some of them were not even in the valley at the time of the disaster. For example, one survivor was visiting in New Mexico, another was in Florida, some were in jail, and others were in hospitals in nearby towns outside the valley. Nevertheless, we insisted that all of the survivors were entitled to recover for their mental suffering, even if they suffered no physical injury, saw or heard no relative or friend in peril, or were absent from the valley on the day of the disaster. We argued that each resident of the valley, even those who were not there during the flood, was a direct victim of the coal company's reckless conduct and not merely a bystander.

The court agreed with this contention and held that all survivors—even those who were outside the valley at the time of the disaster—could collect for mental injury if we could convince the jury that the coal company's conduct was reckless (i.e., more than merely negligent), and that this reckless conduct caused the survivors' mental suffering (2).

Once the coal company realized that the court would not dismiss the psychic impairment claims of any of the 625 survivor-plaintiffs in this lawsuit, we reached a settlement for a total of $13.5 million, to be divided among the survivors by their own attorneys. We first calculated the payments for real and personal property losses, for wrongful deaths, lost wages, other miscellaneous claims, expenses, and legal fees. This left approximately $6 million to be distributed for psychic impairment. We distributed this money to the 625 plaintiffs using a point system based on their immediate involvement with the disaster, their medical disability, their loss of community ties, and the disruption of their way of life. Each survivor received between $7,500 and $10,000 after all expenses and legal fees were deducted. Approximately $2 million of the $6 million was placed directly in a trust fund for the 224 children under the age of 18 who were plaintiffs in the case.

The court's approval of this substantial monetary settlement for survivors' psychic impairment established a significant legal precedent for recovery in cases of mental suffering. The court was not bound by concepts of space and time. Instead, the court recognized that it is the permanence of loss, rather than the witnessing of the disaster, that causes mental suffering. In other words, the court (and eventually the coal company) was persuaded that the relief provided by the law should be determined not by narrow traditional legal principles but by fairly modern psychiatric and sociological principles.

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Loss of Communality at Buffalo Creek

BY KAI T. ERIKSON, PH.D.

The survivors of the Buffalo Creek disaster suffered both individual and collective trauma, the latter being reflected in their loss of communality. Human relationships in this community had been derived from traditional bonds of kinship and neighborliness. When forced to give up these long-standing ties with familiar places and people, the survivors experienced demoralization, disorientation, and loss of connection. Stripped of the support they had received from their community, they became apathetic and seemed to have forgotten how to care for one another. This was apparently a community that was stronger than the sum of its parts, and these parts—the survivors of the Buffalo Creek flood—are now having great difficulty finding the personal resources to replace the energy and direction they had once found in their community.

THE TRAUMA experienced by the survivors of the Buffalo Creek disaster can be conceptualized as having two related but distinguishable facets—the individual trauma and the collective trauma.

By individual trauma, I mean a blow to the psyche that breaks through one's defenses so suddenly and with such force that one cannot respond effectively. As the other papers in this section make abundantly clear, the Buffalo Creek survivors experienced just such a blow. They suffered deep shock as a result of their exposure to so much death and destruction, and they withdrew into themselves, feeling numbed, afraid, vulnerable, and very alone.

By collective trauma, I mean a blow to the tissues of social life that damages the bonds linking people together and impairs the prevailing sense of communality. The collective trauma works its way slowly and even insidiously into the awareness of those who suffer from it; thus it does not have the quality of suddenness usually associated with the word "trauma." It is, however, a form of shock—a gradual realization that the community no longer exists as a source of nurturance and that a part of the self has disappeared. "I" continue to exist, although damaged and maybe even permanently changed. "You" continue to exist, although distant and hard to relate to. But "we" no longer exist as a connected pair or as linked cells in a larger communal body.

The two traumas are closely related, of course, but they are distinct in the sense that either of them can take place in the absence of the other. For instance, a person who suffers deep psychic wounds as the result of an automobile accident, but who never loses contact with his community, can be said to suffer from individual trauma. A person whose feelings of well-being begin to wither because the surrounding community is stripped away and no longer offers a base of support (as is known to have happened in certain slum clearance projects) can be said to suffer from collective trauma. In most large-scale human disasters, of course, the two traumas occur jointly and are experienced as two halves of a continuous whole. For the purposes of this paper, however, it is worthwhile to insist on the distinction at least briefly, partly because it alerts us to look for the degree to which the psychic impairment observed in settings like Buffalo Creek can be attributed to loss of communality, and partly because it underscores the point that it is difficult for people to recover from the effects of individual trauma when the community on which they have depended remains fragmented.

I am proposing, then, that many of the traumatic symptoms experienced by the people of Buffalo Creek are as much a reaction to the shock of being separated from a meaningful community base as to the actual disaster itself.

It should be noted that "community" means much more in Buffalo Creek than it does in most other parts of the United States. Much has been said in the literature on Appalachia about the importance of kinship and neighborliness in mountain society. Although it is true that coal camps like the ones along Buffalo Creek differ in many ways from the typical Appalachian community, the people of Buffalo Creek were nonetheless joined together in the close and intimate bonds that sociologists call *gemeinschaft*. The rhythms of everyday life were largely set by the community in general and governed by long-standing traditions, and the social linkages by which people were connected were very strong. In Buffalo Creek, tightly knit communal groups were considered the natural order of things, the envelope in which people live.

Long stories must be made short in a presentation like this, so I will simply summarize my theme by stating that the human communities along Buffalo Creek were essentially destroyed by the disaster and its after-

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Dr. Erikson is Professor of Sociology and Chair, American Studies Program, Yale University, New Haven, Conn. 06520.

math. The flood itself forced the residents of the hollow into a number of nearby refugee camps from which they were, for a variety of reasons, unable to escape. The result was that the majority of the Buffalo Creek survivors remained in the general vicinity of their old homes, working in familiar mines, traveling along familiar roads, trading in familiar stores, attending familiar schools, and sometimes worshipping in familiar churches. However, the people were scattered more or less at random throughout the vicinity—virtually stranded in the spots to which they had been washed by the flood—and this meant that old bonds of kinship and neighborhood, which had always depended on physical proximity, were effectively severed. People no longer related to one another in old and accustomed ways. The threads of the social fabric had snapped.

A year after the disaster (which is roughly when most of the authors represented in this section first encountered these people) visitors to Buffalo Creek were struck by a number of behavioral manifestations that seemed to be exhibited by almost everyone in the valley and, for that matter, continue to this day. Several of these manifestations are discussed elsewhere in this section. I would like to mention three by way of illustrating a larger point.

DEMORALIZATION

First, the survivors clearly suffer from a state of severe demoralization, both in the sense that they have lost much personal morale and in the sense that they have lost (or so they fear) most of their moral anchors.

The lack of morale is reflected in a profound apathy, a feeling that the world has more or less come to an end and that there are no longer any sound reasons for doing anything. People are drained of energy and conviction, not just because they are still stunned by the savagery of the flood but because activity of any kind seems to have lost much of its direction and purpose in the absence of a confirming community surround. They feel that the ground has been pulled out from under them, that the context in which they had worked, played, and cared for others has more or less disappeared. One survivor said,

> I don't know. I just got to the point where I just more or less don't care. I don't have no ambition to do the things I used to do. I used to try to keep things up. But anymore I just don't. It seems I just do enough to get by, to make it last one more day. It seems like I just lost everything at once, like the bottom just dropped out of everything.

I suppose the clinical term for this state of mind would be depression, but one can hardly escape the impression that it is, at least in part, a reaction to the ambiguities of postdisaster life in the valley. The survivors are literally out of place and uprooted. They had never realized the extent to which they relied on the rest of the community to reflect a sense of security and well-being, or how much they depended on others to supply them with a point of reference.

The people of Buffalo Creek are also haunted by a suspicion that moral standards are beginning to collapse all over the valley, and in some ways it would appear that they are right. As is so often the case, the forms of misbehavior people find cropping up in their midst are exactly those about which they are most sensitive. The use of alcohol, always a sensitive problem in Appalachian society, has apparently increased, and there are rumors everywhere that drugs have found their way into the valley. The theft rate has also gone up, and theft has always been viewed in the mountains as a sure index of social disorganization. The cruelest cut of all, however, is that younger people seem to be slipping away from parental control and are becoming involved in nameless delinquencies. This is an extremely disturbing development in a culture so devoted to the family and so concerned about generational continuity.

This apparent collapse of conventional morality has a number of curious aspects. For one thing, observers generally feel that there is much less deviation from community norms than the local people seem to fear. Moreover, there is an interesting incongruity in these reports of immorality—one gets the impression that virtually everyone is coming into contact now with persons of lower moral stature than they did formerly. This, of and by itself, does not make very much logistical sense. One survivor said flatly,

> The people of Buffalo Creek tended to group themselves together; therefore the breaking up of the old communities threw all kinds of different people together. At the risk of sounding superior, I feel we are living amidst people with lower moral values than us.

Perhaps this is true—but where did all these sordid people come from? Whatever else people may say about their new neighbors in the refugee camps, they are also from Buffalo Creek, and it is hard to avoid the suspicion that their perceived immorality has as much to do with their newness as with their actual behavior. It may be that relative strangers are almost by definition less "moral" than familiar neighbors. To live within a tightly knit community is to make allowances for behavior that might otherwise look deviant. New neighbors do not qualify for this clemency—not yet, at least—and to that extent, their very unfamiliarity may seem to hint at vice all by itself.

The collapse of morality in Buffalo Creek thus seems to have two edges. We have sufficient evidence to believe that certain forms of deviation are actually on the increase, although this is a difficult thing to measure accurately. However, we also have reason to believe that the breakdown of accustomed neighborhood patterns and the scattering of people into unfamiliar new groupings has increased the level of suspicion people feel toward one another.

DISORIENTATION

The people of Buffalo Creek are also clearly suffering from a prolonged sense of disorientation. It has often been noted that the survivors of a disaster are likely to be dazed and stunned, unable to locate themselves meaningfully in time and space. Time seems to stop for them; places and objects suddenly seem transitory. They have trouble finding stable points of reference in the surrounding terrain, both physical and human, to help fix their position and orient their behavior. All of this can be understood as a natural consequence of shock, but the people of Buffalo Creek seem to have continued to experience this sense of dislocation for months and even years after the crisis. "We find ourselves standing, not knowing exactly which way to go or where to turn," said one individual. Another survivor noted, "We feel like we're living in a strange and different place, even though it is just a few miles up Buffalo Creek from where we were."

Professional observers who have gone into the valley on medical or research errands have noted repeatedly how frequently the survivors seem to forget simple bits of everyday information—the names of close friends, their own telephone numbers, etc. People are often unable to locate themselves spatially, even when they are staring at fixed landmarks they have known all their lives. It is not at all uncommon for them to answer factual questions about time—their own age or their children's grade in school—as if history had indeed stopped on the date of the disaster. In general, people all over the valley live with a lasting sense of being out of place, disconnected, and torn loose from their moorings, and this feeling has far outlasted the initial trauma of the catastrophe itself.

People normally learn who they are and where they are by taking soundings from their fellows. As if employing a subtle form of radar, we probe other people in our immediate environment with looks, gestures, and words, hoping to learn something about ourselves from the signals we get in return. But when there are no reliable objects off of whom to bounce those exploratory probes, people have a hard time calculating where they stand in relation to the rest of the world. In a very real sense, they come to feel that they are not whole persons, not entirely human, because they do not know how to position themselves in a larger communal setting.

> Well, I just don't feel like the same person. I feel like I live in a different world. I don't have no home no more. I don't feel normal anymore. I mean, sometimes I just wonder if I'm a human being. I just feel like I don't have no friends in the world, nobody cares for me, nobody knows I even exist.

LOSS OF CONNECTION

A third manifestation of the disaster's psychosocial effects is a condition that might be described as loss of connection—a sense of separation from other people. For better or worse, the people of the hollow were deeply enmeshed in the tissues of their community; they drew their very being from them. When those tissues were stripped away by the disaster, people found themselves exposed and alone, suddenly dependent on their personal resources. The cruel fact is that many of the survivors proved to have few resources—not because they lacked the heart or the competence, but because they had spent so many years placing their abilities in the service of the larger community that they did not really know how to mobilize them for their own purposes.

Many people feel that they have lost meaningful connection with themselves. Much of their apparent former strength was actually the reflected strength of the community, and they are learning—to their very great discomfort—that they cannot maintain an enduring sense of self when separated from that larger tissue. They find that they are not very good at making individual decisions, getting along with others, or establishing themselves as separate persons in the absence of a supportive surround. "Lonesome" is a word many of them use, and they do not use it to mean the lack of human company. One woman who has moved to the center of a large neighboring town said of her new home: "It is like being all alone in the middle of a desert." A man who continued to live in his damaged home on Buffalo Creek said,

> Well, there is a difference in my condition. Like somebody being in a strange world with nobody around. You don't know nobody. You walk the floor or look for somebody you know to talk to, and you don't have nobody.

In addition, the inability of people to come to terms with their own individual isolation is counterpointed by an inability to relate to others on a one-to-one basis. Human relations along Buffalo Creek took their shape from the expectations that pressed in on them from all sides like a mold: they were regulated by the customs of the neighborhood, the ways of the community, and the traditions of the family. When that mold was stripped away, long-standing relationships seemed to disintegrate. This is true of everyday acquaintances, but it is doubly—and painfully—true of marriages. Wives and husbands discovered that they did not know how to nourish one another, make decisions, or even to engage in satisfactory conversations when the community was no longer there to provide a context and set a rhythm. There has been a sharp increase in the divorce rate, but that statistical index does not begin to express the difficulties the survivors have relating to their spouses. It is almost as if communal forces of one sort or another had knit family groups together by holding them in a kind of gravitational field, but when the forces of that field began to dissipate, family members became scattered like aimless individual particles. Each individual nurses his or her own hurts and tends to his or her own business. They

do not know how to care for one another or to coordinate emotionally, because the context that lent substance and meaning to their relationships has disappeared. Two survivors put it this way:

> Each person in the family is a loner now, a person alone. Each of us is fighting his own battles. We just don't seem to care for each other anymore.

> The family is not what they was. They're not the same people. I don't know how you'd put this, but before there was love in the home. But now it seems like each one is a different person, an individual by himself or herself, and there's just nothing there.

Finally, the difficulty people experience in sustaining warm relationships extends beyond marriages and families, out into the rest of the valley. In places like Buffalo Creek, relationships are part of the natural order—being inherited by birth or acquired by physical proximity—and the very idea of "making" friends or "forming" relationships is hard for these people to understand and harder still for them to achieve.

One result of all the problems I have described is that the community (what remains of it) seems to have lost its most significant quality—the power it gave people to care for one another in moments of need, to console one another in moments of distress, and to protect one another in moments of danger. In retrospect, it is apparent that the community was indeed stronger than the sum of its parts in this regard. When the people of Buffalo Creek were clustered together in the embrace of a community, they were capable of remarkable acts of generosity; when they tried to relate to one another as separate individuals, they found that they could no longer mobilize the energy to care. One woman summed it up in a phrase: "It seems like the caring part of our lives is gone."

CONCLUSIONS

To end with an oversimplified metaphor, I would suggest that the people of Buffalo Creek were accustomed to placing their individual energies and resources at the disposal of the larger collectivity—the communal store, as it were—and then drawing on those reserves when the demands of everyday life made this necessary. When the community more or less disappeared, as it did after the disaster, people found that they could not take advantage of the energies they once invested in that communal store. They found themselves almost empty of feeling, devoid of affection, and lacking all confidence and assurance. It is as if the cells had supplied raw energy to the whole body but did not have the means to convert that energy into usable personal resources once the body was no longer there to process it.

Children of Disaster: Clinical Observations at Buffalo Creek

BY C. JANET NEWMAN, M.D.

Most of the 224 children who were survivor-plaintiffs of the Buffalo Creek disaster were emotionally impaired by their experiences. The major factors contributing to this impairment were the child's developmental level at the time of the flood, his perceptions of the reactions of his family, and his direct exposures to the disaster. The author focuses on children under 12, describing their responses to fantasy-eliciting techniques and their observed behavior after the flood compared with developmental norms for their age and reports of their previous behavior. These children share a modified sense of reality, increased vulnerability to future stresses, altered senses of the power of the self, and early awareness of fragmentation and death. These factors could lead to "after-trauma" in later life if they cannot make the necessary adaptations and/or do not receive special help to deal with the traumas.

AS PART of the psychiatric evaluation of the survivors of the Buffalo Creek flood, 224 children were interviewed and evaluated; most were found to be significantly or severely emotionally impaired by their experiences during and after the flood. In this paper I will focus on children under the age of 12, using as a sample 11 of the children I assessed personally.

As has been described elsewhere in this section, the evaluation procedure began with an interview of the total family and proceeded to individual interviews. In interviews of mothers, outlines of each child's developmental history and functioning before and after the disaster were obtained. This information was passed on to the child psychiatrists in order to help us place each child in his parents' developmental perspective. Children were usually seen in their own rooms. They were encouraged to recall their own experiences of the flood; such expressions had often been submerged or inhibited amidst the outpourings of more vocal family members. The issues we discussed included past and present family life, personal feelings, school experiences, and the childrens' perceptions of future hopes, the nature of the disaster, and the meaning of the lawsuit.

We used such fantasy-eliciting techniques as "three wishes," "draw a person," and story telling. Preadolescent children were asked to draw a picture of the flood as they remembered it. Special educators from the Children's Psychiatric Center obtained school data to confirm or correct parental impressions of major losses of academic achievement that persisted long after the disaster.

The effects of the disaster on children can be attributed to three major factors: 1) their developmental level at the time of the disaster, 2) their perceptions of the family's reactions to the disaster, and 3) their direct exposures to the disaster. This paper illustrates each factor and examines their numerous interactions.

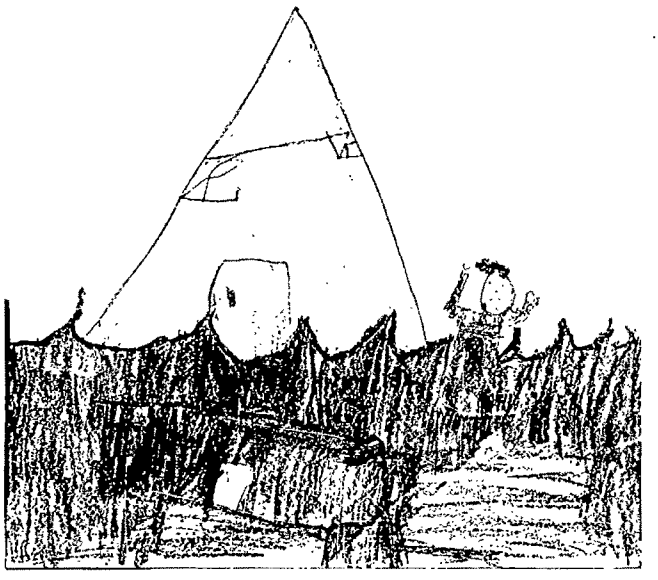
DEVELOPMENTAL LEVEL

The developmental factor will be illustrated by contrasting the clinical evidence gathered from 3 latency age children and 2 preschoolers. A depressed, hopeless, and guilt-ridden 11-year-old boy who had discovered human remains in his immediate environment after the flood drew a starkly realistic picture of a completely submerged trailer that contained two people screaming for help. A house above the trailer was half filled with water, and a panic-stricken figure tried to keep afloat amidst the waves and debris of the flood waters. Drawings by 2 younger children showed a possible symbolic meaning of mountains to Appalachian children, i.e., the provision of humanlike functions of cradling and life sustenance (this contrasts with Lifton and Olson's remarks about the "overall environment, including nature itself, as threatening and lethal" [1]). An 8-year-old boy with a chronic anxiety reaction drew the "house-mountain" depicted in figure 1 as life-saving compensation for his temporarily lost and helpless parents. The drawing represents a traumatic regression to a wishful merging of parental security into a house-mountain in a partially beneficent environment. A 7-year-old child also indicated security in nature in a picture of himself climbing a steep hill behind his mother and aunt. He drew a tree below him, saying, "This is a tree I can hang onto if I slide down." Nature offers support when human beings suddenly seem helpless. In Appalachia, the mountains represent not only slag heaps and flood threats but tangible, reassuring security.

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Dr. Newman is Director, Day Hospital Elementary Unit, Children's Psychiatric Center, 3140 Harvey Ave., Cincinnati, Ohio 45229, and Associate Professor of Child Psychiatry, Department of Psychiatry, University of Cincinnati College of Medicine.

FIGURE 1
An 8-Year-Old's Drawing of a "House-Mountain"



*Note that the trailer is destroyed, while the safe "mountain" acquires door and windows.

The experiences of 2 younger boys will be described to isolate developmental factors at the preschool level. Henry, who was 3 years old at the time of the disaster and 5 at the time of our interview, was the only son and favorite child of his mother's second marriage. He was the first to awaken on the morning of the flood. Looking out his window, he viewed the uncanny and perplexing sight of a house moving down the creek. Unsure whether this was real or a dream, he awakened his parents, conveying more cognitive bewilderment than fright or anxiety. He remembered saying, "Come and look!" The parents rose instantly and managed to evacuate the family to safety on high ground just in time.

Two years after the flood, his mother told us that Henry frequently slept in the same bed with her and loved to be rocked, although he had rarely needed this type of attention before the disaster. She reported that he often talked about the houses and cars that had floated by in the flood and how they "went boom!" I felt that because Henry was the first to awaken and, in a sense, rescued his family, they regarded him with special gratitude and admiration. During the family interview Henry was hyperactive—he was friendly but restless. In his individual interview, he recalled seeing the house going by his window and asking his parents to come and look. He denied being scared, but said, "I didn't like it." Henry also remembered seeing a screaming baby on top of an upside-down store that was floating downstream. He said, "I didn't like that either. I hated it."

Henry's flood picture started with a creek as an almost perfect circle. Then a curving line showed "how the creek goes here." A rounded object near the path of the flood conveyed its force; as he explained, "The flood threw this rock." Then Henry spontaneously drew a 3-sided rectangular but bottomless form nearby with "windows" for eyes, explaining that this was a person killed in the flood. Most children Henry's age, drawing their first human figures, use crude circles for heads or head-and-body combinations: in a precircle phase they use primitive scribble-strokes to indicate human figures. Henry had already manifested a capacity for drawing circles but had applied this skill only to his representation of the flood, using a bottomless rectangle with windows for eyes as a human figure. Such faces or human figures were interpreted as condensations of humans with buildings, stimulated by this 3-year-old's view of a peculiarly and perhaps awesomely floating house and other buildings, including the one the screaming baby was on. His flood experience started as he awakened from sleep, and sorting dream from waking perception and reality is typically difficult for young children. Developmentally, Henry was at a stage of tenuous differentiation of dream from reality and animate from inanimate objects, and motility is the first characteristic differentiating living from inanimate objects.

To summarize, Henry's drawing showed the human figure as dehumanized and fused with a seemingly animated building. The bottomlessness of the human face-figure suggests his lack of security, which was shown clinically by hyperactivity and an excessive need to be closer to his mother than he had been before. The circular flood moving huge rocks suggests the projection of superhuman powers to nature. His barely developed abilities to separate animate from inanimate and actuality from fantasy or dreams help to explain his current bewilderment, excessive anxiety, and hyperactivity. His favored position in the family and his role as "rescuer" have accentuated his sense of narcissistic omnipotence, which allays his underlying feelings of helplessness and anxiety. It is likely that his problems of immaturity, anxiety, and developmental deviations in cognition will become more evident when he starts school.

Peter was interviewed 2 years after the disaster, which occurred when he was 26 months old. His parents reported that he had been a happy baby, developing at normal rate. His father, a chronically disabled miner, described how the family scrambled up the side of the mountain behind their home and watched as their house was swept away and a nearby bridge crumbled from the force of the flood. A frantic passerby yelled to Peter's father to aid in the rescue of two children clinging to a floating mattress. As he ran to help, his wife screamed for him to come back. Realizing the danger, he ran back to his own children and led them to higher ground, carrying Peter on his chronically weak back.

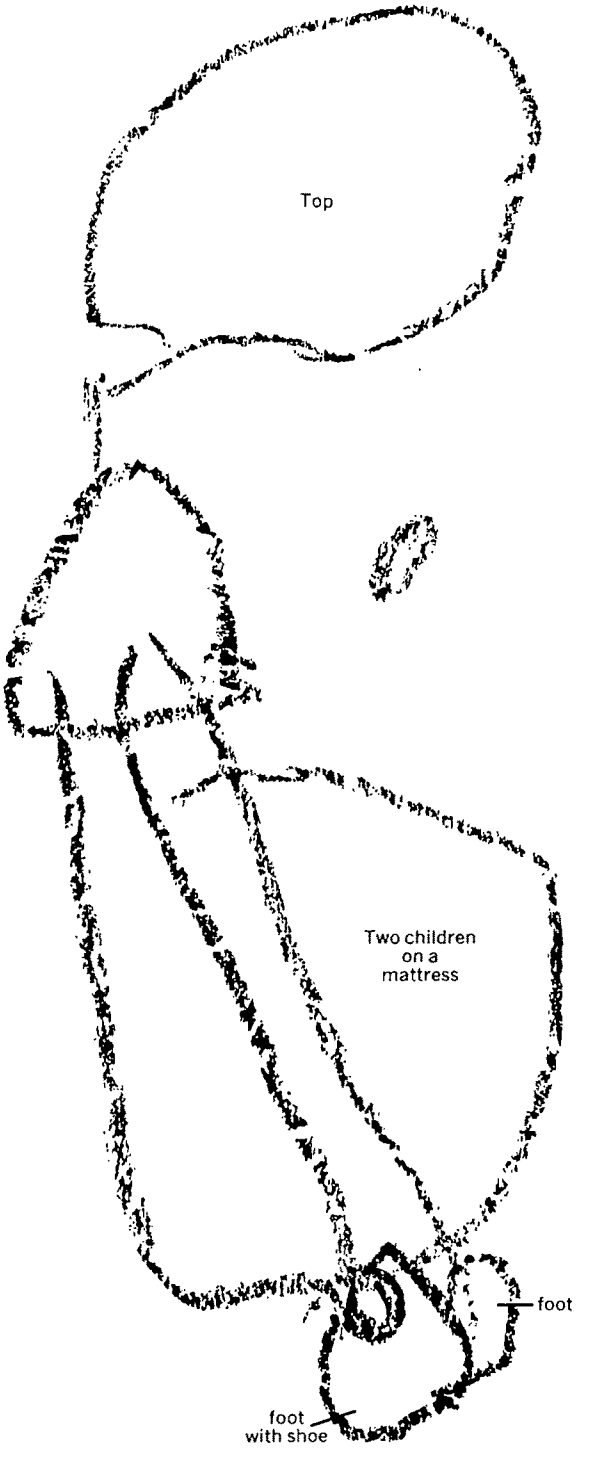
Since the disaster, Peter cannot take a bath without screaming, and he still wets the bed frequently and screams in his sleep. He gets mad easily and always

wants his own way. Peter's memories of the flood involved concerns over the safety of his friends and an older brother's loss of his best friend. He referred to the "two kids on a mattress" and worried aloud whether "their daddy, he might just not want them." This must have represented his own fears at the hands of his own father, who did not save all children, limiting his efforts to the rescue of his own family. When asked about troubles or worries he said, perhaps stoically, "I don't be sad, that's all." When asked what would make him happy he said, "I don't know, maybe if my daddy was handy."

Peter's response to the three-wishes question was touching and highly original and was probably related to a 4-year-old's determination to hold on to reality, with a resulting fear of pretending, even for a moment: "I don't wish," he said proudly. His drawings, made at age 4, about his flood experience when he was 2 years old, should remind the reader of Henry, who, although he was perfectly capable of drawing good circles, drew a person as a house with a rectangular bottomless face. Peter, although younger, is involved with deeper, more sophisticated, more human views of the disaster.

Peter's first flood picture included irregularly round or oval outlines of what he then chose to call windows—an "ugly" window, a "shoe" window, and a "big" window. (Windows with views of the creek became highly important for many families.) As an afterthought, Peter drew a longer shape and told me to write "two kids on a mattress in the creek" within it. I then said, "Let's do that one again on a bigger page." Peter drew a mattress and started to draw the 2 children in the form of lines. However, in the process, he appeared to convert the children into 2 legs (one barefoot and one with a shoe)[1] and then drew a body and a head labeled "top," creating a total human figure. He was influenced by the fact that his siblings were drawing people. Figure 2 is Peter's second drawing: the writing is that of the interviewer during the process of drawing and records Peter's words. This is a powerful condensation of a traumatic scene, combining his father's failure to save all of the endangered children, particularly those on the mattress. These 2 children became the 2 legs of the larger total human figure in the picture. As with Henry, we see a variety of serious developmental interferences and emotional distortions in the development of perception and cognition as manifested in body image concepts.

FIGURE 2
A 4-Year-Old's Picture of Two Lost Children Condensed into a Primitive Human Figure



REACTIONS BASED ON DIRECT FLOOD EXPERIENCES

Marie was the cute, articulate daughter of a strong father and a dominating hypertensive mother; she was 8 years old at the time of the disaster. During the flood Marie's mother bundled her in blankets and carried her to shelter, never allowing the child to see the acute stages of the flood.

During Marie's interview, her "draw-a-person" pic-

[1]Many children were barefoot or half barefoot in the escape from the flood and suffered frostbite.

ture was of smiling, childlike parents in flowered clothing. Her flood picture, drawn from hearsay, seems traumatic at first glance. However, the bubbly clouds she drew were duplicates of the floral prints of the father's shirt and the mother's skirt in the draw-a-person picture. The houses stood high above the languid flood, and the many bodies appeared mostly in cheerful upright positions. Only 2 small figures yelled, "Help!" Marie exemplifies a child reacting to maternal anxieties, reminding us of the children described by Anna Freud and Dorothy Burlingham in *War and Children* (2), who reacted far more strongly to maternal emotions than to bombs.

The major clue to the anxieties underlying the facade of pollyannaish denial was Marie's response to the Despert Bird Fable, which elicits a child's story of what a baby bird who can fly a little will do if a strong wind blows the family nest from the tree, scattering the mother and father and baby bird. Marie asked, "Were they all close together or were they far apart?" I then said, "What do you think?" Marie replied,

> Mother makes another nest, with twigs, on a stronger branch. The little bird grows up to have a family. Or, maybe the mommy bird might get sick or die, or a cat might eat her. Or maybe the little bird might get sick or poisoned. It might mistake weed-killer for seed. That *could* happen. Oh well, the little bird probably got old and then died.

This story reveals a rapid descent from superficial health into violent and even paranoid ideation, involving the death of both mother and baby by violence and poison. This rapid weakening of defenses reveals Marie's vulnerability to and identification with a chronically anxious mother, whose exacerbated anxieties she had been intimately exposed to in the apparent service of being protected herself.

Richard, who was 7½ years old at the time of the disaster, was the middle child of 3, born to mature parents. On the day of the disaster, as soon as the water level fell, Richard and his father searched for relatives. They were concerned about the safety of Richard's older sister, who had stayed overnight with a girl friend. The sight of the mutilated body of a boy Richard's age was shocking to both the child and his father. Richard was described as a changed boy since the flood, having become tense, nervous, talking little to his parents, and suffering from terrifying nightmares of someone coming back from the black water to take him to the spirit world. When interviewed, he said that he usually slept with a blackjack under his pillow.

Richard's flood picture conveyed a firm sense of reality, a strong sense of form combined with creative sensibility. He drew a truck carrying 5 bodies wrapped in sheets, set against a background of a burning slag pile and a house with a large chunk missing. The sky was overcast and it was raining. His draw-a-person picture, a clever, strutting, colorful comic book character drawn in profile, shows color, movement, and detail and indicates creativity and ego strength. Despite enduring strengths in peer relationships, good school performance, and basically warm family ties, Richard has a chronic traumatic anxiety reaction manifested by trembling hands, tension, inner tremulousness, difficulty sleeping, and nightmares. In contrast to Marie, who looks deceptively healthy and self-assured but whose reawakened inner problems stem from close ambivalent ties to a chronically anxious mother, Richard's symptoms represent more purely a chronic overt traumatic reaction to the disaster, in the context of considerable ego strength.

David, 7 years old during the flood and 9 when interviewed, was apparently well-adjusted before the disaster. Afterward his grades fell, he tended to keep to himself, and got into fights. His most severe symptoms, however, were crying in his sleep, sleep-talking (saying he wants to "get home again"), and somnambulism—he seems to be walking *out* of the house. Although others direct him back to bed, he does not wake up during these episodes but appears frightened. Since the disaster he has been wetting his bed several times a night, something he did not do previously.

In his interview he appeared attractive and cooperative but quiet and somber. He did not recall the content of his sleepwalking episodes, although he vividly remembers people screaming while they were drowning during the flood. David drew a bizarre person with a strange colorless face looking backward and a brightly multicolored body with feet pointing in the opposite direction from the face (figure 3). Diagnostically, he had symptoms of a traumatic neurosis with a dissociative-type hysterical neurosis (exemplified by his somnambulism) encapsulated within it. It seemed likely that his trancelike sleepwalking was a repetition of the original escape; this interpretation was supported by his grotesque drawing of a person whose profile, while colorless, had a fixed smile and slightly quizzical or puzzled eyes. Facial distortions and poor fit to the body are evident in the picture.

David's pathology was focused and severe. Sleepwalking usually occurs in stage 4 sleep when central nervous system motoric inhibition of REM dreams cannot occur (3). Certain types are called "somnambulistic trances" and may represent physical reenactments or abreactions of traumatic situations (4). David's bizarre picture suggests an unconscious connection between his sleepwalking state and his conscious imagery.

Marie has become subtly but severely traumatized through her direct relationship with a chronically anxious and flood-traumatized mother, while Richard and David's more conspicuous and overt traumatic reactions stem more directly from their flood experiences. The contrasts in the symptom choices of Richard and David are probably multiply related to their constitutional backgrounds, developmental experiences preceding the trauma, and the exact circumstances of the moment of greatest trauma each experienced in the disaster.

FIGURE 3
A 9-Year-Old's Drawing That Suggests a Link Between His Somnambulism and Conscious Imagery



OBSERVATIONS ON OTHER AGE GROUPS

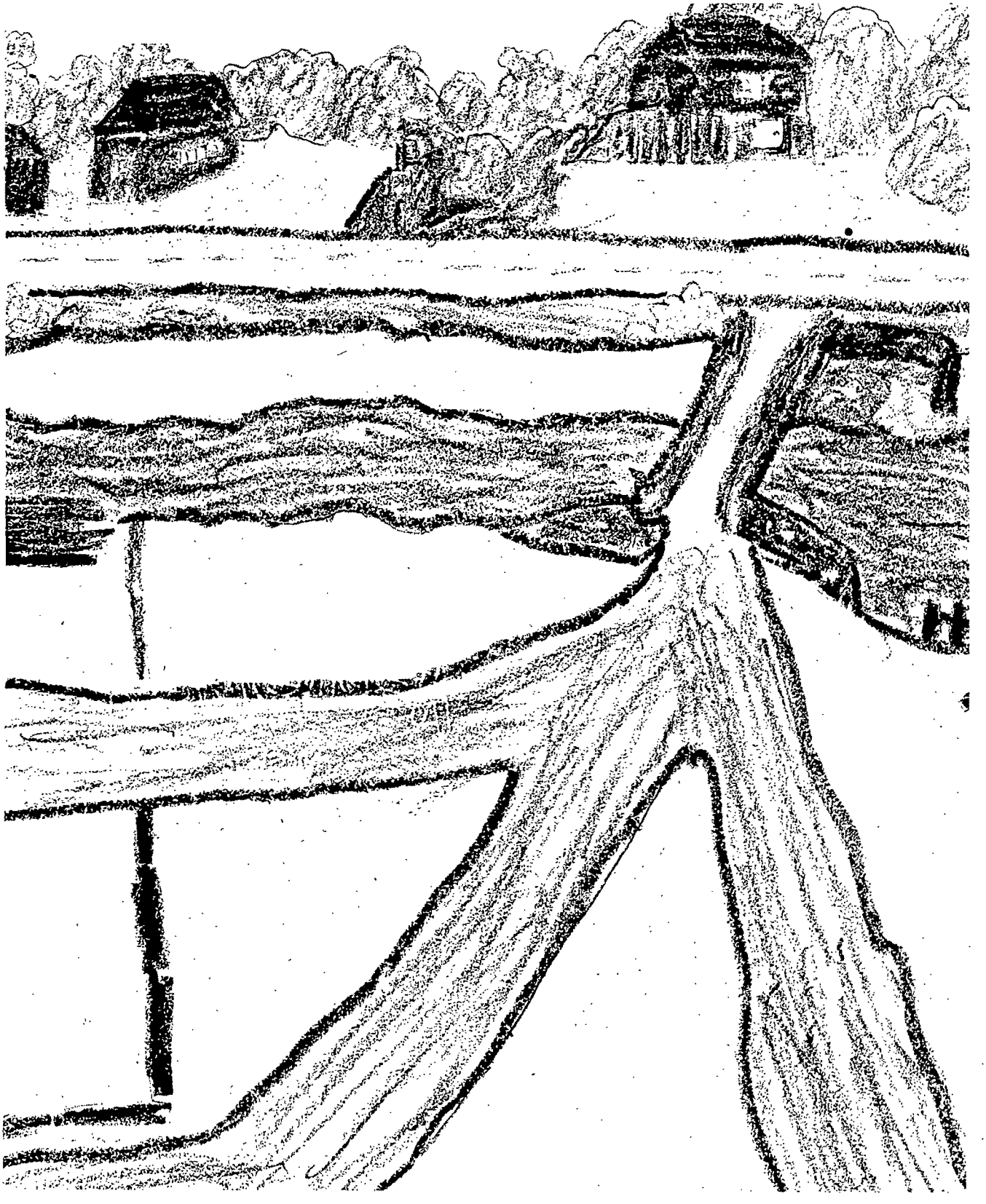
I have not mentioned another group of children, those who were in utero during the flood. Often, their preciousness lies in having survived the pregnancy of a frantic mother. Among their future stresses may be the never-ending tales or the silent allusions of the family about the disaster these children never experienced. The parents may see them as magically and profoundly linked with the flood. These children, as well as those born later (who will also feel left out, yet involved), will be unpredictably but importantly influenced by the catastrophe.

Although this paper has focused on preadolescent children, a few words must be said about adolescents' special vulnerabilities to the psychological effects of the disaster. Because the almost total community destruction, the loss of communality described elsewhere in this section by Dr. Kai Erikson, was so disruptive, especially to adolescents, they often had to choose between rebellious predelinquent behavior or compliant social withdrawal. They suffered deeply but privately when their parents broke down under stress. For example, in one family, the favored older brother, who had been the "good one" before the disaster, changed his behavior markedly—he missed 68 days of school, threatened the teaching staff, was suspended five times, and is currently on probation because of his behavior. At home he sat up at night apprehensively listening to rain or roared away on his motorcycle. However, his next younger brother continued to attend school regularly and made every effort to concentrate. The contrast between his very chaotic flood picture and a carefully drawn pink dove of peace that his teacher had praised as "best in the class" shows the range and conflict of his inner experiences, which he has internalized, but with unknown emotional strains.

Creative expressions emerged in many cases. Out of a highly disturbed large family living in two trailers in a state of chaos came a touching picture drawn by an 11-year-old boy (figure 4). Denying the turbulence of the flood, he drew an intellectually complex picture with excellent perspective that showed a trestle, road intersections, and a quiescent creek. He labeled one "the road to where we used to live." In the background, brightly colored idyllic homes in red, blue, and orange nestled among the woods on the hillside. There were no people in the picture, but there were possibilities for human reconstruction. The three dramatically split roads suggest important choices to be made, and the colorful homes in the background suggest hope.

Finally, some older children did follow the lawsuit and psychiatric interviews with great interest and sophistication. They recounted hopes of being lawyers and nurses, even though they were often having problems in their basic school courses. They wrote themes on safety regulations and dam construction as school projects and tried to master their experiences emotion-

FIGURE 4
A Hopeful, Creative Picture Drawn by an 11-Year-Old Child



ally and intellectually. They will never forget this experience, and they will be watchful of all the adults who have participated in it; they either idealize or are disillusioned with parents and other adults. When they grow up, they will watch the world closely. They will have learned enormously both in and out of school.

CONCLUSIONS

Children in traumatized families within a shattered community form their own theories of a disaster from their own reactions and their perceptions of the reactions of their parents and other adults. Their conceptions are also influenced by the social and legal processes associated with the disaster. All of these factors permanently affect their sense of self in growing up. The common heritage of most children of disaster is a modified sense of reality, increased vulnerability to future stresses, an altered sense of powers within the self, and a precocious awareness of fragmentation and death. In contrast to most of their parents, some of the children manifested clear and enduring evidence of hopefulness and creativity, despite obvious current limitations in their ability to achieve specific goals. Their sense of hope existed side-by-side with serious signs of developmental limitations and serious pathology. Indeed, the widening discrepancies between social sensitivities and academic achievement could lead to a severe "after-trauma" in later life. They would require unusual life adaptations or special help to respond constructively or creatively to the traumas they had undergone.

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Discussion of the Buffalo Creek Disaster: The Course of Psychic Trauma

BY LEO RANGELL, M.D.

The specific contribution of the psychiatrist to the team study of the human disaster at Buffalo Creek focuses on the course of psychic trauma. The initial violent intrusion by the flood waters was followed by a second phase of the traumatic cycle, the physical dislocation of the survivors, with disruption of their "ground" and "surround." During this long subsequent period the level of trauma did not recede but kept rising, although at a slower pace. Distant effects of the trauma may succeed the more immediate ones. The finite psychic space of the survivors is encroached upon by traumatic memories for an indefinite period of time, leaving fewer resources available for normal effective living. The absorption and merging of traumatic stimuli into a traumatophilia poses still another potential problem. The unprecedented legal decision as to the linear effects of psychic trauma on a succession of connected individuals will need further interdisciplinary clarification.

IN OFFERING OPINIONS on the overwhelming human experience of the Buffalo Creek disaster, the challenge is to separate our reactions of empathy and horror (which, as Lifton and Olson [1] have pointed out, were quickly and painfully shared by all mental health professionals who came to the valley after the disaster) from the potential contributions of our specific professional expertise. Toward this end, I will focus on the realm of psychic trauma (2), its nature and its effects, to complement the findings of the interdisciplinary team of sociologists, psychologists, attorneys, and others who took part in the evaluation of the effects of this experience on those who survived it.

The articles in this section range from the individual to the collective, from the child to the adult, and from the deepest inner effects to the widest outer consequences of this sudden, unassimilable disruption of man's relationship to his physical world. These studies address not only the inundation of psychic structures in a horizontal sense but also the longitudinal effects of the flood—the disturbed continuity with the past, the shattering of the present, and the inescapable portents for the future.

The work of the interdisciplinary evaluation team retained by the law firm representing the 625 survivor-plaintiffs to assess the psychological impact of this catastrophe has profound implications, reflected in its effects on the social decision-making process. The unprecedented legal decision, reported elsewhere in this section by Mr. Gerald Stern, permitted people who were not on the scene of an accident to be awarded reparations for the psychic damages they suffered. The principle acknowledged in this case could well shake all existing rules of the social order about the responsibility of man to man.

THE FIRST PHASE: PSYCHIC NUMBNESS

To turn to the central focus about which psychiatrists can reflect and from which other observations will stem, the Buffalo Creek flood was a violent intrusion into the peaceful psychic life of the community massively beyond the "average expectable environment" (3). This eventuality was not, however, completely unexpected—the people in the valley had long lived with this possibility and knew it could be prevented, but they said they had put it out of their minds. Actually, it had been put not out but deeply in, had become part of their living unconscious. It was in some ways like the earthquake situation in California but worse and more constant. The dam that finally broke physically existed just above the valley and was visible and ever present. Another difference, which added the makings of a latent inner eruption to the potential external occurrences, was that there were in the minds of valley residents people (the owners of the dam) who could and should have done something about the situation. Like the cave-in of a coal mine in a community that has always lived in dread of such an occurrence, the flood had been part of the mental as well as the physical geography of Buffalo Creek, a feared event buried in the minds of the people. The massive convulsion of the physical world that took place on February 26, 1972, was a mental imprint come true. It was a nightmare from which this time they did not awake.

The black waters that roared through Buffalo Creek

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Dr. Rangell is Clinical Professor of Psychiatry, University of California, Los Angeles, School of Medicine, and immediate Past President of the International Psychoanalytic Association. Address reprint requests to Dr. Rangell at 456 North Carmelina Ave., Los Angeles, Calif. 90049.

valley when the fantasied, feared, and repressed event became reality could be said to have flooded the egos of those who lived through the disaster. All control functions were overrun by the sudden influx; it was a maximum dosage per unit time, a psychological overkill. The result was universal and with a common base to all, the traumatic state, the condition of psychic helplessness that the anxiety signal presages and that all of the ego's defenses constantly work to prevent (4, 5). This was the phase of psychic numbness observed in all of the studies of the survivors. The goal was merely to live through it, to survive. Each individual held on to whoever or whatever was left. "Psychological conservatism," which Drs. Titchener and Kapp describe in "Family and Character Change at Buffalo Creek," served to reduce excitation; no further stimuli were wanted.

This stage of psychic numbness, of apathy, withdrawal, and sluggishness, was still visible when the evaluation teams arrived in Buffalo Creek 2 years later. Some of the observers were surprised that the traumatic neurosis was still visible, but what evidence is there to assume that the residual or even basic effects of so massive a trauma would not last a lifetime? Psychological testing of survivors of the Nazi holocaust has shown that after 30 years they still show such lasting effects as impairment in perceptual-cognitive functioning, withdrawal from objects, inability to sustain close relationships, and other subtle and overt sequelae.

While the legal experts perhaps needed to be concerned about the claim of preexisting states, I would prefer to maintain clarity and not to have to use the word "impairment," to blur the fact that there *was* loss, injury, and illness. The effects of the disaster were as ravaging as an epidemic of typhus or plague, which is similarly visited upon a city from without, and the preexisting vulnerabilities in the hosts to receive the invading organisms are not an issue. The traumatic neuroses overshadowed psychoneuroses. I do not mean to say that there were no preexisting psychoneuroses, but rather that they no longer had a chance to assert themselves. This is an issue that has plagued every psychiatrist who has served in the military. Combat neuroses in their acute phases are more uniform than different in their presenting syndromes; only later can individual differences reappear and assert themselves again. During the years to come, as normalcy returns to the Buffalo Creek survivors, they will have the luxury of becoming individually neurotic again.

THE SECOND PHASE: "GROUND" AND "SURROUND"

What happened to this community in terms of incoming traumatic stimuli did not stop with the cataclysmic events of that Saturday morning. As disorienting and time-stopping as the flood was, it was only the first phase. An individual who suffers a loss, however shattering, generally returns to his home to start the long and difficult process of repair. His "ground," the background into which the self can merge, is the base for his security, the source of the nurturing supplies that sustain the processes of reparation. This was not possible in Buffalo Creek. The dazed survivors were returned not to their familiar ground but to new and strange surroundings. This may have been the only possible course, but it was still depriving and further threatening. Rather than nurturance, the survivors continued to face challenges, for a long time and during a raw and vulnerable state.

In this double and almost death-dealing blow, these survivors repeated the experiences of other holocausts known to our generation. Those who survived and escaped from Nazi Germany wandered into new lands. The survivors of the atom bomb returned to craters where their homes had been. Many South Asians have had to leave their land after having left their dead. In all of these cases, the survivors' earth is also gone after their fellow humans have disappeared.

Years ago, writing at another level and about more comfortable aspects of human troubles, I described "attachment to ground" as the psychic prerequisite for the maintenance of the social state of poise (6, 7). The opposite condition, a wavering hold on one's surrounding psychic ground, results in a basic anxiety with the threat of crumbling and even annihilation of the self. This is the source of the primitive anxiety that people feel at the first threatening tremor of an earthquake. In the more mundane case of seasickness, the fear has been said to be not that one will die, but that one might not. Underlying both of these disturbing states is an elemental anxiety that stems from a disorientation in the relationship of the organism to the earth under its feet.

In the course of ontogenetic development, this relationship extends from the ground beneath one to the space around him, to people, institutions, atmosphere, and the culture. This is the common background in the phenomena described by Freud in the oral stage (8), in the attachment studies of Bowlby (9), in Mahler's contributions on separation-individuation (10), the effects of motherlessness on the primates studied by Harlow (11), and in the natural experiments on human infants studied and documented by Spitz in cases of marasmus resulting from early and massive deprivation (12).

Not only did the ground of the Buffalo Creek valley literally sink beneath the feet of its inhabitants, initiating the most primeval regression man can experience, but when the survivors reached solid high ground, they were again deposited on an insecure terrain, into a social "envelope,"—an intriguing term used by Dr. Kai Erikson—of unfamiliar space. The trauma thus did not end, it merely changed. There are strain traumata (13), shock traumata (14), cumulative traumata (15), and sudden overwhelming disruptive events. The stimulus barrier can be bent as well as rent (16). At this point, the trauma of the survivors changed from

sudden to cumulative. The flood receded, but the level of trauma did not; rather, it kept rising, although at a slower pace.

This was the phase studied intensively by Dr. Kai Erikson, described in this section from the sociological standpoint as a loss of communality, the social tissue that binds people together. While this loss was universal, we should not overlook the fact that it was an individual trauma as well. Just as the original traumatic state, although universally shared, was individual in suffering, so was it with this second phase of the traumatic cycle. The change from the familiar to a strange surround during the period when rest and nurture were needed superseded the initial trauma and prolonged and compounded its effects in each survivor.

THE THIRD PHASE: FUTURE EFFECTS OF THE TRAUMA

The articles presented in this section survey the effects of the disaster to date. Less measurable are the future effects, especially those which are more subtle and internal. Are the children Dr. Newman interviewed who are now getting along well in school less vulnerable to future stresses than those who are currently more turbulent and disturbed? Or is the reverse the case? Only long-term longitudinal studies (which are not likely to be practical or feasible) would answer this and similar questions.

There are subtle and far-reaching issues facing the survivors. In spite of the vastness of the unconscious, psychic space is limited. There is room and time in any individual psyche for only a limited amount of cognitive ideation and a finite number of memories, fantasies, and accompanying affects. The product of such space and time comprises the psychic life of an individual, the amount already spent and the amount still left. Mourning is a model of such an occupation of psychic space, a paradigm of how obsessive thoughts and memories related to psychic work that needs to be done crowd and consume the psychic capacity. Traumatic memories of any kind encroach on this psychic time-space and reduce its available quantity; this is why psychic traumata age people.

I have been treating a woman in her mid-seventies and have discovered that her apparent senility is due not to an organic aging process but to the repression of decades of a traumatic life. She had told herself—she brought this out with clarity through her foggy memory in sessions that had a hypnotic quality—that she did not want to remember any part of her married life of close to 50 years. The volume and intensity of the traumatic memories being repressed left her almost no room for normal living. She had by now assumed the posture, both mentally and physically, of a diffuse cortical atrophy, without evidence, either neurological or radiological, of any organic syndrome nor even convincingly of cerebrovascular disease. She was like a young, acute, traumatic amnesia, except that this was chronic, old, and massive. Her mental state undulated dramatically with the emergence and rerepression of forbidden thoughts. This poignant clinical experience has made me wonder about the general psychopathology of "old age."

How much space will the Buffalo Creek experience occupy in the minds of the survivors in their future lives? We routinely treat patients who react to a deprived childhood by sacrificing a certain percentage of their psychic lives. I have treated a patient who has occupied perhaps a quarter or a third of her free associations with obsessive preoccupation over her screaming mother; her thoughts are similarly occupied outside of the analysis. Another of my patients has been unable to enjoy his current life because of the constant crowding of his psychic space by the coalesced memories of the threats of castration that pervaded his tortured childhood. I have pointed out elsewhere (17) the role of such chronic traumata in producing the cacophony of human relationships in ordinary life.

These situations represent fairly common developmental traumata. How much more of a role do cataclysmic traumata like the Buffalo Creek disaster play? What will be the long-term effects of the vivid, massive "death imprint" described by Lifton and Olson (1)? What will be the effects on children in whom death anxiety has been violently added to the normal anxieties of separation and castration? It seems likely to me that their memories will repeat the accumulated traumata over time like a long-acting timed-release capsule.

There was an element in this disaster that is not present in truly natural catastrophic events, which serves to explain further why the "Buffalo Creek syndrome" is not limited to reactions to external events, but rather reflects added internal idiosyncratic forces. I am referring to the human element, the thought and the accusation that this horrible occurrence could have been prevented. Unlike a natural disaster such as a tornado, where inanimate forces of nature are solely responsible, the human object was involved in the Buffalo Creek Flood, which arouses impulses of aggression and retaliation. Channels for discharging these impulses do not keep pace with the amount and quality of the impulses aroused. The ego is bombarded from two directions, and feelings of rage, impotence, anxiety, guilt, and depression are added to the usual responses to disaster.

The more external normalcy returns, the more will traumatic neuroses and psychoneuroses be in a reciprocal relationship to each other. The residual trauma will stimulate individual neuroses, and latent neuroses will feed upon and perpetuate the traumatic state. Such restitutional movements are already evident in the survivors and will increase with passing time. Phobias, obsessions and depressions, and private anxieties and conflicts have already been noted by various observers, and survivors' dreams are beginning to reveal their predisaster concerns.

There are other more subtle unknowns to cloud the future. What happens when a traumatic effect merges

over time into a traumatophilia? Such an outcome can represent a repetition compulsion not in the service of mastery but to satisfy a sense of guilt or a need for punishment, a trauma that is absorbed and utilized by the psychic forces "beyond the pleasure principle" (18). Or what will be the result when the pleasure formulae or safety mechanisims themselves become altered and individually fashioned as a result of the traumatic experience? I am reminded of a patient who was traumatically raped and now finds her husband and all other men to whom she turns passive and weak. Or a patient who, from a traumatic rejection in her first love, has come to no longer believe in love. Another patient, similarly hurt, now feels "I'll never again have a best friend." What will be the effects of the life-threatening insult at Buffalo Creek, seen by the survivors as a result of neglect by people in authority, on trust, love, and object relations? One can hardly begin to tell, but one can be prepared so as not to be surprised.

In surveying this event and the reports that have been presented in this section, we should not overlook the effects of the studies themselves on the 625 survivor-plaintiffs evaluated. Aside from the legal result, the interest displayed by caring individuals from the society outside the valley probably introduced a therapeutic influence, however circumscribed. This influence might be compared to the effects on a therapeutic ward of the mere announcement of a program of treatment. However, there may also be negative effects: divisiveness has been introduced in the valley. Just as the untreated "control ward" suffers by comparison with the therapeutic community, those survivors who were not among the litigants may feel left out and discriminated against.

While an important and unprecedented legal decision has been achieved that greatly extends the definition of psychic trauma following an external event, the full implications of the human phenomenon described in this section cannot be estimated. Anyone who is lost, hurt, or otherwise affected under traumatic circumstances affects others in an endless chain that is attenuated only by emotional distance. It would be illusory to believe that it is within our power or professional expertise to accurately describe ethical guidelines for the rectification of the linear progress of traumatic effects. I recently knew of an elderly couple who were being displaced from their home for the building of a federal project. During the process, the husband, distraught over the dislocation, suffered a fatal heart attack. What can we say or what should we do about the effects on his wife? Or the children? Or a chain of others? There are more questions than we can answer. We must work side by side with the law, with sociology, philosophy, and all thinking and feeling people. No one or no group has a corner on ethics or on wisdom.

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Carbon Monoxide Encephalopathy: Need for Appropriate Treatment

BY ROY GINSBURG, M.D., AND JOHN ROMANO, M.D.

The authors describe severe psychiatric and neurological sequelae in a patient who suffered carbon monoxide poisoning as a result of a suicide attempt. A review of the literature revealed that 15 to 40% of survivors of carbon monoxide poisoning develop neuropsychiatric symptoms, often following a period of apparent recovery. The authors advocate an aggressive treatment approach to carbon monoxide poisoning, emphasize the diagnostic value of extensive laboratory testing, and suggest that 2 to 4 weeks of bedrest may prevent delayed neuropsychiatric sequelae.

RECENT OBSERVATION of a patient with carbon monoxide poisoning drew our attention to the great variety of neurological and psychiatric symptoms in this disorder and to the lack of clarity concerning its clinical course and treatment. This paper describes the clinical course of a patient with carbon monoxide encephalopathy, reviews some of the major findings from the literature, and suggests some new approaches to treatment.

CASE REPORT

The patient, a 59-year-old housewife of Italian extraction, became depressed in June of 1973 and tried to commit suicide by car exhaust inhalation. A neighbor who was a registered nurse investigated within an hour and found the patient cyanotic and unresponsive. She was taken to the nearest hospital emergency room, where she was stuporous but not comatose, and was not "cherry red." Neurological examination was unremarkable, and a carboxyhemoglobin level was not obtained. An EEG and brain scan were within normal limits. There was no history of neurological or psychiatric illness.

After admission the patient became more alert but appeared depressed and could not concentrate. She was given three electroconvulsive treatments within a week. At first the patient seemed less depressed, but then she became increasingly confused and withdrawn, refused to eat, and developed stereotyped picking movements. Three more ECT treatments were given 3 weeks after admission, but the patient's confusion increased. Because of the unclear nature of her illness, she was transferred to Strong Memorial Hospital for further evaluation.

A physical examination on admission revealed a mute, uncooperative patient with intermittent posturing. She responded with body movements to some commands but would not open her eyes or talk. The only positive physical or neurological finding was a bizarre gait; the patient would stand in place and make small shuffling movements with her feet while moving her body in a clockwise rotation.

Laboratory tests revealed that the patient's hematocrit was 35% and the hemoglobin revealed beta-thalassemia. Electrolyte levels, calcium, phosphorous, blood urea nitrogen, serum glutamic oxalacetic transaminase, lactic dehydrogenase, alkaline phosphatase, creatinine, total protein, albumin, glucose, uric acid, urinalysis, serology for syphilis, T-4 by Murphy Pattee, brain scan, and skull X-rays were all within normal limits. A lumbar puncture revealed normal opening and closing pressures and no cells; cerebrospinal fluid protein and glucose levels were within normal limits. Electroencephalogram on admission and repeated five times since to the present revealed persistent symmetrical monorhythmic three-cycle per second activity which was only present during drowsiness.

The patient spent most of her 4 months in the hospital on the floor, sitting, rocking, and staring blankly. She occasionally responded by giving her name in a faint whisper but otherwise would not talk to the nurses or her physicians. She often defecated in bed, and was sometimes physically aggres-

When this work was done the authors were with the Department of Psychiatry, Strong Memorial Hospital, University of Rochester School of Medicine and Dentistry, Rochester, N.Y., where Dr. Ginsburg was Associate Resident and U.S. Public Health Service Fellow in Psychiatry, and Dr. Romano is currently Distinguished University Professor of Psychiatry. Dr. Ginsburg is now a Grant Foundation Fellow in Psychopharmacology, Department of Psychiatry and Behavioral Science, Stanford University, Palo Alto, Calif. Address reprint requests to Dr. Romano at the Department of Psychiatry, University of Rochester, Rochester, N.Y. 14620.

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sive. An interview with intravenous sodium amytal revealed that the patient was oriented for time, place, and person, and that she could talk about her depression and of her bizarre behavior as being like "something out of a book." After the first amytal interview, the patient was able to talk to the nursing staff, dress herself, and come to the dining room to eat. However, by the next morning she was again mute and lying in bed. For a period of 3 months the patient was given frequent amytal interviews and these were, with some exceptions, the only times she would talk. Treatment with trifluoperazine and then chlorpromazine in high doses was tried without effect and then discontinued.

After 4 months without improvement, the patient was transferred to the Rochester Psychiatric Center for long-term care. Since then the patient's clinical course has not changed significantly. She has generally remained mute, but when she does speak she confabulates. It became clear to the staff that she had considerable difficulty remembering recent events. Repeated neurological examination revealed bilateral tendon hyperreflexia, mask-like facies, and a parkinsonian gait.

LITERATURE REVIEW

The toxic effects of carbon monoxide (CO) have been known for centuries. In the past, household gas containing CO was the principal source of both accidental and suicidal ingestion. With the increasing use of natural gas and electrical heating this type of exposure has diminished. However, the development of the gasoline-burning internal combustion engine, which produces exhaust gases containing between 5 and 9% CO, stimulated an increased use of this gas as a suicidal agent (1).

The incidence of nervous and mental sequelae following CO poisoning was first reported to be 0.8% in 1936 (2). More recent reports describe a remarkable increase in the incidence of aftereffects to between 10–30% (3–6). Between 60 and 75% of patients with carbon monoxide poisoning survive the initial trauma, and 15–40% of those who survive have neuropsychiatric sequelae (4–6). Approximately 10–30% of patients develop neuropsychiatric signs 1 to 3 weeks after exposure (2, 6).

CO acts by displacing oxygen from the hemoglobin molecule and shifting the oxygen-hemoglobin dissociation curve to the left, thus increasing the binding of oxygen and hemoglobin. It displaces oxygen because the affinity of hemoglobin is 200 to 290 times greater for CO than for oxygen. A small quantity of CO can reversibly inactivate a substantial percentage of the oxygen-carrying capacity of the blood. The significance of a small amount of carboxyhemoglobin concentration is seen in the tremendous reduction this has on arterial oxygen tension (P_{O2}). For example, a carboxyhemoglobin concentration of 9% may be equivalent to a 30% reduction of hemoglobin or a 46% reduction of arterial P_{O2} (7). This shift substantially decreases the oxygen tension supply to the tissues (8).

The development of neurological signs has not been correlated, either clinically (1, 6, 9) or in experimental work (10), with the amount and length of exposure to carbon monoxide or the carboxyhemoglobin level. However, there may be a gross correlation between the patient's level of responsiveness on admission to the hospital and development of neuropsychiatric sequelae (4, 5). The half-time of excretion for low concentrations is from 2 to 4 hours (8); higher concentrations may take 7 to 10 days to be completely eliminated (11). CO in concentrations as low as 50 parts per million can produce notable effects on mentation (8). The inhalation of a 1% concentration is usually fatal to a resting man in 30 to 40 minutes and to a working man in 10 minutes. A concentration of 0.01% produces light headache and 0.2% produces coma. Children, who have a higher respiratory rate, die more rapidly than adults (9).

The entire range of focal and general neurological and psychiatric syndromes has been reproduced by carbon monoxide (2, 5, 6, 9, 12–17). The "cherry red" color traditionally associated with CO poisoning is actually uncommon; in one series it was found in only 6% of patients (3). Patients presenting in the emergency room will often have nausea and, depending on the degree of cardiovascular competence, may also have hypotension, metabolic acidosis, and angina pectoris (18). The first neurological symptom to appear is frontal headache (19). The headache is often a delayed phenomenon; thus, lethal concentrations of CO may not produce any warning symptoms. Prompt administration of oxygen before the headache becomes intense usually abolishes the pain (19).

Often the fluctuating symptoms from CO mimic those of psychotic depression or of hysteria (5, 6, 12, 20). In particular the rapid changing of apraxias, agnosias, astasia-abasia, and the amnestic-confabulatory state often contributes to a false diagnosis of hysteria. It is also reasonable to assume that "hysterical" defense mechanisms may be used by patients who realize that they are damaged. Weinstein and Kahn (21) have described many patients with severe illness who used rather bizarre denial mechanisms rather than face the overwhelming task of confronting their disability.

Recovery rates of those patients who do not relapse vary from several days to as long as 8 months (5). As would be expected, the highest cerebral functions recover last.

Delayed neurological reactions after carbon monoxide poisoning have been of increasing concern. Patients often have negative neurological and psychiatric examinations within several days after their original exposure and are either discharged from the hospital or allow to become ambulatory. Plum and associates (20) stated that "anoxia is usually severe; most patients are in deep coma when found but awaken within 24 hours. Nearly all patients resume full activities in 4 or 5 days." The patient usually does well for between 2 to 21 days and then can develop the whole spectrum of neuropsychiatric signs and symptoms. Deterioration may either progress to coma or death or be-

come arrested at any point. Some patients have a second recovery period which can lead to full health (20). There is no way to distinguish those patients who will recover from those who will relapse.

Laboratory tests can be of some value in cases of CO poisoning. Initial screening for toxic agents can rule out the presence of central nervous system depressants, which have been found in as many as 20% of CO ingestions (3). Electrocardiograms can reveal inverted T-waves and ST wave depression (6, 8). Cerebrospinal fluid pressure may occasionally be elevated or a few lymphocytes may be seen (22). Pneumoencephalogram (in patients with marked damage) shows enlarged ventricles and cortical atrophy. Over 90% of patients have electroencephalogram abnormalities, consisting of slow waves (2 to 5 cycles per second) of low voltage, frequently with a frontal preponderance (10, 23–26). In general, the severer the EEG changes, the severer are the sequelae. Patients with frontal dominance most often show general and focal neurological signs and dementia. Improvements in EEG findings parallel the patient's clinical condition (26). Sleep EEGs may predict improvement; patients who have the most REM sleep usually have the best clinical course (27).

The pathological effects of CO poisoning are present in almost all organs of patients who die from acute intoxication. However, the most important changes occur in the brain, where edema, capillary and venous dilatation, and hemorrhagic necrosis are acute (6, 9, 20, 28). These lesions are similar to those resulting from cardiac or respiratory arrest, hypoglycemia, or cyanide poisoning (5, 11).

Later pathological changes include demyelination of the white matter (anoxic leucoencephalopathy) in contrast to the usual predominance of lesions in the gray matter during the early stages (6, 9). The demyelination is often speckled and gives a "moth-eaten" appearance. The gray matter regions of the brain most commonly involved are Ammon's Horn; the part of the cortex situated below the cerebral sulci, particularly levels II, III, and IV; the pallidum; the thalamus; and the Purkinje cells of the cerebellar cortex. This predictable distribution of anoxic lesions is not consistent with the tremendously varied and unpredictable clinical constellation.

Three factors have been persistently identified in the production of anoxic lesions from carbon monoxide: hypoxidosis, edema, and circulatory disorders (10, 28, 29). The edema is thought to occur as a result of anoxic lesions to the vascular wall, and it brings about not only changes in the nerve and glial cells but also intracranial hypertension and compression of various vessels. Circulatory disorders are thought to arise from hypotension because of decreased output from the left ventricle (10), increased venous pressure with poor venous drainage because of right ventricular malfunction (29), and possible obstruction of small blood vessels by swelling of the endothelium or vasospasm (30). The need of white matter for oxygen is five times less than that of gray matter and therefore it is remarkable that the white matter may show the more extensive lesions (30). This has been explained by the hypothesis that white matter may be more susceptible to anoxia and edema because of a less redundant arterial supply. Lesions may occur more frequently in the subcortical white matter because there are fewer small vessel anastomoses than in deeper cerebral white matter (10). The size of the white matter lesions and the neurological signs have been correlated in experiments with the degree of metabolic acidosis and systolic hypertension but not with the extent of hypoxia (10).

CASE DISCUSSION AND TREATMENT RECOMMENDATIONS

Our patient presented a very confusing clinical picture. Although carbon monoxide poisoning remained the primary diagnosis, hysterical psychosis, schizophrenia, and psychotic depression were also considered as possible diagnoses. The diagnosis was difficult because of the patient's bizarre behavior, the wide and frequent clinical fluctuations, and her apparent lack of dementia under amytal sedation. However, the course of severe dementia over several years, the amnestic-confabulatory state, mutism, parkinsonian facies and gait, hyperreflexia, and the slowing on the EEG have implicated carbon monoxide poisoning as the diagnosis.

We suspect that some realization of her damaged state led the patient to increase her bizarre behavior to assure herself that she still had some control over her actions. This bizarre behavior, which disappeared during amytal interviews, is consistent with both the hysterical and denying defense mechanisms discussed by Weinstein and Kahn (21). Consequently, amytal interviews, which eliminated the hysterical aspects, were not helpful in diagnosis.

A severe depression led to our patient's carbon monoxide ingestion. Her hematocrit of 35% secondary to beta-thalassemia may have added to her susceptibility to CO. She initially improved, and was given six ECT treatments for depression. We cannot predict what her subsequent course would have been without ECT, but the increased metabolic demands of convulsive activity on the central nervous system may be related to the delayed onset of anoxic leucoencephalopathy (20, 28, 31).

Because many physicians are unfamiliar with this disorder, patients with carbon monoxide poisoning are often not treated as the data above indicate they should be. We recommend the following regimen:

1. When the patient is first seen, blood should be drawn for a carboxyhemoglobin level. Although the carboxyhemoglobin level does not correlate consistently with eventual neuropsychiatric deficits, it is useful as a guide to determine the extent of exposure because clearing is relatively slow.

2. Because of the high incidence of associated intake

of other drugs, blood and urine should be studied to determine if central nervous system depressants are present.

3. Since acidosis frequently accompanies carbon monoxide poisoning, arterial blood gases should be determined. The metabolic state of the central nervous system lags several hours behind that of the circulatory system; thus, acidosis that is not severe in the peripheral vascular system may be significant in the central nervous system, and treatment with bicarbonate may be indicated.

4. Electrocardiograms should be obtained, and patients with abnormal EKGs should be monitored for several days. The incidence of neuropsychiatric complications is higher in older patients with decreased cardiac reserve. Consequently hypotension and arrhythmias should be actively treated.

5. All patients should be treated with hyperbaric oxygenation if possible.

6. Tranquilizing drugs that decrease REM sleep should be avoided.

7. Because of the occurrence of edema, treatment with an agent such as dexamethasone or mannitol should be considered (26). Normal cerebrospinal fluid pressures cannot rule out central nervous system edema in CO poisoning.

8. Bedrest for 2 to 4 weeks seems necessary to avoid delayed onset of neuropsychiatric symptoms. Although the exact etiology of this delayed onset is not known, there is a clear correlation between the onset of delayed symptoms and increased patient activity. Any activity that involves decreased perfusion of the central nervous system or increased central nervous system metabolic activity should be avoided (28). ECT should be particularly avoided following exposure to carbon monoxide (31).

Although these measures are more extensive than those currently used in most hospitals, we think they are medically indicated and economically feasible. The cost of long-term psychiatric hospitalization for patients with permanent neuropsychiatric sequelae of CO damage is much greater than the cost of prompt and appropriate management of patients exposed to carbon monoxide. Because the pathology and pathophysiology of all types of anoxic encephalopathy appear to be similar to carbon monoxide poisoning, it may be worth considering extending this treatment regimen to all patients who have experienced anoxic episodes.

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Psychiatric Presentations of Seizure Disorders

BY JOHN LAWALL, M.D.

Seizure disorders may present with signs and symptoms that are usually associated with psychiatric disorders. Physicians must be alert to this possibility; they should use careful history taking in conjunction with EEGs to avoid misdiagnosing certain cases of psychomotor, petit mal, and grand mal epilepsy.

ONLY FAIRLY recently has epilepsy been recognized as a group of disorders that are primarily organic in origin. Although paroxysmal brain discharges may disturb affect, thinking, and behavior, unusual symptoms of epilepsy may be thought to represent so-called functional disorders. An automatic assumption that bizarre symptoms indicate psychiatric disorders or a narrow view of the manifestations of seizure disorders may lead to misdiagnosis, especially in emergency rooms, crisis centers, and similar situations.

The primary care physician and the psychiatrist must be alert to the possibility of psychiatric presentations of seizure disorders and take appropriate steps to ensure accurate diagnosis. Even though pressure may exist in some settings to forgo seemingly excessive lab tests and to leave physical examinations to other physicians, our patients may benefit if we as psychiatrists use our neurologic expertise. The incidence of misdiagnosis of seizure disorders is probably small, but to the individual patient the significance is great.

Although the incidence of psychiatric disorders is greater in epileptics than in the general population (1, 2), my concern here is with those symptoms which occur as part of the basic seizure disorder. Grand mal, psychomotor, and petit mal epilepsy are the seizure types I will discuss, although it is conceivable that other seizure types could also be mistaken for psychiatric disorders.

PSYCHOMOTOR EPILEPSY

Psychomotor epilepsy is perhaps the quintessential example of a disorder on the borderline between psychiatry and neurology. It may manifest in such symptoms as laughter (gelastic epilepsy), hallucinations, coprolalia, assaultive behavior, hypo- and hypersexuality, and hypergraphia, among others. Occasionally a picture suggesting a full-blown psychosis may be seen, especially if the aura is complex and vivid. Flor-Henry (3) has characterized the aura as analogous to a transient psychosis. Déjà-vu, jamais-vu, depersonalization, derealization, paranoia, changes in mood, and hallucinations may occur in an organized fashion during the aura. It has been maintained that psychomotor epilepsy predisposes one to schizophreniform psychosis (2, 3).

Complex behavioral syndromes have also been reported as equivalents of psychomotor epilepsy. Abdominal epilepsy (4), characterized by abdominal discomfort, nausea, vomiting, and postictal confusion, is one such syndrome (4). According to Babb and Eckman (4), such patients are often labeled neurotic. Twilight states with complex automatisms have also been reported. In the case reported by Escueta (5), confusion (with some responsiveness to the environment), automatisms, and amnesia were accompanied by 1.5 to 4 cps high-amplitude EEG tracings and represented a form of psychomotor status epilepticus.

GRAND MAL EPILEPSY

Grand mal epilepsy is somewhat less dramatic from a psychiatric standpoint, but it can still present with psychiatric symptoms. Confusional states, psychotic states, and prolonged twilight states may occur (5, 6). Occasionally a patient with grand mal seizures may even simulate such seizures when malingering or during a conversion reaction (7). Probably the most common source of error in this area is in not obtaining an adequate history when confronted with some of these symptoms.

PETIT MAL EPILEPSY

Of least importance because of its relatively low incidence is petit mal epilepsy. However, we have often heard anecdotally of a child "diagnosed" by a teacher, parent, or other lay person as lazy, depressed, having a learning disability, etc., when in reality he/she is suffering from petit mal. In addition to the brief staring spells, petit mal has recently been reported to produce affective manifestations. Wells (8) reported two cases in which depression characterized by withdrawal, veiled hostility, and suicidal thoughts was accompanied by the typical 3-cps spike and wave pattern of petit mal. It

Dr. LaWall is Treatment Director, Mental Health Services, St. Mary's Hospital and Health Center, 1601 West St. Mary's Rd., Tucson, Ariz. 85703.

is also of note that the onset in this case was in late life, even though it is often said that this disorder does not have adult onset.

CASE REPORTS

The following case reports will demonstrate some of the principles to keep in mind in diagnosing epilepsy with predominantly psychiatric symptoms.

Case 1. A 28-year-old woman was brought to the emergency room by her mother, who said that she had been acting strange for about a week. A psychiatric consultation was requested by the intern. The patient was found to be awake but unresponsive to questioning, although she seemed to want to reply. Being unable to do so, she got up from her bed and attempted to leave once but was easily led back to bed. Finally, she was asked whether she was having difficulty thinking, in response to which she simply nodded. The mother could only add that her daughter had seemed distant and quiet, had eaten little, and had paid little attention to her husband and children for about a week; she had been well before this.

After rather extensive but fairly unproductive questioning, the mother was asked if her daughter had convulsions. She said she thought so. Further questioning revealed that the patient had had grand mal seizures for about 2 years but had not been evaluated. Her mother recalled that about a week before admission the patient had been found semiconscious on the bathroom floor at home. An EEG was obtained and a constant pattern of slow high-voltage waves was found. A diagnosis of epileptic twilight state was made and the patient was admitted for further neuropsychiatric evaluation.

This case illustrates the need to consult family members in taking histories, especially when the patient's sensorium is clouded, and the need to consider organic causes when the patient's consciousness is altered. Fortunately, an EEG could be obtained on an emergency basis.

Case 2. A 26-year-old man was sent to the emergency room by his family physician with the brief statement that he was "psychotic and needed hospitalization." The patient said that he had smelled acetylene gas at work. Concerned about a possible explosion, he investigated, and found tanks of gas on the roof of his shop pouring into the air-conditioning system. He called the fire and police departments, but they could find nothing wrong and suggested that the patient see a physician.

A psychiatric consultation was requested in the emergency room and no history of behavioral problems could be elicited from the patient or his wife on routine history taking. He denied drug use and there was no evidence of delusions or hallucinations on mental status examination. The patient was puzzled about what had happened. However, further questioning revealed a history of short lapses of consciousness, one of which had resulted in a serious car accident, and the fact that the patient had once been in a building during a gas explosion.

Psychomotor epilepsy was suspected and, following a neurological examination, an EEG was obtained. This revealed frequent random sharp spikes superimposed on an 8–13 cps background in the left temporal area. He was then admitted to the neurology service, where idiopathic psychomotor epilepsy was diagnosed.

This type of case presents the greatest diagnostic problem. There is already a psychiatric label, and the symptoms are consistent with a psychiatric disorder. However, the isolation of the symptoms in time, the bewilderment of the patient and his spouse, and the negative history are inconsistent with most psychiatric disorders. The fact from the history that led to a strong suspicion of a seizure disorder was the patient's lapses of consciousness. Thus, we should search for alterations in consciousness in the history in many psychiatric cases.

Case 3. An 18-year-old girl was referred for admission to the psychiatric service because of a history of violent outbursts. She stated that at times a feeling of rage, with or without provocation, overcame her and she became very violent, usually destroying property and occasionally attacking people. She said that after these episodes, which lasted only minutes, she felt drained, remorseful, and somewhat confused.

Psychiatric evaluation revealed a rather immature, somewhat depressed young woman with a long-standing history of conflict with her parents. Her mother had been in a psychiatric hospital for several months when the patient was 5 years old. Psychological testing supported the evaluation findings and revealed no significant signs of intellectual deficit.

Neurological work-up revealed some drift of the left arm on sustained extension and an EEG with right temporal spiking. Brain scan and cerebral angiography were negative. The neurological consultants were reluctant to diagnose epilepsy. However, the patient was tried on 300 mg/day of diphenylhydantoin (Dilantin) and remained free of such episodes until she was lost to follow-up some 8 months later.

This case illustrates the difficulty in defining epilepsy. While the patient did not have a typical psychomotor seizure, she did have paroxysmal outbursts of ego dystonic behavior with an EEG that suggested a seizure disorder. This kind of clinical picture has been called "episodic dyscontrol" and has been studied by Maletsky (9) and others. In his series, as in the present case, diphenylhydantoin was helpful in controlling such outbursts. Thus, even in the face of some ambiguity, clinical suspicion should lead to diagnostic consideration of a seizure disorder.

CONCLUSIONS

There is a wide range of disturbances in behavior, thought, and affect that, although they are usually associated with psychiatric disorders, may be seen as symptoms of epilepsy. Such symptoms may be the presenting complaints in undiagnosed cases of epilepsy. In order to ensure accurate diagnosis, physicians should maintain a high degree of suspicion of this possibility. Family members should be interviewed, and the periodicity of symptoms and any alterations of

consciousness should be noted. In addition, electroencephalography should be used on an emergency basis in such cases.

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Decreased Blood Platelet MAO Activity in Unmedicated Chronic Schizophrenic Patients

BY EDWARD F. DOMINO, M.D., AND SUMATHY SAMPATH KHANNA, PH.D.

The authors found lower platelet monoamine oxidase (MAO) activity in 13 unmedicated chronic schizophrenic male patients in comparison to 13 mentally normal control male subjects—a finding that agrees with previous studies showing MAO activity to be low in the platelets of chronic schizophrenic patients. The authors urge that studies exploring endocrine factors and the role of hospitalization be undertaken to attempt to explain this finding.

THE RECENT FINDING that brain monoamine oxidase (MAO) activity of deceased chronic schizophrenic patients was normal (1) agrees with findings of other investigators (2–4). Reports of abnormally low platelet MAO activity in chronic schizophrenic patients (4–6) are therefore surprising. Furthermore, normal MAO platelet activity in acute schizophrenic patients has also been reported (7–9).

Inasmuch as drug medication cannot easily be controlled for the vast majority of psychiatric patients studied, it seemed important to confirm that platelet MAO activity was decreased in a group of unmedicated chronic schizophrenic patients. We have had a unique opportunity to study a group of drug-free chronic male schizophrenic patients given a nutritious, vitamin-supplemented diet and housed in a pleasant research ward where physical exercise and occupational therapy were readily provided. This group of chronic schizophrenic patients was highly selected on the basis of being formerly drug-resistant. Because these patients did not respond favorably to neuroleptic medication and because of the possibility of undesirable long-range side effects, all psychoactive medication was withdrawn for a minimum of six months.

Dr. Domino is Head, Laboratory of Pharmacology, the Lafayette Clinic, 951 East Lafayette, Detroit, Mich. 48207, and Professor of Pharmacology, University of Michigan, Ann Arbor, Mich. Dr. Khanna is a Research Associate, Department of Pharmacology, University of California, San Francisco, Calif.

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METHOD

Subjects

A total of 13 chronic schizophrenic men and 13 control (mentally normal) men were studied. The criteria for inclusion of each patient in the study were similar to those previously described (1). The control subjects consisted of drug-free men from 20 to 41 years of age, chosen from Lafayette Clinic personnel and medical students with normal medical histories and psychiatric status. All of the control subjects had been drug-free for one to two months prior to their participation, except that acetylsalicylic acid and ethyl alcohol were allowed on rare occasions. However, these drugs were never allowed during the 24-hour period immediately preceding blood withdrawal.

All subjects were instructed to go about their usual daily activities. Thus the control subjects as a group probably had less exercise than the patients, for whom recreational therapy was vigorous. Grossly overweight subjects were excluded except for matching purposes with an occasional overweight patient.

The chronic schizophrenic patients ranged in age from 28 to 44 years. All had been mentally ill since adolescence or young adulthood. They had all received neuroleptic medication at one time, some for many years, but had shown no significant therapeutic benefit. Hence, medication could ethically be stopped at least six months prior to the study.

The patients showed classical primary symptoms of schizophrenia of the process type. They were on a diet of 2300 calories a day, with multivitamin and mineral supplements (MYADEC) given once a day. The control subjects were asked to eat a balanced diet and to take a vitamin supplement.

Procedure

All patients and subjects were asked to fast overnight, and a blood sample was drawn between 7:30 and 8:00 a.m. the next morning. Fifty milliliters of venous blood was drawn with plastic syringes into an ACD solution (acid-citrate-dextrose formula A, Becton-Dickinson Co.). Samples were immediately placed in ice. Platelets were separated in a refrigerated centrifuge using several different spins. The first was spun at about 175 g, the second at about 300 g, and the final at about 600 g. The platelet-rich plasma was separated from red cells with a silicone-treated pipette.

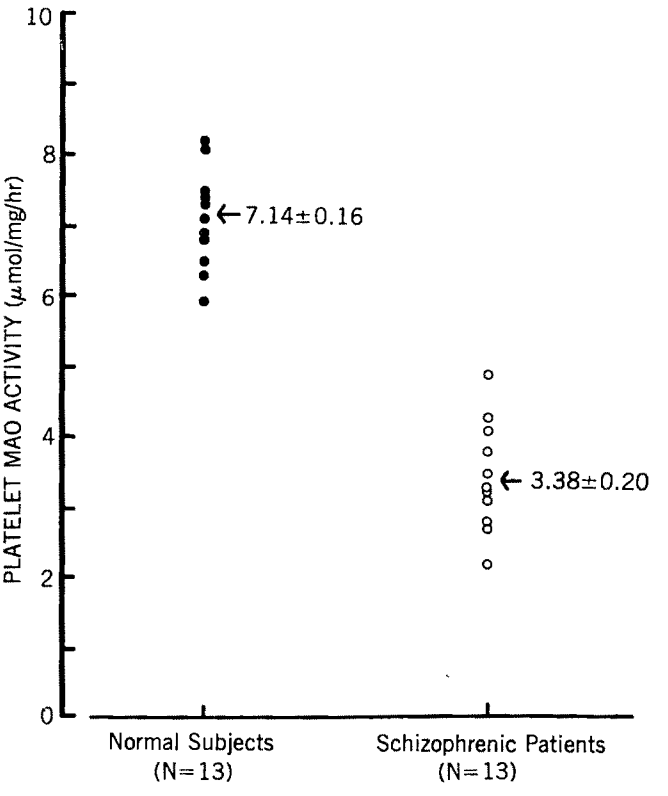
Subsequently, the platelet-rich plasma was transferred to a small plastic centrifuge tube and centrifuged at 3000 g for 5 minutes. A pellet of platelets remained in the bottom, and the platelet-poor plasma was removed. The platelets were then rinsed gently with 0.9% NaCl to remove plasma, by placing 1 ml of 0.9% NaCl into the tube from a pipette, inverting the centrifuge tube, and allowing the fluid to run out.

Platelet MAO activity was assayed using ^{14}C-tryptamine to form ^{14}C-indole acid aldehyde (which can be further oxidized by aldehyde dehydrogenase to form indoleacetic acid [10]). In addition, platelet protein was determined (11). Platelet MAO activity was expressed in micromoles of substrate per milligram of protein per hour. The reaction mixture was incubated for 15 minutes at 37° C and was terminated by the addition of 0.1 ml of concentrated perchloric acid.

RESULTS

Platelet MAO activity for control subjects and drug-free chronic schizophrenic patients showed no overlap in level of activity between the two groups (see figure 1). The mean platelet MAO activity was 3.38±0.20 μmol/mg of protein per hour for the drug-free chronic schizophrenic patients and 7.14±0.16 μmol/mg of protein per hour for the mentally normal control subjects.

FIGURE 1
Scattergram of Platelet MAO Activity for Control Subjects and Drug-Free Chronic Schizophrenic Patients



This difference was highly statistically significant using a group-comparison t test ($t=14.7$, $p<.001$). The mean age of the chronic schizophrenic patients was 36.5±1.8 years and of the control subjects 32.2±2.4 years. This difference was not statistically significant.

DISCUSSION

Platelet MAO activity in psychiatric populations has been studied by many investigators and has been found to vary markedly depending upon psychiatric state as well as genetic and hormonal factors. Nies and associates (4), Murphy and Wyatt (5), Wyatt and associates (6), and Meltzer and Stahl (7), using tryptamine as well as other substrates, reported that platelet MAO activity was significantly lower in chronic schizophrenic patients than in control subjects. Meltzer and Stahl (7) pointed out that platelet MAO activity was lower in acute schizophrenic patients than in the control subjects only when *m*-iodobenzylamine or tyramine were used as substrates. When octopamine and tryptamine were used, acute schizophrenic patients had normal platelet MAO activity.

Friedman and associates (8) reported that when tryptamine was used as the substrate no differences were found among groups of normal control subjects, patients with acute schizophrenia, and patients with

other psychiatric diagnoses. In their control group, women had significantly higher enzyme activity than men. Female schizophrenic patients showed a trend toward lower activity than did female control subjects, but the female patients did not differ from the male schizophrenic patients. Friedman and associates suggested that platelet MAO activity may be related to hormonal, dietary, or genetic factors but not to psychiatric diagnosis. These findings are consistent with those of Wyatt and associates (6), who have suggested that MAO activity in platelets is a possible genetic marker for vulnerability to schizophrenia.

Endocrine factors are extremely important and should be given prominent attention. It has been shown in studies with rats that estrogen and corticosteroids decrease MAO activity while progesterone increases MAO activity; furthermore, there are marked variations in various tissue (including brain) MAO activities as a function of the estrous cycle (12). Human platelet MAO activity varies as a function of the menstrual cycle and is lowest just before menses (13).

It should be pointed out that platelet MAO activity and brain MAO activity are probably unrelated. For example, human brain MAO consists of four isoenzymes (14), while human platelet MAO is electrophoretically homogenous (15). Furthermore, on the basis of substrate specificity brain MAO has been suggested to be both A type (dopamine, norepinephrine, or 5-hydroxytryptamine as substrate) and B type (benzylamine as substrate). Platelet MAO is primarily B type (16). Tryptamine, the amine substrate used in this study, is oxidized by type A and B MAO.

Both Wyatt and associates (6) and Nies and associates (4, 17) have emphasized the importance of a genetic factor by studying the levels of platelet MAO activity in families with lowered enzyme activity.

Murphy and Weiss (18) have reported reduced platelet MAO activity in bipolar depressed patients. Nies and associates (4, 19) have emphasized the relationship between MAO activity and aging as well as mental depression. Especially important is the finding that MAO activity in the brain dramatically increases with old age. It is of interest that in this study the chronic schizophrenic patients as a group were slightly older than the control subjects, but this trend was not statistically significant. The patients still showed a lowered platelet MAO activity.

It would appear that the finding of a 50% decrease in platelet MAO activity in drug-free chronic schizophrenic male patients is reliable, for it has now been replicated in at least five different laboratories. Three laboratory studies have shown that acute schizophrenic patients do not have such a decrease. This suggests that factors secondary to the illness may have an important role. In the present study, the schizophrenic patients ate a nutritious diet and had plenty of exercise, so that such environmental factors would seem to be ruled out. However, a chronic debilitating disease, developing in young adulthood and lasting for many years, could have obvious and important effects secondary to hospitalization, as is made evident by the many reports of biologic abnormalities in psychiatric patients (20–25). The role of endocrine changes also is important. A chronic schizophrenic male population similar to ours has been shown to have lowered plasma dehydroepiandrosterone levels (26).

Another point that should be kept in mind is that unmedicated chronic schizophrenic patients tend to have elevated platelet 5-hydroxytryptamine (27). Although platelet MAO is of the B type, there may be some interaction between endogenous platelet 5-hydroxytryptamine and exogenous ^{14}C-tryptamine added as the substrate. Further studies exploring the possible role of endocrine factors and duration of hospitalization are clearly needed to explain our finding of lowered platelet MAO activity in chronic schizophrenic male patients.

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Consultation-Liaison Psychiatry: Current Responsibilities

BY WALTER A. BROWN, M.D., AND ERIC M. JACOBSON, M.D.

The authors describe their experiences as consultation-liaison psychiatrists in two general teaching hospitals. Current medical house staff members appear to be more aware of the importance of psychosocial issues in patient care than were interns and residents in the past. Consultation-liaison teaching may now focus on the application of this psychologic awareness to patient management, the correct use of psychotropic drugs, and the differentiation of delirium from functional psychoses.

THE CONSULTATION-LIAISON psychiatrist has traditionally focused his teaching efforts on sensitizing medical students and house staff to the relevance of psychosocial issues in medical illness and management, interview technique, personality assessment, recognition and management of psychiatric syndromes, and concepts of psychogenicity and psychosomatic medicine (1, 2). However, the reluctance or inability of medical house staff to think in psychologic terms and accept the importance of psychosocial variables in medical practice has been noted as a primary obstacle to these teaching efforts (2, 3). The orientation of nonpsychiatric house staff to "disease" rather than "person" has been sufficiently frustrating to liaison teaching efforts to lead McKegney (3) to suggest that such teaching should be directed instead toward medical students.

Much of the literature regarding consultation-liaison psychiatry records the experiences of the pioneers in this area, who did their initial work before 1960. Our experience over the past academic year with medical house staff and medical students rotating through teaching medical services suggests that the approach of house staff of the 1970s to psychologic issues may be different and that the teaching role of the present-day consultation-liaison psychiatrist may require a different emphasis.

One of us, W.A.B. (a psychiatrist), makes liaison rounds once weekly with medical house staff and students rotating through medicine at two general hospitals affiliated with a medical school; he also provides formal psychiatric consultation to teaching medical and surgical services. The other, E.M.J. (a second-year resident in psychiatry), makes morning work-rounds with medical house staff two times weekly; in addition, he provides psychiatric consultation to the

Dr. Brown is Director, Psychiatric Consultation-Liaison Service, Veterans Administration Hospital, Davis Park, Providence, R.I. 02908, and Assistant Professor of Psychiatry, Brown University, Providence, R.I. Dr. Jacobson is senior resident in the Department of Psychiatry, Brown University, Providence, R.I.

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medical and surgical services of a general hospital. Through these activities we have had contact with about 50 medical residents and interns from a variety of medical schools and geographic areas. Neither general hospital had psychiatric consultation-liaison teaching prior to this academic year.

TEACHING AREAS

Psychopharmacology

Although clinical psychopharmacology has been mentioned by previous authors as one of the teaching areas of the consultation-liaison psychiatrist, it is not one of the teaching functions that has been emphasized (1). A considerable portion of our time was taken up in teaching proper use of psychotropic drugs. Our knowledge of psychopharmacology was probably called upon as frequently (if not more frequently) as our fund of information about psychodynamics and psychosomatic medicine. Medical house staff seemed to be aware of the general classes of psychotropic drugs but had only a vague sense of how and when they should be used. Much of our teaching in psychopharmacology was directed toward defining those conditions appropriate for treatment with specific psychopharmacologic agents; for example, indicating the type of depression most appropriate for the use of tricyclic antidepressants and identifying the target symptoms of these medications. Staff members typically tended to prescribe neuroleptics and tricyclic antidepressants in dosages far below the therapeutic level and also tended to prescribe benzodiazepines indiscriminately. In addition, the side effects of psychotropic drugs and the organic conditions that contraindicate their use were not generally appreciated. After appropriate explanations, medical house staff members appeared quite willing to prescribe full therapeutic doses of these medications.

This emphasis on psychopharmacology is, of course, partly a result of the current availability and effectiveness of a variety of psychopharmacologic agents. It may also reflect a trend among young medical professionals to view the psychopharmacologist, the manipulator of brain neurochemistry, as the current "wizard" in psychiatry.

Recognition of Delirium

Although house staff members were familiar with the features of the chronic brain syndromes, the features differentiating delirium from functional psychoses were not generally appreciated, and the subtle manifestations of delirium were not usually recognized. There was a tendency to attribute psychotic behavior and confusion to schizophrenia, without an adequate medical or psychiatric evaluation for the presence of an organic etiology. House staff tended to rely on whether patients were oriented or not as the indicator of cognitive functioning.

The readiness of house staff to make diagnoses of functional psychosis and their relative insensitivity to the manifestations of delirium may be related to the fact that the organic brain syndromes, particularly delirium, fall somewhere between the cracks in medical school teaching; they are not emphasized by the departments of psychiatry, neurology, or medicine. Clinical clerkships in psychiatry tend to focus on the functional psychoses rather than on the organic brain syndromes. We believe, along with previous authors (4), that delirium is underdiagnosed on medical services.

ATTITUDES TOWARD THE LIAISON PSYCHIATRIST AND PSYCHOLOGIC CONCEPTS

Contrary to our expectations, the house staff of the two general hospitals in which we taught were not resistant to our presence, but were instead pleased to have psychiatric consultation-liaison services available. In one hospital the medical house staff itself had initiated the development of liaison psychiatry teaching. We found that residents and interns were aware of the importance of psychologic issues in patient management and almost invariably considered situational stress, personality style, and psychologic symptoms to be worthy of serious consideration in dealing with their medical patients. Thus it became clear that indoctrination regarding the relevance of psychosocial issues in the practice of medicine would not be one of our major functions.

This is not to say that the residents and interns we encountered had a well-developed sense of the role of psychologic variables in medical illness and medical management, or that their psychologic awareness was necessarily reflected in their style of patient care, but rather that they accepted the importance of psychologic issues in their work. This psychologic mindedness of the house staff—their orientation to both person and disease—was in contrast to the attitudes recorded by previous workers. This apparent change in attitude may be related to the social awareness and humanitarian concerns characteristic of the college students of the 1960s and to the increasingly firm place of behavioral science in the medical school curriculum.

DISCUSSION

In developing psychiatric consultation-liaison services in two medical school-affiliated general hospitals over the past academic year, we found ourselves dealing with somewhat different issues than we had expected. This is not surprising in view of the fact that the consumers of our teaching arose out of a different social and medical school context than did their forebears. Although some of our time was spent teaching and discussing concepts of psychosomatic medicine, psychoanalytic developmental psychology, and character style, a considerable portion was spent teaching psychopharmacology and the differential diagnosis of

psychotic behavior. Thus, we feel it is crucial that the psychiatrist who undertakes such teaching should be thoroughly grounded in clinical psychopharmacology and in the recognition and management of organic brain syndromes, as well as in psychosomatic and psychodynamic theory.

The lack of resistance to psychologic concepts and the sensitivity of the house staff we encountered to psychosocial issues may represent a change in attitude from that confronting previous workers. At present, however, this possible change in attitude is more impressive than any apparent change in style of patient care. The important task of current consultation-liaison psychiatrists may be to guide new nonpsychiatric physicians in applying their psychologic awareness to their management of patients.

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The Role of the Chief Resident: Expectations and Reality

BY JACK F. WILDER, M.D., ROBERT PLUTCHIK, PH.D., AND HOPE R. CONTE, PH.D.

The job expectations of 35 incoming chief residents were tested by questionnaire when they attended a weekend workshop on leadership and were reexamined after they had had six months' experience on their jobs. Such personal qualities as a sense of humor and persistence and such administrative skills as the ability to exercise authority and set objectives significantly increased in importance six months after these residents began work. The authors recommend educational programs in administration for chief residents.

THE ROLE OF THE CHIEF RESIDENT in psychiatric residency programs has received little systematic examination. However, the Department of Psychiatry at Albert Einstein College of Medicine had a unique opportunity in June 1974 to investigate the attitudes and functions of chief residents in psychiatry from many medical schools throughout the United States. The department offered a weekend workshop designed to increase leadership skills and sensitivities of incoming chief residents. Notices were sent to all medical schools throughout the country, and a total of 65 chief residents decided to attend. During their stay at the workshop, they were exposed to formal and informal course material. In addition, they were asked to complete a questionnaire designed to determine their attitudes concerning the roles they would soon assume as chief residents. This report compares the results of this initial survey with the results of a follow-up survey after the chief residents had had approximately six months' experience on their jobs. Thirty-five of the chief residents completed both surveys.

Dr. Wilder is Associate Dean for Planning and Operations, Dr. Plutchik is Associate Professor, Department of Psychiatry, and Dr. Conte is Research Associate, Department of Psychiatry, Albert Einstein College of Medicine, Yeshiva University, 1300 Morris Park Ave., Bronx, N.Y. 10461.

METHOD

A 46-item questionnaire was constructed.[1] The information used for the development of the items came primarily from two sources. The first was the 19-item questionnaire used by Grant and associates (1) for their resident-staff survey. This questionnaire was based principally on information provided by the proceedings of a panel on the function of the chief resident at the 124th annual meeting of APA, Washington, D.C., May 3–7, 1971. The second source of information for questionnaire items was consultations with

[1]Copies of the complete questionnaire and results are available by request from Dr. Wilder.

several psychiatrists who have supervised psychiatric chief residents.

The questionnaire covered a wide variety of issues relevant to the role of the chief resident: academic skills, personal qualities, administrative skills and practices, relationship between faculty and staff, organizational arrangements, and ways of improving leadership performance. The chief residents were asked to indicate on a scale from 1 to 5 the relative importance of each role or characteristic.

The first questionnaire was administered at the beginning of the workshop, and the second was completed by mail. The reliability of the questionnaire was determined by randomly dividing the questionnaires completed at the workshop into two subgroups. The mean score for each item within each subgroup was computed, and the mean scores for all corresponding pairs of items were then correlated (2). After correction for attenuation, the correlation was found to be +.94, indicating high reliability for the questionnaire.

Thirty-five chief residents, representing 31 colleges of medicine and 20 states across the nation, completed the questionnaire on the two occasions. The mean importance rating for each item at both times was calculated. Tests of significance for paired comparisons (t test) were then performed to determine whether the mean importance rating for each item changed over time.

RESULTS

The most obvious finding of the study was that there was very little change in the mean importance ratings of items after the chief residents had had six months' experience. For example, the item concerned with facilitating communication between house staff and permanent staff was given a mean importance rating of 4.71 at the start of the workshop. Six months later, this item was given a mean importance rating of 4.60. This change was not significant. However, the high rating this item was given on both occasions indicated that the chief residents considered facilitating communication to be an extremely important function. Similarly, at the time of the workshop the chief residents gave a mean importance rating of 1.77 to the question, "How important is it that the chief resident be a fourth-year resident rather than a third-year resident?" At the time of follow-up, the mean rating for this item was 1.91. This item thus appeared to be relatively unimportant at the time of both surveys. The product-moment correlation between the two sets of mean scores was .93. This supports the interpretation of relatively little change in the rank order of importance of most of the items over a six-month period. (The rank order was determined on the basis of the mean rating for each item.)

However, there was a significant change in the mean ratings of 11 of the 46 items over the six-month period. These items, 8 of which increased in importance and 3 of which decreased in importance, are presented in table 1. Also presented are the rank orderings of these items at the two testing periods.

The increases and decreases in importance of the issues dealt with in these 11 questionnaire items may be considered in terms of the discrepancy between the expectations of incoming chief residents and the reality they faced on the job. For example, 1 of the 3 items that decreased in importance concerned the way in which a chief resident is chosen: "How important is it for the appointment of the chief resident to be determined by a vote of his peer residents?" The incoming chief residents, most of whom had been appointed by faculty, had expressed concern at the workshop that their peers might react to their appointments with envy and hostility. A few felt that chief residents should therefore be chosen by their peers. However, after six months in a leadership role, they were apparently more comfortable in their identification with faculty and in their exercise of authority. Therefore, they were less concerned with how their peers might view their selection.

In a similar vein, the chief residents attached less importance to being able to get along with everyone after six months on the job. They apparently came to the conclusion that one could not get a job done without generating a certain amount of conflict and frustration.

The third item that decreased in its mean importance rating concerned research skills. It should be noted that the importance of these skills was relatively low to begin with; it appears that chief residents, like most busy administrator-clinicians, generally have little time for research activities.

Items that increased in importance also reflect the chief residents' greater awareness of the administrative complexities of their positions. It would seem that they had come to their jobs with great expectations of what they could accomplish, overlooking the fact that theirs was a transitional, time-limited role with little authority and with ill-defined responsibilities. As pointed out by Grant and associates (1), the chief residency requires "living with uncertain consequences, divided loyalties, and at times insolvable ambiguity" (p. 503). The second survey revealed a significant increase in the importance attached to exercising authority, telling people what to do even if they do not like it, and setting objectives for the year.

Three personality items increased in importance: having a sense of humor, having an optimistic outlook, and being persistent. After struggling with the ambiguities and frustrations of their administrative roles, the chief residents appeared to realize that their personal sense of well-being and their success in effecting change required a lack of defensiveness, a belief that situations can be improved, and stubbornness in the face of obstacles.

The last 2 items that increased in importance concerned training in leadership skills: an elective seminar on administrative skills and management in a residency training program and the helpfulness of the weekend workshop in becoming a good chief resident.

TABLE 1
Rank Ordering and Chief Residents' Mean Ratings of Importance of 11 Aspects of Their Jobs Before and After Six Months' Experience

Question	Rank Ordering*		Mean Rating of Importance**		
	Before Starting	After Six Months	Before Starting	After Six Months	Significance***
How important is it to have a sense of humor?	12.0	1.0	4.29	4.77	$p<.001$
How important is the quality of persistence in doing the job?	9.0	2.0	4.34	4.71	$p<.01$
How important is it to tell people what to do even if they don't like it?	23.5	10.0	3.74	4.43	$p<.01$
How important is it to have an optimistic outlook?	21.5	14.0	3.77	4.31	$p<.01$
How important is it to set objectives for what you hope to accomplish during your year on the job?	20.0	16.0	3.89	4.29	$p<.05$
How important is it to exercise authority in doing your job?	29.0	18.0	3.49	4.26	$p<.001$
How important is it to have at least an elective seminar on administrative skills and management in a residency training program?	33.0	24.0	3.39	3.97	$p<.001$
How important do you expect this workshop to be in helping you to become a good chief resident?	32.0	27.0	3.35	3.71	$p<.05$
How important is it to be able to get along well with everyone (house staff and permanent staff)?	25.0	33.0	3.71	3.26	$p<.05$
How important are research skills?	38.0	43.0	2.60	2.23	$p<.01$
How important is it for the appointment of the chief resident to be determined by a vote of his peer residents?	37.0	44.0	2.71	2.11	$p<.01$

* 46 items were rank-ordered.
** 5-point scale: 5 =very important, 3 =moderately important, 1 =not important.
*** By t test.

Another way to consider the data is in terms of ranking of items at the time of follow-up. Two personal qualities, sense of humor and persistence, were ranked 1 and 2, respectively. Management skills and practices were the remaining most highly ranked items, e.g., facilitating communication, consistency, and accessibility to staff. Such fiscal and organizational arrangements as a salary premium, a faculty appointment, and the selection process were ranked low at both testing periods. Although these were issues that evoked considerable discussion at the workshop, the chief residents apparently realized even before beginning their jobs that competent people, not organizational procedures, make for results.

In addition to having varying degrees of administrative responsibility, almost all of the chief residents had academic assignments. Skills related to these assignments, usually considered to be the sine qua non for selection of a chief resident, all dropped in importance at the time of follow-up. Teaching skills went from a rank of 2 to that of 12, clinical skills from 9 to 16, and research skills from 38 to 43. The decrease in importance of these academic skills and the increase in importance of management and interpersonal skills probably reflect the realistic demands of the job. In all positions of leadership in an academic setting there is a tendency to underestimate the administrative skills required and to overestimate the opportunities to engage in academic activities. The development of a smoothly running setting is a precondition for the application of academic skills.

One series of items concerned ways of acquiring leadership skills. These items were generally ranked in the middle range. Among these items, a good role model, on-the-job experience, and an elective seminar on administration were judged to be most important, whereas a required seminar, clinical training, and the reading of management literature were viewed as less helpful.

DISCUSSION

The results of the survey have two implications. First, they identify a number of personal character-

istics and administrative skills that chief residents consider to be important in the successful performance of their jobs. This information may be useful to those responsible for selecting chief residents. Second, the survey highlights the difficulties chief residents face in assuming positions of authority and responsibility, usually without previous training or ongoing support. Parenthetically, these difficulties were a major focus in group discussions at the chief residency conference. As has been pointed out by Barton (3), the lack of adequate training is a general problem in the field of administrative psychiatry.

It is reasonable, therefore, to conclude that departments of psychiatry should not allow the chief resident to sink or swim on his own and, possibly, because of failure, become turned off to future leadership positions. Rather, psychiatry departments should view the chief residency as an educational assignment and opportunity that requires a systematic and thoughtful educational program (4). Future research will be directed at examining the administrative careers of chief residents who had special support and training during their chief residency in comparison with those who did not.

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The Effect of Haloperidol on Stuttering

BY PETER B. ROSENBERGER, M.D., JULIE A. WHEELDEN, M.ED., AND MADELINE KALOTKIN

Haloperidol was compared with placebo in the treatment of 8 young adult stutterers in a double-blind crossover study involving 6 sequential observations over periods of 6 or 12 weeks. The drug was found to have a significant effect on the percentage of time subjects were dysfluent but not on the number of dysfluencies per minute. The authors discuss possible reasons for this finding.

RECENT STUDIES of the effect of haloperidol on various tic syndromes (1) and especially on the convulsive tics of Gilles de la Tourette's syndrome (2) have led to the suggestion that the drug might be useful in the syndrome of common stuttering. Several studies on this subject have recently appeared in the literature. Tapia (3), studying 11 patients, showed that only 2 of the 6 patients who reported improvement actually depressed the number of nonfluencies in their verbal output. This report commented that "a surreptitious placebo produced relapse." Gattuso and Leocata (4) claimed 80% complete relief in 50 patients aged 5 through 12 but used no control subjects. The study of Wells and Malcolm (5) was placebo controlled and double blind and examined haloperidol in combination with orphenadrine and speech therapy. Of 12 patients on haloperidol, 10 showed improvement as judged by a reduction in the number of nonfluencies per 100 words.

We decided to test the effect of haloperidol on stuttering using a double-blind crossover design and attempting to show repetitive fluctuations in speech fluency as a result of change from placebo to haloperidol over several weeks in a small number of patients.

METHOD

Our subjects were 8 young adult stutterers who were attending a weekly group stuttering therapy session in the Language Clinic of the Massachusetts General Hospital. There were 6 men and 2 women, all between the ages of 20 and 32. The 6 men and 1 of the women

The authors are with the Massachusetts General Hospital, Boston, Mass. 02114, where Dr. Rosenberger is Director and Ms. Kalotkin is Research Assistant, Learning Disorders Unit, and Ms. Wheelden is Speech Pathology Supervisor, Language Clinic; Dr. Rosenberger is also Assistant Professor of Neurology, Harvard Medical School.

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had been stuttering since early childhood; the other woman, according to her own report, had been stuttering since age 12. All had had previous speech therapy, with experience ranging from 1 to 10 years. None had had prior drug therapy. All of the subjects were neurologically intact, of normal intelligence, and gainfully employed. Informed consent was obtained from all subjects after explanation of risks and benefits.

Medication, either haloperidol or an identical appearing placebo, was given uniformly in a dose of 1 mg three times a day. The medication was dispensed to the patients in envelopes at six regular intervals: every two weeks in the case of subjects A and E and weekly in the case of the other 6. Subjects were told that the tablets dispensed to them would contain haloperidol for three of the six study periods and "another medication" for the other three. Patients were assigned by a neutral third party to one of a list of six schedules submitted by the experimenters. Neither the experimenters nor the subjects were aware of which schedule was in effect, and in addition the subjects were not told six schedules might be used.

If side effects to the medication were reported before the particular study period ended, the patient was advised to reduce the dosage of the medication by one tablet per day and to attempt to tolerate the side effects. Three previous subjects, all teenagers, had been unable to complete the study because of inability to tolerate the side effects.

Testing sessions were conducted at the end of each study period (every two weeks in the case of subjects A and E and every week in the case of the other 6). Sessions were videotaped in their entirety by a camera located behind a one-way observation window, of which the subjects were aware.

The sessions tested three speaking conditions: spontaneous conversation, reading, and picture description. In the conversational section, the subject was first asked to identify himself and give some biographical information and then to describe his speech problem briefly. He was next questioned about his activities during the past week and asked his opinion concerning the fluency of his speech. Finally, he was asked to describe any side effects that had occurred during the previous week. In the reading section, the subject was given a written paragraph of about 100 words and asked to read it aloud. Selection of the material was made at random from three standard paragraphs commonly employed by speech therapists to test enunciation of all phonemes in the language. In the third section, the subject was given one of three complex pictures and asked to describe the action in it.

Data concerning the subject's speech fluency were gathered at a later time by an impartial observer. The observer sat viewing the videotape and holding two hand switches, each of which was connected both to a sequential counter and a running time meter. The observer pressed one switch (and held it down) whenever the subject was talking, and the other switch whenever the subject was dysfluent. Thus a measure of both percentage of time dysfluent and of dysfluencies per minute of spoken speech was acquired. The observer made repeated observations of a single session with each subject until two sequential observations differed by not more than 5%; then he continued with single observations of subsequent sessions.

RESULTS

Figure 1 shows the percentage of verbal output that was dysfluent for each subject during the baseline evaluation and in each of the six testing sessions that followed haloperidol or placebo therapy under the conditions of spontaneous conversation, reading, and picture description.

From these raw data we concluded that subjects A, B, C, and F showed a definite response to the drug; subjects D and G showed a probable response, subject E definitely showed no response, and subject H had too low an initial stuttering rate to allow a decision. In addition, the figure indicates a mild general placebo response as a function of time throughout the study.

Comparisons of the performance of all subjects under the three speaking conditions show a general similarity, with some interesting exceptions. Subject A, whose dysfluency in spontaneous conversation frequently rendered him effectively incoherent, was almost entirely fluent when reading aloud, even in the baseline session. Subjects C and E showed a similar pattern, although it was less dramatic. In contrast, subjects D and F were significantly more dysfluent when reading aloud. This finding of varying patterns of nonfluency is in line with the clinical experience of most speech pathologists. Our hypothesis, currently in the process of being tested, is that this difference is related to the stutterer's reading comprehension skills.

Tests for statistical significance of the drug effect were run separately for the three speaking conditions. Figure 2 shows averaged data for spontaneous conversation. Subjects are arranged on the abscissa in order of severity of stuttering according to the baseline measurement. When relative percentages of dysfluency are compared by the Friedman analysis of variance, the significance of the drug effect seems to be related to the severity of initial stuttering. The relationship between severity of initial stuttering and drug treatment results is significant at the .004 level for those subjects who were more than 30% dysfluent at baseline. When this analysis is extended to those who were more than 15% dysfluent at baseline, the significance decreases to $p=.072$; for the entire sample the level of significance is .079.

As may be gathered from figure 1, statistical comparisons for reading and picture description were similar to those for spontaneous conversation, with the exception that for reading, significance was achieved for the five subjects whose baseline dysfluency was greater than 15% ($p=.024$).

Data for stuttering *rate* (number of dysfluencies per

FIGURE 1
Percentage of Time Subjects Were Dysfluent Under Three Speaking Conditions

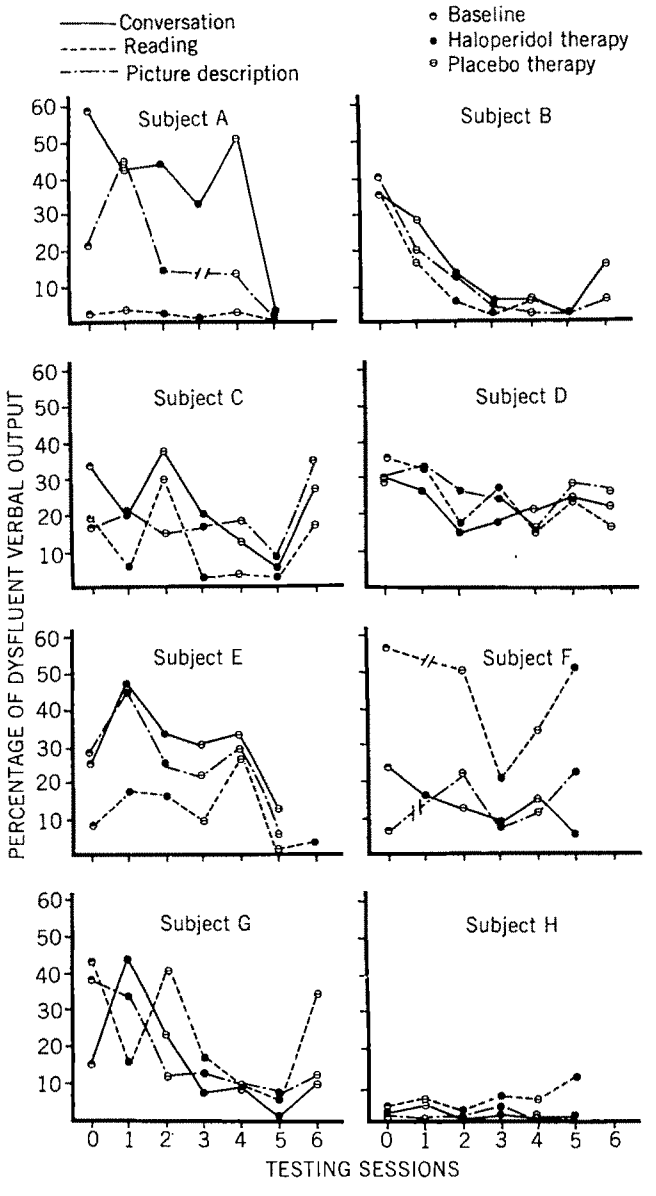


FIGURE 2
Average Percentage of Time Dysfluent During Spontaneous Conversation

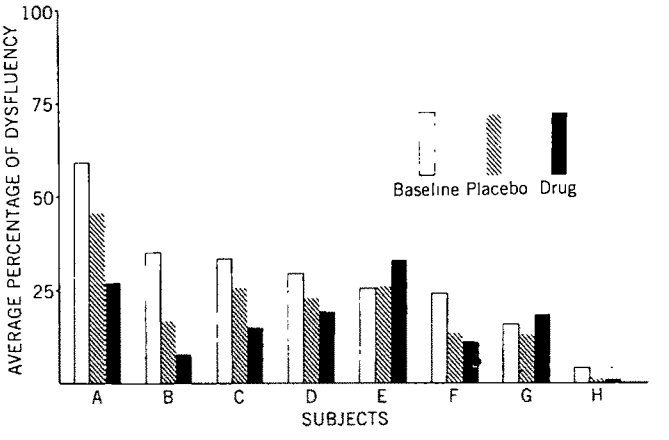
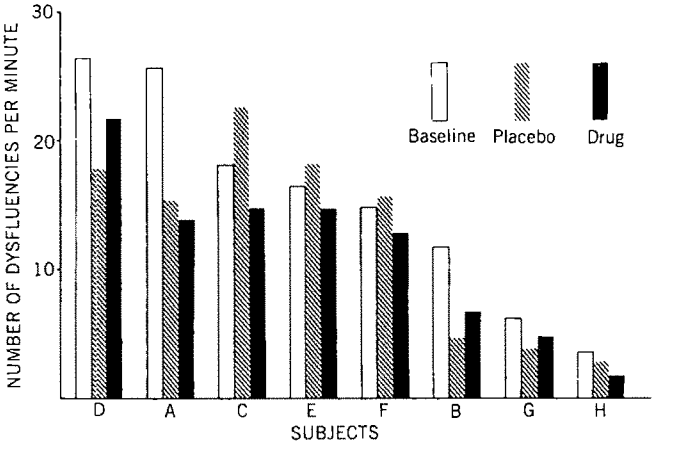


FIGURE 3
Average Number of Dysfluencies per Minute During Spontaneous Conversation



minute) were gathered by counting the number of times a minute during verbal output that the switch registering dysfluency was depressed. Figure 3 shows averaged data for this quantity during spontaneous conversation. Again, subjects are arranged on the abscissa in order of initial severity with respect to this measure. As might be expected, these findings differ somewhat from those for percentage of dysfluency. For example, subjects B and D, whose percentage of time dysfluent improved under the drug, actually showed more dysfluencies per minute; the experience of subject E was just the reverse. In the case of the tonic stutterer, of course, an inverse relationship between these two measurements is to be expected, i.e., a decrease in the number of tonic spasms will result in more syllables per minute, which if they are repetitious will be scored as discrete dysfluencies. This finding confirms the experience of Wells and Malcolm (5), whose findings showed a greater reduction in the amount of time needed to speak 100 words than in the percentage of repetitions as a result of the drug.

Although several subjects, notably C, E, and F, showed significant decreases in their stuttering rate on the drug as compared with placebo, results for the group as a whole did not reach statistical significance by the Friedman analysis.

DISCUSSION

Haloperidol, a butyrophenone, was developed in the course of efforts to increase the analgesic potency of meperidine compounds. According to Janssen (6), who is credited with its synthesis, the drug derives its neuroleptic properties from its substituted propyla-

mine structure, which it shares in common with phenothiazines. Haloperidol accelerates the accumulation and subsequent disappearance of dopamine formed from ^{14}C-tyrosine in the mouse brain (7). It is more potent in this regard than chlorpromazine, which has been shown (8) also to increase the concentration of homovanillic acid in the caudate nucleus of the cat. These findings have suggested a possible mechanism of action of these drugs through central dopamine metabolism.

As we have mentioned, it was the widespread success of haloperidol as a treatment for Gilles de la Tourette's syndrome which suggested that it might be effective with stuttering. The literature on haloperidol with Gilles de la Tourette's syndrome has been exhaustively reviewed by Snyder and associates (2), who have suggested that the apparent effect of haloperidol on central dopamine metabolism might account for the resemblance of the symptoms of Gilles de la Tourette's disease to the side effects of L-dopa therapy and explain the therapeutic efficacy of the drug.

It is our clinical impression from observation of subjects A and C that it is the hesitation and phonemic stoppage of the severe stutterer which are most dramatically affected by haloperidol. We would expect this to be reflected in the measurement of percentage of time dysfluent but not necessarily in dysfluencies per minute. Each of these two patients, under the conditions of severe stuttering, showed a characteristic facial contortion that was almost totally absent from the videotape records taken after haloperidol therapy. Further study may demonstrate that the patients with such severe phonemic stoppage will benefit most from this drug.

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Transsexualism in Two Male Triplets

BY EMBRY A. MCKEE, M.D., HOWARD B. ROBACK, PH.D., AND MARC H. HOLLENDER, M.D.

The authors present clinical and case material on two male triplets with aberrant gender identity. Their findings coincide with those of the few family studies reported in which there were two or more transsexual members; they also reveal patterns generally in keeping with the psychological determinants of transsexualism suggested by Stoller.

The authors are with Vanderbilt University School of Medicine, Nashville, Tenn. 37232, where Dr. McKee is Assistant Professor of Psychiatry, Dr. Roback is Associate Professor of Psychiatry (Psychology), and Dr. Hollender is Professor and Chairman, Department of Psychiatry.

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FEW REPORTS have appeared of two or more members of a family with crossed gender identities. It therefore seemed important to report on two anatomically male triplets (the third triplet being female) who were studied when they requested sex-reassignment surgery. It is of additional interest that a third cousin, an anatomical male with whom the triplets had had only recent, passing contact, was also being evaluated for sex change.

This paper is based on psychiatric, psychological, and chromosomal studies of the two anatomically male triplets. The sister was unwilling to be interviewed, but information about her gathered from medical records and respondents seems consistent and reliable. In addition, we reviewed records containing medical and sociodevelopmental information about the triplets be-

fore and during their placement in a foster home at the age of 3 years and interviewed an aunt who raised the children after they left the foster home at the age of 5 years. We also obtained some information about the third cousin.

FAMILY HISTORY

The mother of the triplets died 1 hour after their delivery. She was then 23 years old. Little information about her is available.

The father, a tenant farmer and laborer, drank heavily and worked irregularly. As a boy he had been kicked in the head by a mule, resulting in an injury that required the insertion of a silver plate. At the age of 34, when the triplets were 3 years old, he was admitted to a state psychiatric hospital with the diagnosis of chronic brain syndrome with psychosis due to trauma.

Little is known about the triplets' development during the first 3 years of life other than that they were often neglected. When placed in a foster home at the age of 3 years, they were physically and mentally retarded and their speech was incomprehensible. Having received no training in the use of eating utensils, they ate with their hands and scattered food over everything within reach. Since they had seldom received sufficient food, they picked up and ate scraps thrown in the yard for the dogs. The foster parents, described as an above average family, gradually taught them how to use eating utensils and how to play games.

At 5 years of age the triplets were sent to live with an aunt and uncle with whom they remained until they were 18 years old. The aunt described her husband as an alcoholic who was seldom home. Although she divorced him when the triplets were "very young" (she could not recall the exact year), she permitted him to return home for care whenever he became ill. The triplets could remember little about their uncle except that he was seldom home and when he was, he was usually inebriated. The boys felt close to the aunt's two daughters, with whom they played many feminine games (e.g., dolls and tea parties). In high school they dated boys and had many girlfriends.

CASE REPORTS

Case 1. Marvin-Maureen, who was 20 years old when interviewed, was 5 feet 8 inches tall and feminine looking. "She" had long black hair, used makeup, including lipstick and eye shadow, and wore bracelets and earrings. The overall picture was that of an attractive, perhaps even exotic-looking woman.

While in the foster home Maureen had become more outgoing and began to make friends easily. She seemed to be the favorite of her foster parents and developed a strong attachment to her foster mother.

Maureen, who had always felt feminine, began to wear her sister's clothes at the age of 7. Initially her aunt and uncle permitted her to wear only girls' underclothing; not until she was 13 was she permitted to wear feminine outer garments. Her first sexual encounters with boys occurred when she was 7 or 8 years old. She never used her penis in intercourse; she preferred anal intercourse but occasionally performed fellatio. At age 16, she was arrested for cross-dressing and placed in a juvenile detention home for 2 weeks. She was then required to attend weekly therapy sessions with a group of adult male homosexuals, lesbians, and transvestites for a 2-year probation period.

Maureen referred to herself as a "transvestyke" and mentioned her long-standing desire for sex-reassignment surgery. She stated that her life had been miserable in certain respects and would remain so until after the operation. She commented, "Then I will have a new life to live. I want to show my family that I haven't been the trashy person they thought I was." She is currently in business school and hopes to become an executive secretary. If accepted for the sex-reassignment operation, however, she plans to get a new start in another city. She wants to attend college and to become a model.

On psychological testing Maureen scored in the dull-normal range. As would be expected, there was a nonidentification with the culture's prescription for maleness and, specifically, indications of extreme femininity on the Freund Gender Identity Scale (1). Results of the Minnesota Multiphasic Personality Inventory (MMPI) suggested serious underlying pathology. There was the distinct danger that she would become cognitively disorganized under stress. Testing also revealed much clinging dependence on a maternal figure and a lifestyle best characterized as passive-dependent.

Case 2. Jack/Janice dressed as a woman during some interviews but in others (during working hours) she was dressed in male clothing with a definite feminine touch. Her build was delicate and her manner feminine but somewhat less so than Maureen's.

According to a social casework report, Janice, like Maureen, was physically and mentally retarded at 3 years of age. Like Maureen, she had had a strong attachment to her foster mother, but unlike her "sister" she could tolerate brief periods of separation without becoming unduly upset.

Janice had felt like a girl for as long as she could remember and had cross-dressed regularly since the age of 13. Like Maureen, she preferred anal intercourse, dated only men, and avoided all sexual activity involving the use of her penis. Others thought of her as homosexual, and that is how she usually regarded herself. At times, however, she considered herself a transsexual. Many of her social contacts were with bisexuals.

Although Janice was very curious about sex-reassignment procedures, she was ambivalent about having the operation. Part of the ambivalence appeared to be due to her fear of pain. She commented that if she ever had the operation she would want to be a "classy lady," not an "everyday lady." Like her sister, she believed that she would find greater acceptance in another city.

During psychological assessment Janice was anxious and guarded. She was of borderline intelligence, manifested extreme feminine gender identity, and revealed no indication of psychosis. In her character makeup, passive-dependent and hysterical features were prominent.

Case 3. Joan, the anatomically female triplet, refused to meet with us. She was described as being headstrong, stubborn, and independent. Like her siblings, she was probably

of borderline intelligence. It was reported that she lived with a laborer and had one child.

Case 4. Robert/Rennie, the 23-year-old third cousin, was a student in a beautician school when interviewed. She had met the triplets only 2 years before and had had minimal contact with them. Like her two male cousins she was slender and decidedly feminine in appearance. She wore a reddish-brown Afro wig and a small amount of makeup.

Rennie's father had died when she was 4 years old, and she had always lived with her blind mother. She had felt like a female for as long as she could remember, and she began to cross-dress as a child. At 16 she became a female impersonator. She had dated only males, and her sexual preference was anal intercourse.

Psychological assessment revealed little other than the expected elevated score on the Masculinity-Femininity scale of the MMPI. Rennie's IQ was in the average range. Projective tests revealed no serious psychopathology.

Chromosomal Studies

Chromosomal studies, using blood cell cultures on all three patients, revealed 46 XY, normal-looking male cells.

REVIEW OF THE LITERATURE

As recently as 1973 Stoller and Baker (2) wrote, "Although hundreds of male transsexuals have been reported in the literature, there have until now been no reports of more than one in a family." They then presented information about two anatomically male siblings with normal XY chromosome configurations who fitted their criteria of transsexualism. The boys' father was in the service and was home only intermittently during their formative years. As young children, both "girls" preferred female-type activities (e.g., hopscotch and jump rope) and were not discouraged from taking part in them by their mother. Similarly, in 1973 Hore and associates (3) reported on two transsexual male siblings. The "sisters" had been raised primarily by their maternal grandmother, who was described as the "dominant force" in their upbringing.

Sabalis and associates (4) described a case of three siblings who fulfilled Stoller's criteria for male transsexualism (5). Their mother was said to have had excessive contact with her children. Their natural father reportedly was absent from the home much of the time and paid little attention to the children when he was there. When the father died their mother married a man who was "effeminate, submissive, and passive and also spent a great deal of time outside the house."

Green and Stoller (6) described two pairs of monozygotic twins discordant for gender identity; one of these pairs were 8-year-old boys. The prefeminine twin, Paul, was reported to be more attractive and cuddly. Because of an illness at age 3, the year in which gender identity consolidates, Paul spent much time with his mother; his brother was engaged in sports with his father during this period. At age 4½, Paul expressed a preference for typically feminine activities such as playing with dolls, cleaning house, and playing with neighborhood girls. His brother developed conventional sex-role interests such as baseball and kite-flying.

The second set of twins were 24-year-old women. The masculine twin related that since she was the physically larger and stronger of the two, she had been expected to help her father in typically masculine jobs (e.g., roofing the house and doing stone work), while her more fragile sister was given typically feminine tasks to do around the house. Green and Stoller concluded, "Thus, different early childhood events were experienced by both the male and female co-twins, which were in accord with later manifestations of masculinity and femininity."

COMMENT

Theories concerning the cause of aberrant masculine and feminine development are diverse, ranging from prenatal to postnatal factors and from endocrinological to environmental influences. Male-to-female transsexualism has occurred in persons with a presumed male hormone deficiency, usually those with Klinefelter's syndrome (6). A particular type of early mother-child interaction has also been implicated. Stoller (5) has called attention to an excessively strong symbiotic relationship between mother and infant son, remarkable for the degree of body contact. Coupled with this closeness to the mother is the relative absence, physically and psychologically, of the father.

The history of the triplets revealed many patterns in keeping with the psychological determinants suggested by Stoller. During the first 3 years of their lives, the triplets received little mothering or fathering. The closeness of the two little boys and their foster mother, beginning at the age of 3, might be characterized as excessive. According to reports, the foster mother spent enormous amounts of time with the children, attempting to compensate for their previous deprivation and lack of socialization. Little information was available about the foster father's involvement with the children. As noted earlier, the boys tolerated separation from their foster mother poorly.

Later, another woman, the aunt, became the dominant influence in the triplets' lives. Their uncle was an alcoholic who was absent or sickly much of the time. Cross-dressing began at 7 years of age for one triplet and at age 13 for the other. It has continued to the present, with no sexual pleasure ever associated with it. Masculine behavior and interests were not manifested at any time. Sexual fantasies were always of being made love to as a woman by a man.

The findings of this study are especially interesting because of the unusualness of the subjects (triplets) and because the family dynamics coincide with those reported in the few recorded instances of more than

one case of transsexualism in a family. Thus a psychosocial explanation for the development of transsexualism has now been offered for atypical gender identity in triplets, discordant gender identity development in monozygotic twins (6), and for two (2, 3) and three transsexual siblings (4) within one family. Although the authors of these papers favored an explanation for transsexualism based on family dynamics, they also encouraged further research to assess the part played by environmental, constitutional, prenatal neuroendocrinological, and other forces on crossed gender identity.

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An Evaluation of Detoxification as an Initial Step in the Treatment of Heroin Addiction

BY AMIRAM SHEFFET, M.A., MARK QUINONES, PH.D., M.P.H., MARVIN A. LAVENHAR, PH.D., M.P.H., KATHLEEN DOYLE, AND HARVEY PRAGER, M.A.

Of 802 voluntary patients admitted to the detoxification unit of a comprehensive treatment system, 69% completed detoxification but only 9.6% of these patients sought long-term treatment. The demographic profile of detoxification patients differed somewhat from that of patients in long-term treatment. The authors suggest that new approaches, including legal pressure, are needed to induce detoxification patients to accept prolonged therapy.

THE TREATMENT approaches to the narcotic-addicted individual are varied; this is due in part to the lack of a precise understanding of the pathogenesis of addiction and to conflicting opinions on the merits of alternative therapeutic modalities (1). One of the oldest and most widely practiced initial approaches to treatment has been the use of detoxification or "unpoisoning." This approach involves prescribing decreasing daily doses of methadone to the narcotic-addicted individual in order to minimize the effects of withdrawal from the drug.

There are few data on the long-term and short-term effects of detoxification and its therapeutic value (2). The available information indicates that the value of detoxification as an effective treatment for drug dependency is questionable (3). Indeed, most data support the notion that detoxification does not reduce or eliminate the frequency of illicit drug use (4). However, Chafetz and associates (5) reported in 1962 that it was possible to engage unmotivated alcohol addicts in significant treatment by having a detoxification program available.

The Division of Drug Abuse of the Department of Preventive Medicine and Community Health of the New Jersey Medical School, Newark, maintained an inpatient detoxification unit from September 1969 to May 1973. This facility was one component of a comprehensive, multimodality treatment system that has

The authors are with the Department of Preventive Medicine and Community Health, College of Medicine and Dentistry of New Jersey, Newark, N.J. Mr. Sheffet is Associate Director and Dr. Lavenhar is Director, Division of Biostatistics. Dr. Quinones is Director, Division of Social Medicine and Training and Division of Drug Abuse. Ms. Doyle is Research Coordinator and Mr. Prager is Assistant Director, Division of Drug Abuse. Address reprint requests to Mr. Sheffet, New Jersey Medical School, 100 Bergen St., Newark, N.J. 07103.

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been in operation since 1969 through funding from the National Institute on Drug Abuse (6). The primary objective of the detoxification unit was to assist drug-dependent patients in becoming drug free before entering long-term treatment in another component of the comprehensive system.

DESCRIPTION OF THE UNIT

At its peak capacity the unit included 6 beds for men and 2 for women in the Martland Medical Center, the major teaching hospital of the New Jersey Medical School. Patients were admitted voluntarily and without legal pressure for, in most cases, a 5–7 day period. Each individual seeking admission was required to appear at the hospital during the regular working hours for a comprehensive interview on health problems and history of drug use. The demand for the unit's services usually resulted in a 2–10 day waiting period between the initial interview and admission.

Upon admission to the unit each patient was placed on daily doses of methadone in decreasing amounts. The usual initial daily dose was 40 mg, but in some cases up to 80 mg were given if the patient's history, symptoms, and physical examination suggested that 40 mg would be inadequate. Methadone was generally administered over a 5-day period.

Staffing for the unit reached a maximum of 4 registered nurses, 3 nursing assistants, and 1 ward clerk. Medical services, along with other supportive services, were coordinated by case expeditors from the drug abuse clinic. These workers were paraprofessionals with a high school diploma or equivalent and at least 2 years of experience in a drug abuse counseling program.

The physical facilities of the unit were similar to those of any traditional older hospital ward, with the men's beds in a large room and the women's beds in a smaller room across the hall. The ward was not locked; security services were provided by a uniformed private guard. All patients agreed to abide by the following restrictions: no visitors, no violence, and no drug use.

Television was the only recreational facility provided, and planned activities for the patients were minimal. One structured activity was a weekly orientation session designed to familiarize patients with treatment modalities available to them after detoxification. The available treatment alternatives included methadone maintenance, counseling for ambulatory drug-free patients, and 1 of the 3 residential therapeutic communities within the system. Four hours were set aside for each orientation session.

This study was undertaken to evaluate the performance of the detoxification unit in achieving its goal of assisting drug-dependent patients to enter long-term treatment. It was also designed to address the broader issue of the role of detoxification in a comprehensive treatment program for heroin addiction.

METHOD

The primary sources for the data base were the 6 affiliates comprising the New Jersey Medical School treatment system and the drug abuse clinic at Martland Hospital. Upon entering the detoxification unit each patient completed a drug registry form (7). Registration data were coded and punched onto tabulation cards for data processing in order to analyze the epidemiological, sociological, and medical correlates of drug addiction. A computerized system for monitoring patients was designed with the capability of providing comprehensive statistical data on the movement of patients within the confines of the treatment system. All data were screened automatically for consistency and accuracy, and errors were promptly corrected (6).

The variable-length records that were generated by the monitoring system were structured and merged with the drug registry information by means of a summarizing computer program. The *Statistical Package for the Social Sciences* (8) was used to analyze the structured files.

RESULTS

This analysis covers the time period between February 1, 1971, and May 31, 1973. During this period a total of 802 patients were admitted to the detoxification unit; of that number, 552 (69%) eventually completed detoxification. There were 1,028 individual attempts at detoxification (on the average, 1.3 attempts per patient); 226 patients entered the unit 2 or more times.

The age and race of 11 patients could not be ascertained. Of the others, almost half (44%) were black, male, and 21 years of age or older; 24.6% were black, female, and 21 years or older.

Sixty-one percent of the patients who entered detoxification as the first phase of drug treatment completed it on the first attempt. When comparing each demographic subgroup's rate of success in completing detoxification treatment with the overall success rate of 61%, it appeared that patients who were 20 years old or younger had a higher than average success rate (72%, which is statistically significant [$p<.05$, z test for standard proportions]). Students had a high (79%) success rate. On the other hand, white patients had a low (51%) success rate. We have compared other demographic variables including residency, occupation, marital status, education, duration of drug abuse, total convictions, and time in jail. Success in completing detoxification did not correlate with any of these demographic characteristics.

Only 53 (9.6%) of the 552 patients who completed the prescribed detoxification treatment sought further long-term treatment within 4 weeks after leaving the detoxification unit.

We then compared the demographic characteristics of addicts admitted to the detoxification program with those entering the 6 treatment programs under our pur-

view (treatment patients). Only 53 of the latter entered treatment after detoxification; the other 2,678 entered treatment directly. The detoxification group had greater percentages of blacks (84% versus 63% treatment patients) and of Newark residents (83% versus 48% treatment patients). Almost half of the detoxification group came from the central ward of Newark and manifested deficiencies in education and work skills. The detoxification program included proportionately more female patients than the long-term treatment group (35% versus 24%) as well as a larger group of patients 26 years of age and older (38% versus 28%).

DISCUSSION

Successful detoxification could be judged by two outcomes: completion of the prescribed course of detoxification and commitment to long-term treatment.

With 69% of the patients eventually completing the prescribed course of detoxification, the first objective appears to have been achieved. Others have proclaimed success with a far smaller percentage of patients completing detoxification (2). We were unsuccessful in regard to the second outcome objective. Our findings in this area confirm those of Glasscote and associates (1).

O'Malley and associates (4) reported that outpatient detoxification was an utter failure regardless of patients' demographic characteristics; very few patients completed detoxification, and there were no candidates for long-term drug treatment. Bass and Brown (9) implied that younger patients may be suitable candidates for detoxification followed by long-term treatment and suggested that older patients are better off in a methadone maintenance program. Aron and Daily (10) reported that 23% of their detoxification patients entered significant treatment, but 86% of these patients were under legal pressure to do so.

Renner and Rubin (2) noted that patients with a short history of heroin use and small daily habit are more likely to succeed in detoxification; they concluded that successful detoxification is related to commitment to long-term treatment. Our study does not confirm these findings. Young patients, who were the most successful in completing detoxification, showed the least interest in further treatment, and it seems that success in regard to the first outcome measurement does not imply success in the second outcome measurement.

The group of patients who sought detoxification was somewhat different demographically from the long-term treatment group. They were more likely to be black, female, older, and Newark residents. These patients entered detoxification for a variety of reasons including a true determination to stop drug use, a desire to reduce the level of addiction, inability to maintain their habit, and pressure by the courts to solicit detoxification services (4). Most of our patients were not able or ready to maintain a long-term commitment to drug treatment even though the detoxification unit was intended as a beginning of the rehabilitation process. However, the unit did provide a needed service for this particular population.

The program was useful in another respect. Although 76% of the patients who entered detoxification did not attempt further treatment or repeat detoxification as of July 31, 1974, of those detoxified patients who eventually did enter long-term treatment, 80% continued beyond the initial 4 weeks, a better than average retention rate.

We believe that our patients were often difficult to deal with in part because of lack of daily structured activities. Other than the medication times, mealtimes, and the weekly orientation sessions, structured activities were nonexistent, and this created boredom and encouraged acting out behavior. In addition, many of the personnel were not well versed in counseling techniques and therefore were not adequately equipped to deal with this difficult and demanding population.

It is obviously incumbent upon treatment staffs to continuously evaluate their programs in an attempt to improve them. We closed the detoxification unit and have now reopened it in a new environment with special attention to counseling and structured activities. It will be interesting to see whether the proportion of patients entering long-term therapy increases significantly.

The observed differences between patients in long-term treatment and those entering and completing detoxification suggest that these 2 groups represent different populations with different degrees of motivation for treatment. It thus may well be that detoxification as a part of long-term treatment will only succeed with a large proportion of patients if there are legal restrictions that compel the individual to accept more prolonged therapy.

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The Use of Problem-Oriented Medical Records in Community Mental Health Centers

BY GARRY M. VICKAR, M.D., AND MARIJAN HERJANIC, M.D.

The authors' questionnaire survey of 147 community mental health centers revealed that 36 (24%) were using problem-oriented medical records (POMR), 34 (23%) planned to use them, 23 (16%) were uncertain, and 54 (37%) had not considered using them. According to the responses of centers that were using POMR and the authors' experience, the advantages of POMR in psychiatry are similar to their advantages in other specialties, and the difficulties in implementing this system are related to the definition of "problems."

WEED'S INTRODUCTION of problem-oriented medical records (POMR) (1) has led to a reevaluation of record keeping in many medical centers. Following the adoption of this system, publications appeared endorsing POMR and encouraging their acceptance in all specialties of medicine (2–4). More recently there has been some questioning and attempts at critical evaluation of this new approach (5, 6). Less is known about the use of POMR in psychiatric practice than in other medical specialties. Considering the many advantages claimed for POMR in psychiatry (7), we were surprised to find only an occasional report of their use in this field (8, 9). It may be that psychiatrists have benefited from the experience of colleagues in other branches of medicine (10, 11) and approached the subject with caution.

The POMR was first used in the inpatient service of Malcolm Bliss Mental Health Center, St. Louis, Mo., on July 1, 1974. We wondered about the extent of its use in other psychiatric facilities and undertook a survey of other mental health centers. Using the September 1973 *Directory of Federally Funded Community Mental Health Centers* (12), we sent questionnaires to all community mental health centers in operation at the end of August 1974. We inquired about the adoption, acceptance, and advantages of POMR.[1]

Dr. Vickar is Instructor in Psychiatry and Dr. Herjanic is Associate Professor of Psychiatry, Washington University School of Medicine, 1420 Grattan St., St. Louis, Mo. 63104. Dr. Herjanic is also Assistant Superintendent, Malcolm Bliss Mental Health Center, St. Louis, Mo.

FINDINGS

Three hundred thirty-four questionnaires were sent out in August 1974; 158 (47%) were returned answered. Eleven replies had to be dropped from calculations because they were incomplete.

Thirty-six of the 147 centers (24%) were using POMR. These centers were apparently distributed randomly throughout the United States. Of these 36 centers, 19 used POMR for both inpatients and outpatients, 8 for inpatients only, and 9 for outpatients only. The average duration of use was about 12 months (1 center claimed to have been using a modified form of POMR for 27 years).

Thirty-four of the 147 centers (23%) planned to implement POMR, and 23 of the centers (16%) were uncertain. The remaining 54 centers (37%) had not considered using POMR.

Twenty-four of the 36 centers using POMR (67%) agreed that their use improved their record keeping in terms of organization, readability, and identification of problem areas. The better organization also made for easier supervision of residents and data collection for research purposes. When asked about improved patient care, the 36 respondents who were using POMR had mixed reactions, but 23 (64%) be-

[1]Copies of the questionnaire are available from Dr. Vickar on request.

lieved that patients benefited from the more consistent approach in documentation. Thirteen (36%) were not aware of a change in length of stay, but one respondent replied that length of stay increased because "people note [in the record] that [the] problem hasn't been resolved."

One of the interesting observations made by centers using POMR was that physicians were less favorably inclined to accept POMR than other members of the treatment teams. Only 9 of the 36 centers (25%) reported that the attending medical staff liked POMR. The replies expressed this through such comments as, "Approve in principle, frequently find it hard to change their own [physician's] way of doing things," "Are somewhat resistive," "It requires a higher level of medical acumen; this affects some staff who don't know what they are doing very well."

We wondered if the acceptance or rejection of POMR was related to the theoretical orientation of the institution. The 147 respondents indicated their treatment philosophy on a spectrum from purely psychoanalytic to predominantly physical therapies. There was no difference among groups with different theoretical orientations in terms of use of POMR. However, the more psychodynamically oriented centers expressed more difficulties in adapting POMR to their own use, e.g., "They produce stereotyped records that fail to yield dynamic appreciation of the patient's problems."

Aside from difficulties in adjusting individual therapeutic styles to a new system, many criticisms of POMR were directed at the extra clerical work involved, e.g.: "Some versions contain a great deal of information that may prove useless, and progress notes may require a good deal of paper shuffling." "The POMR require about twice as much writing by the doctors; we have enough trouble getting records written without doubling the work; what we need is a system that requires less writing."

DISCUSSION

After six months of using POMR on our inpatient service, our impression is that they do not add much in terms of actual patient care, despite the fact that they result in better organization of the records. Some people at Malcolm Bliss feel that POMR discourage charting because of their seemingly formalistic style, but the notes that are written within this system tend to be more complete and informative.

It appears that there is considerable interest among community mental health centers in problem-oriented medical records. One-half of the 147 centers who answered our questionnaire said they would be using this system by the end of 1975, and one-sixth were undecided. Even though one-third had no plans to introduce POMR, almost all requested the results of this survey, and many asked for practical references. With the increasing need for treatment evaluation and accountability (13), POMR may gain additional significance; they could become mandatory for all mental health centers.

Criticisms leveled at this system point out the difficulties individual therapists have in defining "problems." Obviously, the definition of a problem in psychiatry is a delicate task requiring a high degree of skill and experience. A considerable amount of reeducation and in-service training will be required to avoid overinclusion or particularization.

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IN MEMORIAM

Malcolm J. Farrell
1906–1975

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MALCOLM J. FARRELL, who died August 5, 1975, was a quiet, self-effacing man who devoted his life to improving the care of the mentally retarded. His ability to get things done led others to seek out this reluctant leader for the many top posts he filled so admirably.

We were friends throughout a lifetime, enjoying family picnics together as our children grew up, playing bridge on Sunday nights, and celebrating our wedding anniversaries on the same day. Mal and I were peers who shared the problems of running a state hospital. When he retired on April 4, 1974, he was feted at a testimonial dinner. Those who arranged the occasion knew how to please Dr. Farrell. Some 30 mentally retarded residents of the Fernald School were present and participated not just as guests but in the program as well. The major gift to Dr. Farrell was a check for a summer camp for residents of the school.

Born in Woburn, Mass., on May 7, 1906, Malcolm was the son of Joseph F. Farrell, who came to this country from Ireland to own and operate his own wholesale produce company, and of Elizabeth Thompson Farrell. Malcolm had a sister, Elizabeth (now Mrs. Spaulding of Needham, Mass.), and a brother, Robert, now living in Woburn.

When Malcolm was 14, his brother had an operation that miraculously restored him to health. Mal was so moved he vowed he would become a surgeon. Toward that goal Malcolm enrolled as a premedical student at Tufts University and stayed on course at Tufts Medical School until his fourth year, when a clerkship at Danvers State Hospital changed his career choice. After receiving his M.D. in 1931 and completing an internship at Boston's Long Island Hospital, he began what would now be a residency at Metropolitan State Hospital in Waltham, Mass. Six years later he accepted the post of assistant superintendent at the Walter E. Fernald State School. He was there about three years until World War II thrust him into a key post.

In February 1942 the Surgeon General of the Army established a separate Neuropsychiatry Branch and named Lt. Col. Patrick S. Madigan as its chief. On April 10 Capt. Malcolm J. Farrell was assigned as assistant to the chief in the developing branch. When Col. Madigan was transferred to the Adjutant General's office, the former superintendent of Metropolitan State Hospital, Col. Roy D. Halloran, was named to head the branch. Col. Halloran died suddenly on November 10, 1943 and Major Farrell acted as chief until Brig. Gen. William Menninger took command on Dec. 10, 1943.

Dr. Farrell planned and implemented the Army's neuropsychiatric hospital care system, the training of physicians in psychiatry, the assignment of psychiatrists to Army divisions, the adding of clinical psychologists and social workers to the treatment team, and the shaping of military medical policy. He wrote some of the key policy statements, lifted the blackout on neuropsychiatric matters and instituted an open press policy, and set standards for the transport of returning casualties and for the retention of personnel with neuropsychiatric disorders in the combat area for treatment. He was also involved in the refinement of diagnostic categories that led to the first edition of APA's *Diagnostic and Statistical Manual of Mental Disorders*. When the war ended Farrell was a full colonel and had been awarded the Legion of Merit for his outstanding contributions.

Farrell returned to the Fernald State School and served as its superintendent for the next 30 years. He had a master plan to build the best possible facility for the retarded, and his achievements remain for all to see in new buildings and high quality service, with teaching and research as integral parts. He inaugurated a work-release program, fostered and encouraged a parents' movement, extended the program of patient management into the community, combined education for students with vocational preparation, and established a prestigious research center. Fernald became widely known and Farrell recognized as a national expert on mental retardation whose opinions and advice were widely sought.

Shortly after World War II a group of ex-military officers met for an evening session at an APA annual meeting in Chicago. The Group for the Advancement of Psychiatry emerged out of the planning set in motion at that informal group session. The innovative notion of small groups of experts working intensively on a topic area with the aid of consultants from other disciplines has produced a series of prestigious reports. GAP also exercised enormous influence in reorganizing APA. Through the turbulent years of GAP's growth Dr. Farrell ran the fiscal and administrative affairs of the organization, serving as its secretary-treasurer from 1950 to 1969.

APA was also a major interest of Mal's. Besides serving on a number of committees, including the Committee on Certification of Mental Hospital Administrators (1958-61) and the Contract Survey Board (1962-73), he was especially active in District Branch activities and became Speaker of the Assembly of District Branches (1968-69). Dr. Farrell was attending the Assembly Executive Committee reception as

past speaker at the APA annual meeting in Hawaii when he collapsed and was flown home. It was the start of an illness (vasculitis and multiple strokes) from which he never recovered.

Dr. Farrell had many distinctions besides his GAP and APA activities: instructor at Tufts and at Boston University, bank director, and author of 68 scientific articles, mostly in the field of mental retardation.

Mal's family was very important to him. He met Virginia Corbin at a church meeting when they were teenagers; they were married on July 2, 1932. Two sons, Jay and Dudley, and his daughter, Ginny (now Mrs. Juan Blau of Panama), grew up in an extended family that gathered in friends and relations and shaped all in the Farrell moral mold.

Not seeking leadership, it sought him; avoiding the limelight, he illuminated his world. Dr. Farrell devoted much of his life to helping others and to the shaping of the institutions that serve the mentally afflicted. It is when we forget ourselves that we do things which are remembered.

WALTER E. BARTON, M.D.

LETTERS TO THE EDITOR

"Misleading" Concepts in Family Therapy

SIR: "Basic Concepts in Family Therapy: A Differential Comparison with Individual Treatment" by Leon Robinson, M.D. (October 1975 issue), is most misleading and perpetuates a polarized conception of family versus individual therapies. His basic assumption that "all individual therapies share a common perception of the nature and location of the forces most active in the formation of personality, the appearance of symptoms, and the production of change," including the beliefs that "intrapsychic events determine personality" and "changes in individual behavior are unopposed by environmental factors" is sheer nonsense. This paper perpetuates the silly either/or controversy of external versus internal determination of behavior, and its publication merely prolongs the sterile individual versus family therapy controversy (1).

Actually, most individual therapies, especially psychoanalysis, emphasize the impact of the environment on personality formation. In his first detailed case history (that of Dora), Freud noted,

> It follows from the nature of the facts which form the material of psychoanalysis that we are obliged to pay as much attention in our case histories to the purely human and social circumstances of our patients as to the somatic data and the symptoms of the disorder. Above all, our interest will be directed toward their family circumstances. (2, p. 18)

Why is there still such a reluctance in many settings to treat the family, despite the agreement of almost all theories of human behavior that family forces are among the critical determining variables in the development of personality and symptoms? The reasons for this reluctance would include the individualistic ethic of our culture, the greater practical difficulties in treating the family unit, the absence of an acceptable systematic theoretical framework for family therapy, the lack of any standard-setting association of family psychiatrists, and the as yet undeveloped mechanisms for third-party payments for this relatively new modality. This is most unfortunate, for family therapy holds special promise for the time-limited therapeutic interventions we are increasingly called upon to provide.

There is thus a responsibility for family therapists to evolve a meaningful theory, typology (3), and practice which will clarify those clinical situations most amenable to family treatment rather than continue to make puerile comparisons to the individual therapies.

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F. M. SANDER, M.D.
Bronx, N.Y.

Dr. Robinson Replies

SIR: I can only conclude that the "silly either/or controversy" to which Dr. Sander refers is drawn more from his own prior thought and experience than from the contents of my article. The fact that he goes to the trouble of citing Freud in order to assert that my basic assumptions are lacking because they do not acknowledge the idea that most individual therapies "emphasize the impact of the environment on personality formation" suggests either a biased or inaccurate reading of my paper. I am led to this conclusion because I clearly stated in my article that "the importance of environmental factors in the shaping of personality is recognized by the individual therapies" (p. 1045).

As far as I can see, the major difference between my position on this subject and that of Dr. Sander has to do only with the question of emphasis. I take no either/or positions on family therapy versus individual therapy or on external versus internal determination of behavior. I believe, however, that while family therapy and the individual therapies do operate at shared levels of awareness regarding personality, symptom formation, and therapy, they also emphasize different aspects of these broad conceptual areas. This difference in emphasis is the basis of the ultimate difference in methodology. Dr. Sander's inference that "individual therapies, especially psychoanalysis" emphasize family and environmental factors as much as they emphasize intrapsychic events and cannot be differentially compared with family therapy at this level is poorly founded. His reference to Freud's case history of Dora does not offer much support for his contention because in that paper Freud stated in a footnote (1, p. 20) his belief that the principal causes of his patient's illness were a matter of heredity and constitutional predisposition. I do not see how this can be construed as an emphasis on the impact of environment.

I think the question of emphasis was well explained by Ackerman, who said, "Psychoanalytic treatment focuses on the internal manifestations of disorder of the individual personality. Family treatment focuses on the behavior disorders of a system of interacting personalities, the family group" (2). I concur with Ackerman that a discussion of differences is no cause to view the two approaches as "competitive or mutually exclusive."

I therefore cannot agree with Dr. Sander either in his denial of significant differences between family and individual therapy in their emphasis on environmental forces or in his

statement that attending to such differences stimulates only controversy. I submit that the differences in perspective I referred to in my article not only exist but are worthy of exploration and clarification as a means of facilitating communication concerning the shift in focus required when a person trained in the individual perspective wishes to work with families.

My personal experience in using this kind of comparative approach to introducing family therapy concepts to people who have previously worked only with individuals is that the comparisons are received as useful and do not tend to create controversy.

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LEON R. ROBINSON, M.D.
Philadelphia, Pa.

A Problem of Methodology?

SIR: I am writing to call attention to serious deficiencies in "Methylphenidate and Caffeine in the Treatment of Children with Minimal Brain Dysfunction" by Barry Garfinkel, M.D., and associates (July 1975 issue). These deficiencies are so serious, in fact, that our entire staff at the Ontario County Mental Health Center expressed surprise that the paper was published.

First, the study involved only eight children—hardly an adequate sample. Second (and even more crucial), the dosages of the drugs used were fixed at the same level for all of the children, and they were very low. It is abundantly clear to anyone who has worked extensively with drug treatment of children with minimal brain dysfunction that dosages must be carefully adjusted to fit the individual child. To anyone who has experimented with caffeine for these children, it is equally clear that a dosage of less than 200 mg/day is unlikely to produce worthwhile effects. At our clinic, we have used caffeine with a few children and have seen some good results at levels of approximately 400 mg/day. While our experience with caffeine has not been encouraging enough to lead to its replacing methylphenidate, pemoline, and *d*-amphetamine in our armamentarium, we have felt handicapped by the lack of any reliable work on dosage schedules for its use. The article in question—which has such a promising title—certainly does nothing to answer our questions.

ALLEN W. MARINER, M.D.
Canandaigua, N.Y.

Dr. Garfinkel and Associates Reply

SIR: Regarding the first issue raised by Dr. Mariner, the size of our sample, it is never easy to decide what constitutes an "adequate" sample. If the experimental control over the major variables is inclined to be weak, sample size usually needs to be fairly large. If the experimental control over major factors is strong, the sample size can be very small indeed. Workers in the tradition of radical behaviorism (operant conditioning) have, for example, long eschewed the use of formal statistics, preferring to show the power of variables by the effects they produce on the behavior of a single organism. While it is certainly the case that, in general, drugs are not so easy to manipulate as many environmental variables, it is frequently possible to demonstrate strong effects with relatively small samples. The fact that we were able to show a marked methylphenidate effect with a sample of eight children merely confirms this point. Sample size in itself is, however, not the real issue. If the investigator opts for a small-sample design, he should be prepared to present a reasonably fine grain of data analysis. In such cases, the reader is perhaps entitled to know how the drug or other variable affected each individual subject. This issue, we sense, is the real basis of Dr. Mariner's objection; fortunately it is one we can answer.

We had in fact anticipated this objection. On page 725 of our paper, we devoted a short paragraph to the importance of the effects of the drugs on the performance of individual children. We stated,

> While the overall statistical analysis gives the main important trends, it is interesting to note that individual children demonstrated both a dramatic therapeutic response to methylphenidate and a pharmacological effect from caffeine, although the latter was not as marked.

In our original version of the paper, we had included some sample data drawn from individual children. While it was not possible to include these data in the published version due to space limitations, individual records rather than formal statistical analysis are available in another publication (1). It is good to be able to turn a "serious objection" into an opportunity to provide a colleague with the detailed information he requires.

Dr. Mariner's second objection, that we did not adjust the dosage to fit each individual child, is easy to understand in principle; however, it makes little impact in practice. He is perfectly correct to point out that fixed-dosage studies are inherently weak, especially when dosages have not been adjusted to fit each individual child. It would indeed have been better had we adjusted the dosage level to fit each child, but this would have added considerably to the administrative complexity of the study. Since the children's weights ranged from 72 to 80 pounds, we can hardly be criticized too strongly for not adjusting the dose.

We feel that the use of a single dosage is the only objection possibly meriting the adjective "serious." Again, we anticipated this objection by pointing out the need for exploring the effects of varying doses of caffeine. Obviously, it is much better to vary dosage systematically than to use a single level. However, it is often impossible to accommodate all the niceties of experimental design within a single study. Schnackenberg's article (2) was valuable because it pointed out an important observation: our study is an improvement on Schnackenberg's but is not methodologically perfect. Our latest study, which examines dose-effect relations of caffeine in children with minimal brain dysfunction, is more advanced in design than the study Dr. Mariner refers to. This double-blind crossover study analyzed behavioral changes in response to 150 mg and 300 mg of caffeine alone and in combination with a fixed dosage of methylphenidate. (Prelim-

inary analysis of our results has been completed and a manuscript will soon be submitted for publication.)

If Dr. Mariner's criticism spurs us or others on to attempt further studies, it will have been well placed.

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B. D. GARFINKEL, M.D.
Toronto, Ont., Canada
C.D. WEBSTER, PH.D.
Victoria, B.C., Canada
L. SLOMAN, M.R.C.S., L.R.C.P.
Toronto, Ont., Canada

The Need for Insect Sting Kits

SIR: I am engaged in what seems at times to be a one-man crusade. As an allergist, I am acutely aware of the life-or-death situations that can be created by insect stings. I have run into several cases where death could have been prevented if an emergency insect sting kit had been available.

We physicians give insect sting kits to patients who need them, and we train them in their use. Yet people in positions of responsibility—school nurses or principals, forest rangers, Scout masters, and others who may encounter people in allergic shock—do not have the kits, nor do they know how to use them. Most state laws prohibit sales of such kits without prescription. I think state laws should be changed so that persons in the kinds of positions I have listed above, after receiving appropriate training, could purchase the kits without prescription. I hope others feel likewise and will be motivated to write their legislators to change the laws.

I would also appreciate hearing from other doctors who know of cases where the use of an insect sting kit would have prevented death.

CLAUDE A. FRAZIER, M.D., P.A.
Doctors Park
Bldg. 4-C
Asheville, N.C. 28801

Diagnosis and Treatment in a Case of Tardive Dyskinesia

SIR: In "Deanol in the Treatment of Tardive Dyskinesia" (August 1975 issue), Daniel E. Casey, M.D., and Duane Denney, M.D., appear to have adequately documented that their patient had this disorder. What is unclear, however, is whether this man was initially diagnosed correctly, i.e., did he have schizophrenia? There are several reasons to doubt this diagnosis. First, he was described as being without psychiatric history until 1968, when we surmise he would have been 54 years old; such a late onset is uncommon in schizophrenia and would be more likely to indicate a depressive illness. Second, he was described as "mildly despondent . . . agitated . . . mood was depressed . . . affective responses were appropriate . . . no evidence of thought disorder." Thus there appear to be positive findings of depressive illness and no evidence of schizophrenia. Third, his initial presenting symptoms are described as "social withdrawal, agitation, and inability to think clearly." These complaints are not pathognomonic of schizophrenia by any means but could be accounted for by a severe depression.

Even if this patient was correctly diagnosed as having schizophrenia, a number of questions remain about his course of treatment. He was treated with a combination of chlorpromazine and trifluoperazine; there is no proven benefit of such a combination (1). The course of medication was continued over a long period of time (5 years or so) without apparent attention having been given to drug holidays or discontinuance until the authors assumed his care. This is difficult to understand given the historical information in the article itself and current guidelines for psychopharmacology (2, 3).

In essence, our concern is with the inadequate application of diagnostic criteria and a cogent treatment plan. One would hope that such positive steps would lower the incidence of neuroleptic-induced disorders and obviate the need for experimental pharmacotherapy, i.e., prevent the condition rather than treat it.

A final point regarding the proposed presence of an antagonistic cholinergic mechanism in the nigrostriatal pathways: if deanol, an acetylcholine precursor, improved the patient's condition, how does one account for the failure of anticholinergic drugs to worsen his neurological condition?

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ROBERT A. KUWIK, M.D.
MELVIN J. STEINHART, M.D.
Albany, N.Y.

Drs. Casey and Denney Reply

SIR: The inquiry of Drs. Kuwik and Steinhart primarily addresses basic issues in patient care. We heartily agree that it is imperative to apply diagnostic criteria critically and to formulate a treatment plan based on indicated therapeutic guidelines appropriate to the diagnosis. Although we have the benefit of hindsight, our clinical impression was also that of an affective disorder rather than schizophrenia. Unfortunately, our clinical contact with this patient began in 1973, and his neurological complication was discovered in the course of diagnostic evaluation directed to the very issues raised by Drs. Kuwik and Steinhart.

Prevention of tardive dyskinesia is a primary responsibility of each physician. Currently, we advocate using neuroleptic drugs only for indicated conditions, at the lowest effective dose, with periodic reassessments, and with drug holidays whenever possible. In spite of these efforts, some patients will develop tardive dyskinesia. At an appropriate time, a candid discussion with the patient and the family regarding the risks and benefits of pharmacotherapy may assist

in the management of this unfortunate condition.

The role of cholinergic mechanisms in tardive dyskinesia is not completely understood. The proposed balance between dopaminergic and cholinergic influences implies that excesses or deficits in either could effect a change in the clinical condition. While deanol in large doses benefitted this particular patient, the anticholinergic agent was apparently insufficient to aggravate the dyskinesia. There are inadequate pharmacokinetic data to account for the variations seen in the response of patients with tardive dyskinesia to a variety of cholinergic and dopaminergic agonists and antagonists. Indeed, the "balance hypothesis" itself must be considered tentative at this point.

We hope that further research will lead to both treatment and preventive strategies for this unfortunate iatrogenic disorder.

DANIEL E. CASEY, M.D.
Providence, R.I.
DUANE DENNEY, M.D.
Portland, Ore.

Semantics and Psychiatry

SIR: I enjoyed "Affect, Mood, Emotion, and Feeling: Semantic Considerations" by Richard Ketai, M.D. (November 1975 issue). It represents a much needed attempt at clarifying these terms more precisely and pointing out some of the ambiguities in their current usage. I have no major argument with the author's commendable concluding remarks and the potential usefulness of his proposed classification, but I would like to make a few comments.

Obviously, as the title of the article clearly implies, Dr. Ketai chose to treat his subject explicitly and selectively on a semantically inspired, descriptive-phenomenological level rather than focusing more specifically on the terms' underlying neurophysiological bases and related conceptual considerations. The latter considerations, however, may be just as important as the former or even more so.

In analyzing the meaning of a word, it is always a good idea to go back to its original semantic root (usually Greek or Latin), as this more often than not represents the percolated cognitive wisdom and condensed perceptions of many antecedent generations. The term "affect," for instance, comes from the Latin *afficere*, "to do something to, exert influence on"—which is exactly what the "affect" does through its various physiological substrates and functional components within the respective parts of the thalamus and the limbic system as these influence, pervade, and *affect* in varying degrees subsequent emotional and behavioral responses of a higher phylogenetic order. In this frame of reference, the more primitive, instinctually overriding, id-related nature of the affect is also illustrated by its enhancement, lack, or depletion in such (biochemically determined?) disorders as manic-depressive illness, schizophrenia, and severe endogenous depression. In these disorders, the patient's affectivity and its pathology becomes much more an issue of medical-clinical understanding than of semantic definition.

Similar consideration—although perhaps to a less primarily "biological" degree—can be applied to the emotions and their psychophysiological accompaniments, as well as their practical implications for the practice of psychosomatic medicine. Mood and feeling, on the other hand, are, I believe, much more closely linked to higher cortical functions, which makes them more specifically human phenomena (and therefore part of the general human condition) as to their psychological and psychopathological nature and propensities.

In saying all this, I am conscious of partially disregarding or even neglecting some analytic formulations and theoretical definitions such as those quoted by Dr. Ketai; I am also quite certain that Dr. Ketai, as a practicing clinician, is well aware of these implicit aspects of his topic. However, they seem worth mentioning and emphasizing because in psychiatry we often tend to get caught up in semantics, arguing about words and descriptive terminology rather than focusing our attention on scientifically documented facts and on scrutinizing the validity of our concepts and theoretical formulations.

F. PAUL KOSBAB, M.D.
Northport, N.Y.

Dr. Ketai Replies

SIR: There seems to be little disagreement between Dr. Kosbab and myself about the subject of affectivity. His discussion of affect, mood, emotion, and feeling does not contradict the outlined categorization presented in my article. Certainly, mood and feeling may, among other things, be considered specifically human phenomena, and affect (affective sensations, to be precise) may be seen as a more primitive stimulus for higher order behavior. These descriptions are not inconsistent with my proposed classification, which is based on temporality, subjective-objective perspectives, and distinctions between what exists and how it manifests.

Dr. Kosbab correctly points out that labeling something does not necessarily ensure that we will understand it better. In psychiatry, we are particularly guilty of obscuring understanding with words, especially through the use of jargon.

As far as I am aware, the only ways human beings have to communicate are through words, including the affective display behind spoken words, and body language. Communication by ESP is unproven or at least debatable. Imperfect as words may be, we must depend on them to augment communication and understanding. Without them, Dr. Kosbab and I would not be having this dialogue.

Therefore, to keep communication as unmuddled as possible, words must be used precisely. This requires definitions and distinctions, which was the purpose of my article. I agree with Dr. Kosbab that words should not be reified or substituted for understanding of concepts. Semantic agreement is necessary, however, to begin to communicate better and, eventually, to lead to understanding.

RICHARD KETAI, M.D.
Ann Arbor, Mich.

A White-Coated Tale

SIR: There has been a great deal of debate in recent psychoanalytic literature regarding certain strictly technical fine points in the analytic method. A recent article in that bastion of psychologic know-how, *The New Yorker*, raised the issue of the use of the analytic napkin. Other issues raised include

the choice of pipe versus cigar in the psychoanalytic hour, the bow tie versus the straight knotted tie, and the psychoanalyst's day off, which has, since Freud's time, traditionally been Thursday. Nowhere in the literature, however, has anyone addressed the question of whether or not a psychiatrist should wear a white coat while doing therapy.

We know that in his early years, Freud wore a long white laboratory coat in his consulting room. Only in his middle and late years did he begin wearing a three-piece suit. Since Freud never wrote about this issue, we are left in the dark as to what we psychiatrists should wear. In researching this sartorial issue, I chanced upon a plausible answer to the question, and I am pleased to be able to share it with you.

Having traveled to New York City recently to attend a psychodrama convention and finding myself arbitrarily thrown out for supposed lack of dramatic skill, I found myself wandering in the rain on Manhattan's Lower East Side. At the suggestion of one of my supervisors, I had thought to have cuffs put on a pair of old tweed pants. When I happened upon a small tailor shop, I decided to grab the moment and go in. The tailor was a wise-looking Jew in his mid-eighties. As he worked, we made conversation over the tea and *schnecken* he kindly provided, and I soon found out that he was born in Vienna. I asked him if he had known Freud, and to my utter amazement and good fortune, he said that not only had he known Freud but had been his personal tailor.

Here at last was my opportunity to answer the burning question that so often plagued me. I wasted no time and asked him immediately about Freud's having changed from the white coat of his early years to a three-piece suit. The tailor told me the following tale, which he swore to be the truth.

Apparently, in his early years of practice Freud was considered quite a fashionable dresser in Viennese circles (as a matter of fact, he was twice voted the best dressed analyst in Vienna by the Viennese Analytic Society). The tailor said that the Professor had a penchant for silk shirts, wide wale corduroy pants, long waistcoats, and fur-trimmed overcoats, all of which the tailor made personally. The tailor was also occasionally asked to reweave a hole in one of Freud's white coats; he said that although Freud was a brilliant thinker, he was sometimes absent-minded and put a lit cigar in his pocket. In any case, Freud never wore three-piece suits in the consulting room.

One fine spring day in the last part of the nineteenth century (the tailor could not be more specific), Freud entered the tailor's shop and demanded his full attention. He said that he had decided to purchase a whole new wardrobe for use only in his work. He was through with white coats in the consultation room. Instead, he wanted a dozen three-piece suits made from the finest material to be found, and he stated that the vests must be multicolored. He said he would return in one week to claim his new wardrobe.

The tailor was, as you can imagine, flabbergasted—he had known Mrs. Freud to be somewhat fickle in her taste for fashion, but never the Professor. "These psychiatrists," the tailor thought out loud, "they're all *meshugah*." What could have brought about such a drastic change in haberdashery style? The tailor, being somewhat of an amateur psychologist himself, as well as subscribing to the Austrian edition of *Psychology Today*, thought he knew the answer. Freud, he reasoned (without any particular logic), must be going through his change of life. He resolved to ask Freud about this at their next meeting.

When Freud appeared a week later, the tailor said, "Well, Sigmund, I have known you 10 years now. You are a good dresser. Never have you worn a three-piece suit in the analytic hour—only Victorians wear three-piece suits." Freud responded with a question of his own—as he so often did. He asked the tailor if he read the Bible. The tailor said that unfortunately he had no time for religion, but he remembered a little from his 5 years of Hebrew school as a child. "So," said Freud, "you surely know the story of Joseph and his multicolored vest." Freud then explained how, while doing research for his book *Moses and Monotheism*, he had chanced upon the story of Joseph. Joseph's claim to fame, as you will recall, was his prowess as an interpreter of dreams. He far surpassed the local class of soothsayers, magicians, and wise men. He was often seen around Canaan dressed in a marvelous multicolored vest given him by his father.

From the tailor's description, it appears that after reading about Joseph and his fame as a dream interpreter, Freud gave up his identification with Moses and, along with it, his idea that he was destined to climb the Matterhorn, dressed in a white robe, to receive a set of Ten New Commandments from God. Instead, he formed an intense identification with Joseph, whom he had displaced as the most famous dream interpreter in over three millenia. From that time on, Freud never wore a white coat, but was instead seen in the most stylish of multicolored vests. This is why strict Freudian analysts today no longer wear white coats, but rather three-piece suits with multicolored vests.

RONALD J. BLANK, M.D.
Ann Arbor, Mich.

LETTERS TO THE EDITOR are welcomed and will be published, if found suitable, as space permits. Like other material submitted for publication, they must be typewritten double-spaced. Letters should not exceed 500 words, including references, unless a special arrangement has been made with the Editor, and they will be subject to the usual editing. Receipt of letters will not ordinarily be acknowledged.

BOOK REVIEWS

The Working Brain: An Introduction to Neuropsychology, *by A.R. Luria; translated by Basil Haigh. New York, N.Y., Basic Books, 1974, 371 pp., $12.50.*

Luria's Neuropsychological Investigation: Text, Manual, and Test Cards, *by Anne-Lise Christensen. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1975, text: 203 pp., $24.00 (paper); manual: 55 pp., $7.95 (paper); test cards: 4 sets, $40.00.*

Prof. Aleksandr Romanovich Luria is one of the U.S.S.R.'s most distinguished scientists; his pioneer studies of brain function have earned him worldwide acclaim. He is a member of the Academy of Pedagogical Science of the U.S.S.R. and a foreign associate member of the U.S. National Academy of Science and the U.S. National Academy of Education.

Fortunately Luria's most important writings have been published in English: *Traumatic Aphasia* (1), *Restoration of Function After Brain Injury* (2), *The Role of Speech in Regulation of Normal and Abnormal Behaviour* (3), *Higher Cortical Functions in Man* (4), *Human Brain and Psychological Processes* (5), *The Mind of a Mnemonist* (6), and *The Man with a Shattered World* (7).

The title *The Working Brain* is well chosen. Luria attempts in this book to correlate cortical structure and function according to a unitary thesis. Despite the brain's architectural complexity and the differences among its individual systems, the brain's functions (its "works") are not localized. Rather, they are distributed in broader areas. The contribution of each cortical zone to the whole organization is very specific. Consequently, the behavioral effect produced by each zone is very specific. Therefore, a qualitative neuropsychological analysis must be performed to assess the impairment of cerebral function.

A detailed description of the neuropsychological techniques used by Luria and his collaborators at the Burdenko Neurosurgical Institute in Moscow is available in the work of Anne-Lise Christensen, a Danish clinical psychologist. Her English version of Luria's text is composed of three parts. The first is Luria's own summary of the functional rationale he has developed to specify the "qualification of the [brain] defect" over the last 40 years. The second part describes and illustrates the details of the test procedures used by Luria and associates to examine motor, acoustico-motor, cutaneous, kinesthetic, and visual functions. Tests for the examination of speech, writing, and arithmetic skill as well as mnemonic and intellectual processes are also described. The third part of the text consists of thumbnail sketches of patients whose functional impairments demonstrate various brain lesions. Christensen also provides a manual that contains typical test wordings that are used to elicit responses to a set of cards and pictures. The test cards are also available in a plastic box.

In his conclusion to *The Working Brain* Luria echoes the point he and other contributors to the growing science of neuropsychology have repeatedly stressed: the analysis of psychological processes relies on a qualitative empirical approach that adduces correlations of function with structure.

Although it is becoming progressively more difficult to keep abreast of the burgeonings in all of the disciplines that have multisected the study of human behavior, seminal summaries such as *The Working Brain* are recommended for those of us who struggle to attain the accolade of compleat psychiatrist.

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HOWARD P. ROME, M.D.
Rochester, Minn.

Ganja in Jamaica: A Medical Anthropological Study of Chronic Marihuana Use, *by Vera Rubin and Lambros Comitas. The Hague, The Netherlands, Mouton (Scotch Plains, N.J., Mouton/Macfarland, distributor), 1975, 205 pp., $9.95.*

This is one of the most interesting, reliable, and comprehensive studies of the complex relationships involved in the use of cannabis that has yet been published. It is the result of a study sponsored by the Research Institute for the Study of Man in collaboration with the University of the West Indies and supported by the National Institute of Mental Health. The project took 18 months to complete and a large proportion of the time of 45 staff members and consultants.

It is quite probable that cannabis (or "ganja," as it is called in Jamaica) is used with greater intensity in Jamaica than anywhere else in the world, certainly in the Western hemisphere. An extensive study of 30 ganja smokers and 30 nonsmokers, all of whom were working class native men, revealed no significant differences between the two groups. There was also little correlation between the use of ganja and crime. The effects of ganja smoking on the lung's function were thought to be similar to those of tobacco smoking. The incidence of alcoholism was found to be quite low among ganja smokers, supporting the benevolent alternative theory.

The general findings and conclusions of this study are consistent with those of the Indian Hemp Commission in 1893, the LaGuardia Committee of the New York Academy of Medicine in 1944, the LeDain Commission of Canada in 1973, and the National Commission on Marihuana and Drug Abuse of the United States in 1972.

It now seems apparent that the facts to support more rational attitudes toward marijuana are plentiful; what remains is the development of social policies that will not harm users of the drug more than the drug itself does.

This volume is highly recommended to anyone who wants to get an accurate picture of the nature of the effects of marijuana on human beings as well as its role in social and cultural interactions.

DANA L. FARNSWORTH, M.D.
Boston, Mass.

On Not Knowing How To Live, *by Allen Wheelis. New York, N.Y., Harper & Row, 1975, 114 pp., $5.95.*

It is hard to categorize this slim volume. Possibly it fits best into the genre of obiter dicta, a genre that was prevalent in the last century but is now nearly extinct. An amalgam of philosophical reflection, literary comment, and narrative vignettes (whether fictional, autobiographical, or a mixture of both is not always clear), this brief text might have been transcribed from the author's private journal-cum-commonplace book. As such it is an intensely personal document. Indeed, at times, one feels as if one were listening to free associations from the couch—except that free associations rarely come with such polish.

Wheelis writes well, his prose not infrequently bordering on the poetic in phrasing and imagery that evoke a resonating chord of recognition in the reader. Only rarely does he come close to the shoals of sentimentality—a danger that particularly awaits the author who sails the seas of Weltschmerz. In a manner often reminiscent of Amiel, Wheelis writes sensitively and affectingly of the unfulfilled promises of youth and the sad ironies of human life and love. This is in many ways a melancholy book, but an honest one. It should find a sympathetic ear in those who are introspective by profession or inclination.

JOHN C. NEMIAH, M.D.
Boston, Mass.

Five Lives at Harvard: Personality Change During College, *by Stanley H. King. Cambridge, Mass., Harvard University Press, 1973, 219 pp., $7.95.*

Five Lives at Harvard is the first book to be published as a result of the Harvard student study, a major longitudinal study of personality development during late adolescence and early adulthood. The complexity of this interdisciplinary research (the research team consisted of representatives from sociology, statistics, clinical psychology, social psychology, psychiatry, and psychoanalysis) is reflected in the four objectives: 1) investigation of the process of personality change and stability as this is related to personality structure and interaction with the college, 2) study of the socializing and allocating functions of the college as an organization in society, 3) development of research methods and general research strategies, and 4) development of theory. This last objective is particularly important. According to King, the study was unable to proceed on the basis of hypothesis testing based on well-established theory because such theory did not exist.

As a report of one phase of the Harvard student study, this book makes a number of significant contributions methodologically, through an unusually thorough application of the clinical method; theoretically, through an explication of the applicability of a number of developmental constructs to the course of late adolescent and early adult growth processes; and empirically, as the basic findings provide important evidence in support of the "continuity" model of growth.

In light of the researchers' extensive reliance on the clinical method, their choice of a particular theory as a framework for analysis is particularly important. The team drew upon psychoanalytic theory, beginning with Freud's exposition of ego psychology and continuing with subsequent elaborations of adaptive aspects of ego functioning.

King and his colleagues came to two major conclusions in terms of the adaptation constructs. First, they found that individuals in this age group differed in "adaptive sense": some students seemed to know better where they were going and how "behavior at one life phase will mesh with behavior at a later life phase." A second conclusion was that students showed more instances of successful adaptation, as King defined it, as they progressed from their freshman to their senior year.

This general theoretical set of ego psychology and adaptation led directly to a consideration of the competing "crisis" and "continuity" models of development. In an important contribution to the understanding of how the crisis model relates to late adolescent development, King distinguishes four variations of the crisis model, two of which involve a new integration and can lead to adaptation. He identifies the first and most dramatic as that of upheaval and fragmentation, followed by reorganization on a new level. With recovery from the crisis comes a more realistic self-appraisal. In the second variation, disruption is followed by reorganization on a new level, but it is attained only slowly and painstakingly. King notes, "The person moves ahead in some areas, then seems to lose ground again for awhile. Change may be slow to appear and some years may go by before the reorganization is complete" (p. 30). The "sophomore slump" and the "identity crisis" are popularized versions of this second variation of the crisis model.

A third variation occurs when disruption and upheaval do not lead to a reintegration at a new level but cause a return to some kind or level of personality organization that existed before the crisis occurred. The person may go from crisis to crisis with minimal if any developmental change. King indicates that the common parlance for this variation is "perpetual adolescent."

The fourth variation occurs when the crisis is followed by regression and deterioration. Psychopathology is usually severe in this instance. College can awaken old unresolved issues; a relatively minor problem can become the basis for a major crisis. King states that only a few students in the study were in this category.

The following key findings of the Harvard student study bear directly on the crisis model versus the continuity model:

1. The majority of the students in the sample gave no evidence of having experienced the disruption and turmoil characteristic of the adolescent crisis. King notes that most students did not "feel" that they had gone through crisis (p. 32).

2. King states, "Significant growth occurs more frequently

through finding new applications for successful behavior patterns from the past, through increasing the repertoire of responses, and through modifying and reworking patterns from the past to meet new situations" (p. 32). Thus growth is likely to be unobtrusive in nature because it occurs through a series of small changes.

3. Growth occurs when a person learns how to transfer effective automatic patterns of the past to new situations, a pattern that King calls "growth by extension." In this pattern, common features of quite different situations are recognized (p. 33).

4. Growth occurs when responses that are based on successful experiences from the past are changed to fit the characteristics of new situations. This "growth by modification" pattern is an important sharpening or focusing process in that a person gets deeply involved with something and discovers new ideas and interests or reworks old values as guidelines for present conduct (p. 33).

5. The final pattern identified is "growth by accretion and exploration," in which growth occurs by the "development of new patterns of behavior related only indirectly to material from the past, but in the indirectness there is continuity" (p. 34).

There are a number of important implications of the work of King and associates. If we assume that the continuity model is the basic model of growth in college students, this fact has major unexplored implications for modification of the college experience. What are the curricular implications of growth by extension, growth by modification, and growth by accretion and exploration? What college environments will best facilitate what types of growth? What are the implications for the out-of-class learning environment of a college?

King and associates have completed a pioneering project. Its implications have just begun to be explored. The book is a must for all who are interested in the nature of late adolescent and early adult development and in the learning environments of colleges.

JOHN M. WHITELEY, M.A.ED., ED.D.
Irvine, Calif.

Gesell and Amatruda's Developmental Diagnosis: The Evaluation and Management of Normal and Abnormal Neuropsychologic Development in Infancy and Early Childhood, 3rd ed., *edited by Hilda Knobloch, M.D., and Benjamin Pasamanick, M.D., New York, N.Y., Harper & Row, 1974, 514 pp., $14.95.*

Through the efforts of two of their former students, the legacy of Arnold Gesell and Catherine Amatruda is embodied in this new edition of their classic work on developmental diagnosis.

The appearance of the third edition 25 years after the second signifies the durability of the Yale Child Study Center's early productivity as well as the relative lack of clinically useful advances in the field of child development research. An even more telling point is made by the editors when they point out, "In spite of the lip service that has been paid to the importance of development, there has been little advance in its practical incorporation into pediatric training and practice."

This volume represents a truly new edition with both additions and revisions in content. Not only is the scope of child development extended to preconception, but the developmental norms have been continued through the fourth and fifth years of life. Of particular interest is the inclusion of data validating the criteria employed at each age level. The current utility of the Gesell scales is substantiated by high reliability in administration and by a clinically significant level of predictability. A shift in attitude toward children with mental handicaps is reflected by the replacement of such terms as "brain injury" by "brain dysfunction" and "amentia" by "mental subnormality."

The bias of Gesell's work is reflected through the exclusive focus of the book on child-centered diagnostic procedures. His caveat that psychodynamic formulations have been overemphasized in developmental work is reflected in the philosophy of the editors. Although mentioned in passing, critical interpersonal transactions between parents (particularly the mother) and child are ignored. The young child is still regarded as a self-contained unit rather than an individuating organism. The stance of the book also perpetuates a distinction between heredity and environment without recognition of the influence of function on the structure of the central nervous system. A further difficulty is the lack of inclusion of the growing body of knowledge regarding individual differences. Pasamanick's continuum of reproductive casualty, although useful, continues the viewpoint that deviance in behavior is based on chronic organic brain disorder and not on individual variations.

Perhaps the most significant flaw in this volume is the unchanged attitude toward the clinical aspects of adoption. Although a one-year probationary period before the finalization of adoption is appropriately advised, the concept of boarding foster care in order to ensure a developmental diagnostic study before placement of the infant is perpetuated. Ignored in the process is the infant's need for early placement in a stable caretaking home.

Of great value is a revised and practical developmental screening inventory for use with children between the ages of 4 weeks and 18 months as a means of recording the developmental progress of children in pediatric practice.

The third edition of *Developmental Diagnosis* remains a necessity for those engaged in the primary care of infants and a useful orientation to developmental diagnosis for the child psychiatry trainee. As an easily read, coherently organized, and thorough treatment of the neurobehavioral development of the young child, it deserves a place in the libraries of all professionals dealing with young children.

JACK C. WESTMAN, M.D.
Madison, Wis.

Going Sane: An Introduction to Feeling Therapy, *by Joseph Hart, Ph.D., Richard Corriere, Ph.D., and Jerry Binder, M.A. New York, N.Y., Jason Aronson, 1975, 464 pp., $12.50.*

The distinguished semanticist H. Dumpty once observed, "When *I* use a word, it means just what I choose it to mean—neither more nor less." The reader of this book, an introduction to feeling therapy, must keep this principle in mind and recognize that the authors have chosen to have a number of familiar words mean something other than what most of us usually mean by them.

For example: "Insanity is living with less. It is the way most people live, but seldom recognize." It is also "merely stopping oneself from being." Bedwetting, overeating, chronic smoking, homosexuality, and depression (among other conditions) are points on a continuum between com-

plete and incomplete feeling. They are fictitious ailments created by labeling, because there is "only one ailment and it is insanity—living from incomplete mixed up feelings."

To rid oneself of reasonable insanity one might seek a therapy that is "not just another theory or a new set of techniques, but a way of life," and "since 'therapy' is taking care of oneself, it makes no sense to ask that a human being would ever want to stop taking care of himself."

Feeling therapy is more nearly akin to R.D. Laing's antipsychiatry than to any other mode of treatment. It is a long-term process that begins with actively encouraged abreaction and ends with integration and transformation. The authors believe, probably correctly, that one could not apply their feeling exercises and techniques outside the total context of feeling therapy as a part of an eclectic method. They also emphasize that "Feeling Therapy is not a technique or a theory; it is a community. When the techniques are practiced without the community they will not have the same effects." Feeling therapy is described as a community in which the therapists live and work together; patients who join this community live and work wherever they want within the particular city. In this community and in this context therapists are patients and patients become therapists.

Among the most interesting features of this book are the lengthy transcriptions of therapeutic interviews. Unfortunately, one has little information about the individuals interviewed or their problems beyond the facts that emerge in the interviews themselves.

This book is addressed to the general public as well as to professionals; it is meant to give an overview of feeling therapy. The academic reader who desires data or "proof" is referred to the authors' research articles and monographs.

People who want feeling therapy will find instructions for applying for it in the appendix. One must submit an autobiography and take a number of psychological tests. Sometimes conditions are set for entering this therapy; for example, a woman who wrote that she desired therapy for obesity (lifelong) was told that she would be accepted as soon as she lost 30 pounds.

If one wishes to become a feeling therapist, he or she may apply for a residency training program. This use of the term "residency" may reflect the influence of the medical model or may merely be a manifestation of the authors' tendency to "use clinical words because they evoke beliefs about us and what we do." At any rate the program is open to professionals and paraprofessionals who have completed at least one year of therapy at the feeling therapy center. Community training programs are open to groups of professionals who want to start their own therapeutic communities.

MERRILL T. EATON, M.D.
Omaha, Neb.

Mental Deficiency: The Changing Outlook, 3rd ed., *edited by Ann M. Clarke and A.D.B. Clarke. New York, N.Y., Free Press (Macmillan Publishing Co.), 1975, 856 pp., $25.00.*

The introduction to the first edition of this book stated,

> When action is taken to deal with the medical and social aspects of a particular disability, not only is the lot of those who suffer from it improved, but added knowledge makes possible more effective preventive measures. There is no reason to doubt that this will also happen in mental deficiency.

The publication of this third edition, 16 years after the first, validates the accuracy of that prophecy and verifies the appropriateness of the title *The Changing Outlook*.

An attempt to secure a copy of the second edition was invariably met with, "Sorry, on loan, may we place your name on the waiting list?" I think that alone is testimony to the popularity and usefulness of this volume and makes it easy to predict that this third edition will meet with equal if not more acclaim.

The physical makeup of the book has shown growth and change—the pages are larger and have increased in number from 513 to 856. For some reason—design, style, print, or whatever—the third edition seems much easier to read than the first. The number of contributors has been increased, reflecting the growth of basic information and the interest and involvement of such ancillary disciplines as genetics. What seems noteworthy is the similarity in presentations from a more or less diverse group of contributors. The content hangs together as a unit and not as an uneven collection of essays, as is so often the case in books by many authors. Although there have been vast and even dramatic changes from the first to the third edition; such as the relegation of words like "idiot," "imbecile," and "moron" to obsolescence, the language remains terse, lucid, economical, and at times witty, which places this book above dull pedantry.

The list of contributors lends argument to those who would point a finger at psychiatry for having abandoned the field of mental retardation, an argument that has little validity today when one views the broad spectrum of disciplines that now comprise the area of mental retardation. The book encompasses areas of knowledge ranging from neonatology to parental psychology to governmental involvement and responsibility.

The book's approach to this very broad field is not based on the medical model. It is more directed toward mental deficiency as a "social concept with fluctuating thresholds of community tolerance." In the chapter on cerebral palsy, for example, the following statement is made:

> No attempt has been made to cover the strictly medical aspects of cerebral palsy or to consider the literature of the ancillary medical services such as physiotherapy, occupational therapy and speech therapy.

This kind of approach does not necessarily detract from the content; it emphasizes another approach that physicians might find informative and useful.

Along with the experiences, research results, and ideas of the respective authors there is a very extensive compilation of pertinent literature—I would guess there are no less than 1,500 references in the collected essays. The material is presented in a manner that gives the novice a broad but clear perspective of the field of mental retardation. Equally important, the more sophisticated reader is presented with conflicting arguments and hypotheses to stimulate further questions and thought. The beginner will note the important landmarks and hilltops as well as the clear plains and valleys. The researcher will find directions indicated toward unexplored areas as well as a description of the swamplands, crevasses, and wasteful whirlpools.

Perhaps human psychodynamics does not lend itself readily to categorization and statistical interpretation. For this reason the chapter on "The Effect of the Severely Subnormal on Their Families" was the least satisfactory for therapeutic purposes. There are quotations, references, statistics, and descriptions of socioeconomic factors, but there is

little if any mention of the human tragedy and emotional impact of mental retardation on the individuals or on intrafamilial relationships. Statistics and percentages are of no import to the affected individuals; I was disappointed at the paucity of depth psychology. References are made to papers on "shame and guilt of parents," but there is little discussion of what this means or how it might be of therapeutic service. No mention was made of the various manifestations of denial as the generally accepted most common defense mechanism employed by parents.

I strongly believe that this volume will be most helpful to a broad audience interested in the changing outlook in the field of retardation. Altogether, it is a handsome, helpful, encompassing volume that brings unity to and strengthens the structure of the body of knowledge concerned with mental retardation. Decidedly, it refines the boundaries of this emerging discipline.

JOSEPH MICHAELS, M.D.
New York, N.Y.

Thinking About Crime, *by James Q. Wilson. New York, N.Y., Basic Books, 1975, 221 pp., $10.00.*

This is an unusual book. It grasps the unwieldy subject of crime; it attacks some common attitudes toward crime among the public, the law-enforcement agencies, and the courts; it pricks the bubble of rehabilitative hopes; and then it enters the fog of "public policy." This book can best be described as a clearheaded critic's view of the amorphous and often befogged universe of crime from the vantage point of an expert on government rather than psychiatry or sociology (the author is Henry Lee Shattuck Professor of Government at Harvard University). It is remarkable in that no special school is advanced, no special theories invoked, and no panaceas offered—only a thorough analysis of the myths and practices surrounding crime in the United States.

Professor Wilson explores the increasing crime rates of the 1960s because that was the decade of the emergence of almost universal drug use. It also witnessed the emergence of what Wilson terms a "broad national force" of unknown nature that stimulated the reported rise in crime. The author's method is to study how the community, the police, politicians, criminologists, psychiatrists, and the courts look at and treat crime as well as their underlying attitudes toward crime.

Wilson zeroes in on the problem of crime causation (or, more accurately, the presupposition of causation). Many eminent sociologists have insisted that we first find the "cause of crime" and then develop a program of action from this discovery, whereas Wilson shows—and with acute perception—that there is no way to modify these causes, if and when they are found, in terms of public policy. As he puts it:

> Policy analysis, as opposed to causal analysis, begins with a different perspective. It asks . . . what is the condition one wants to bring into being, . . . what policy tools does a government . . . possess that might . . . produce at reasonable cost a desired alteration in the present condition.

He takes criminologists to task for being "long on theory and short on facts," agreeing at the same time that "explaining human behavior is a worthwhile endeavor." What Wilson objects to is the impossibility of applying "interesting facts" about crime to public policies regarding what is to be *done* about crime.

Other areas the author studied include the use of "random preventive police patrols and community service neighborhood policing teams" to augment the preventive force of police on the streets; the development of human relation skills among police; the complex problem of drug addiction; and the use of probation officers in reducing crime. The author reasons for rather than suggests or pleads for treatment of criminals on the basis of facts rather than myths. For example, he states that employment would reduce crime among young people *if* employment were more profitable than crime. In his words, "If criminal opportunities are profitable, many young persons will not take those legitimate jobs that exist," i.e., the risks of crime should be made more costly.

Some general conclusions arise from the maze of studies in the sociological and penological literature that Wilson cites. These are difficult to summarize. The main thrust of the author's essays (the book is made up of a series of essays separately published over some three years) is to criticize today's "pieties and pretensions." Wilson feels that not much can be done to change the rate of recidivism or juveniles' experimentation with petty crime or to increase the police apprehension rate. What can be done, he asserts, is to improve the judicial system—"reduce the arbitrary and socially irrational exercise of prosecutorial discretion . . . stop pretending judges know . . . how to provide 'individualized justice.' " In short, "Learn what works . . . and abandon . . . preconceptions about what *ought* to work."

The tone of this book is philosophical and ratiocinative. The author's criticisms and his materials are well chosen, but one misses a certain practical contact with offenders, in and out of institutions, in the writing. For example, in the thorny area of drug addiction, his statements that the pusher is a "myth" and that addicted youngsters do not turn to crime to support their habit are not consistent with my experience. Addiction, at least on the west coast, is a fixed, unassailable, unconquerable aspect of some segments of youthful life.

In spite of this and other criticisms that could be leveled against it, this book does state the myriad problems—social, ethical, and psychological—of crime in these United States. Professor Wilson's book is nothing if not "thinking about crime." It is recommended for all those who struggle with that morass.

WALTER BROMBERG, M.D.
Sacramento, Calif.

Borderline Conditions and Pathological Narcissism, *by Otto F. Kernberg, M.D. New York, N.Y., Jason Aronson, 1975, 354 pp., $15.00.*

Otto Kernberg's significant contributions to the understanding and treatment of patients with borderline personality organization and narcissistic personality are brought together in this new volume. It consists of 10 chapters: 7 were previously published, 1 was presented at a symposium in March 1974, and 2 are new. The previously published chapters include Kernberg's basic papers on borderline personality organization and narcissistic character and discuss diagnosis, prognosis, treatment principles, and countertransference issues. The new chapters present a critical review of the recent literature on differential diagnosis and

treatment, discuss the experience of emptiness, and clarify developmental aspects of narcissism.

The differences of opinion between Kernberg and Kohut about the nature of normal and pathological narcissism and their divergent views about the treatment of narcissistic disorders have been an exciting part of the literature during the past five years. Kernberg's views about narcissism and the formation of the grandiose self and his many other disagreements with Kohut are clearly delinated in this book.

Kernberg's work is solidly grounded in a Freudian psychoanalytic framework, which includes the contributions of such writers as Edith Jacobson and Heinz Hartmann. He also assimilates critical aspects of the work of some important British workers: Melanie Klein, Fairbairn, M. Little, P. Heimann, M. Khan, Guntrip, Rosenfeld, and Winnicott. Kernberg's synthesis leads to an object relations approach that makes use of their understanding of early object relations but maintains in theory and practice a careful developmental view of defenses, ego structure, and the self.

Kernberg delineates the developmental issues that distinguish psychotic, borderline, and narcissistic patients. He then describes the treatment implications that follow from these formulations. He reminds us constantly that object relations include relationships with outside objects as well as their intrapsychic representations.

The usefulness of this volume is obvious to anyone who is interested in studying the development of Kernberg's important work during the past 10 years. Because his papers require careful rereading, their publication in one volume assists the reader enormously. The 3 new chapters complement and expand the themes presented in the 7 other chapters and help give the book a sense of integration.

The publisher may have wanted to minimize the fact that this book contains some of Kernberg's previously published work rather than being a totally new book. One does not learn unambiguously until page 345 that 7 chapters were published before. I feel that Kernberg's papers on borderline and narcissistic patients benefit from publication in one volume and can be presented as such by any publisher with pride. This book will be used as one of the basic texts by clinicians and theoreticians who struggle to master an intellectual and emotional understanding of these difficult patients.

GERALD ADLER, M.D.
Boston, Mass.

Progress in Behavior Modification, Vol. 1, 1975, *edited by Michel Hersen, Richard M. Eisler, and Peter M. Miller. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1975, 330 pp., $19.50.*

The past two decades have seen a tremendous growth of interest in what has been termed "behavior modification," most evident in the ever-increasing number of publications in the field. In reviewing these publications, one is often struck by the widely varied approaches, simplistically presented methodologies, exaggerated claims, and widespread generalizations that characterize many. The need for review publications to distill the truly significant contributions from the superfluous and present the material in a way that is balanced and thought-provoking is obvious. The editors of this book have set themselves the task of not only providing a review but continuing to do so on an annual basis.

The book consists of eight review articles that deal with historical perspectives on the field, the behavioral study and treatment of depression, behavioral treatment of phobias and obsessive-compulsive disorders, behavior modification with delinquents, token economies, individual behavior therapy, use of drugs and behavior analysis, and ethical and legal issues in behavior modification. All of the authors deserve to be commended for their enthusiasm with regard to their topics and the completeness of their reviews. More important, the authors have confined their remarks to critical appraisal of past works without attempting to promote any particular approach over another. A constant theme appears to be a plea for some standardization of procedures whereby comparisons can be made with more ease.

Deserving of special note is the chapter on ethical and legal issues. The author presents a very literate and stimulating view of the pitfalls facing behavioral therapists that should be required reading for all practitioners. It is gratifying that this topic receives as much attention as some of the more specific techniques in light of the trend toward patients' rights and voluntary refusal of treatment.

This book is not intended for the neophyte but to serve as a source book for those involved with behavior modification. It succeeds extremely well in this effort, and all involved are to be congratulated. One can only hope that the level of excellence demonstrated here can be maintained on an annual basis, as the editors propose.

THOMAS C. BOND, M.D.
Belmont, Mass.

Psychotherapy and Psychoanalysis: Theory, Practice, Research, *by Robert S. Wallerstein. New York, N.Y., International Universities Press, 1975, 465 pp., $17.50.*

The major focus of the papers in this stimulating volume concern psychotherapy and psychoanalysis as therapeutic modes and as bodies of theory, with the goal of delineating the conceptual and technical relationship between the two. The stimulus for many of the wide-ranging examinations of the many clinical and theoretical problems involved derived from the author's experience with the Psychotherapy Research Project of the Menninger Foundation. Added to this source were the persistent strivings of the author toward a scientifically valid, formalized method of study of clinical phenomena, reflected in his concerns about scientific rigor in research, education, criticism, and theorizing.

Three or four of the discussions cover differential treatment planning and prognostication in psychotherapy, the assessment of change in psychotherapy (including an excellent critical review of the writings on the problem of values in mental health), and an exhaustive study of the current state of psychotherapy. The last-mentioned discussion ranges from a review of the theory of psychoanalytic psychotherapy as reflected in the contributions of such authors as Glover, Knight, Colby, Eissler, Alexander, Fromm-Reichmann, Tarachow, Dewald, Gill, and, of course, Freud to a survey of the nonanalytic models of psychotherapy of Rogers, Ford and Urban, London, Frank, Wolpe, and others.

Although Wallerstein subscribes to Benjamin's idea that a comprehensive and articulated theory of psychotherapy of all sorts should be developed, he recognizes the need to establish an adequate theory of therapy for the psychoanalytically oriented approaches and, indeed, for psychoanalysis itself. Accordingly, in several papers he discusses a variety of viewpoints on the goals of psychoanalysis, the role

of prediction in theory building in psychoanalysis, and the relationship of psychoanalysis to psychotherapy. His summary of the views of the major contributors to the debate on this last-named question is illuminating, and his statement of the issues in the debate is expressed in such incisive questions as the following:

> What are the dividing lines across the range of psychotherapies conceptualized within the framework of psychoanalytic theory? Is the major dividing line between the expressive-uncovering therapeutic modalities (of which psychoanalysis is one) and the suppressive-supportive therapeutic modalities? Which divisions are meaningful, in theory and in practice?

In the middle portion of the book Wallerstein advocates serious attention to the task of developing effective research methods for the scientific validation of psychoanalytic theories and procedures. He includes chapters on research on the psychoanalytic process, research training for psychotherapists, and psychoanalytic education. Noting the scientific limitations of the informal clinical case study approach, he issues a call for research into investigative methodologies that will meet the complex problems of hypothesis testing and verification. He reviews the obstacles that have traditionally been found in the path of such research and research training and effectively demonstrates the necessity and possibility of expansion of efforts in these fields.

The last portion of the book is highlighted by a paper coauthored by Neil J. Smelser titled "Psychoanalysis and Sociology." This chapter starts with an acknowledgment of the inability of psychoanalysis to apply its laboriously acquired knowledge of intrapsychic configurations to the understanding of manifest behavior, especially in the sphere of social interaction. Similarly, the problem of values (that is, relative costs and benefits, moral and other, which must be taken into account in attempting to understand and plan purposive action) cannot be encompassed by psychoanalysis alone. This chapter exemplifies the fruitfulness of the "process of *mutual* informing and interactive modification . . . referred to as the principle of 'complementary articulation' between disciplines" and supports the authors' claim that psychoanalysis is in need of further technical and theoretical advances.

Spanning almost 25 years of scholarly attention to crucial clinical, theoretical, and interdisciplinary problems, Wallerstein's papers profit from being assembled in this admirable collection in several ways. Perhaps the most impressive is the resulting effectiveness in conveying the brilliant, searching, multifaceted style of inquiry that marks the author's consistant thoughtfulness in the course of his many different studies. Another illuminating gain from the combined presentation of this book is the opportunity to see how clinical and theoretical insights jointly contribute to the clarification of the nature and basis of psychotherapy vis-à-vis psychoanalysis. Still another gain is the provision of a sophisticated view of the influence on psychoanalysis of changes and developments in society, as they affect both our understanding of the human mind and our view of and responsibilities toward society.

The combined impact of these and many other incisively conveyed perspectives is to present to the student of psychoanalytic psychology the challenge of participating in the complex process of continuing discovery and change. Wallerstein's call for systematic surveys and explorations demonstrates his belief in the necessity to extend our knowledge, "the better to understand the varieties of functionings and malfunctionings of the human mind and the differentiated techniques by which to influence it toward restored harmony." This book states the relevant issues with great clarity and gives eloquent testimony to Wallerstein's contribution to the extension of our understanding.

HERBERT F. WALDHORN, M.D.
Great Neck, N.Y.

Mental Retardation and Developmental Disabilities: An Annual Review, VII, *edited by Joseph Wortis, M.D. New York, N.Y., Brunner/Mazel, 1975, 356 pp., $15.00.*

This book, the seventh in a series of annual reviews, is of value for the professional trying to keep up with the more significant developments in the rapidly widening field of mental retardation and developmental disabilities.

The 14 chapters are all of high quality, but they vary in style and purpose. Each chapter is valuable in its own right, however. They cover widely diverse areas and will be of greatest value to the professional who has had experience in the field or who is interested in a specific area.

The book can be divided roughly in half. Six chapters are directed primarily toward the clinician or physician and represent comprehensive and informative summaries of a highly technical nature, including clinical aspects, developmental neurophysiology, metabolism, epilepsy, and congenital malformations. Eight chapters deal with program areas, including early stimulation, computers in education, vocational training, normalization, self-help, personnel training, and a description of services in Sweden. The medical and nonmedical chapters present a sharp contrast in objectivity, scope, and purpose. The medical chapters are composed of objective and current literature reviews, and the behavioral chapters tend to be scholarly (the one on Sweden), editorializing (on vocational training), descriptive (on early stimulation), explanatory (on computers), or evangelistic (on normalization). A short summary of the chapters follows:

Kirman's summary of clinical aspects is written for the general practitioner, the obstetrician, or the pediatrician and covers the medical role and decision making in prenatal diagnosis and postnatal screening.

The chapter on developmental neurophysiology integrates a review of the literature on animal studies by Rose with a review of human studies by Tanguay.

Snyderman reviews metabolic disorders; his summary is organized according to disorders associated with specific amino acids and carbohydrates. He also includes prenatal diagnosis and newborn screening in his discussion.

The chapter on epilepsy by Corbett, Harris, and Robinson explores the complex interrelationships of this condition with etiology, other handicaps, and psychosocial determinants of mental retardation.

The two chapters dealing with congenital malformations by Jones and by Warkany and Dignan are erudite; they will be of value primarily for the pediatrician and the obstetrician.

The chapter by Caldwell, Bradley, and Elardo on early stimulation deals with a survey of current programs rather than with the basic research or the theoretical issues involved.

Wiegerink and Simeonsson's review of public schools deals competently with issues currently affecting the mentally retarded, such as segregation versus mainstreaming, litigation and due process, and teacher training.

Stolurow's treatment of computers in education provides a basic description of computers and their use but devotes far too little space to the use or projected use of computer assisted instruction with the mentally retarded.

Gold's chapter, which deals with vocational training, presents an extreme position regarding the capabilities of the mentally retarded for work and the injustices perpetrated by underestimations of their capability.

Zipperlin's discussion of normalization is a humanitarian and sometimes emotional statement of a bias.

Katz's discussion of self-help refers to mutual assistance in groups sharing a common need.

The chapter on personnel training by Zarfas, Lovering, and Robbins surveys in-service training models for personnel working with the mentally retarded.

Grunewals' description of services and developments in Sweden holds high interest value.

GERALD R. CLARK, M.D.
MARVIN ROSEN, PH.D.
Elwyn, Pa.

Therapeutics in Neurology, *by Donald B. Calne, B.Sc., D.M. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1975, 317 pp., $32.00.*

This book is intended for physicians involved with the management of neurologic disorders. It might best be described as an up-to-date primer on the drugs available for neurologic disease. Surgical and physical therapies are only briefly mentioned.

The mechanism of drug action is considered in some detail, although the emphasis is placed on the practical aspects of patient treatment. Toxicity of drugs is discussed, and the optimal medical therapeutic approach is outlined.

This book should be in the consulting room of the physician who treats patients with neurologic disease.

DONALD W. MULDER, M.D.
Rochester, Minn.

Outgrowing Self-Deception, *by Gardner Murphy, with Morton Leeds. New York, N.Y., Basic Books, 1975, 167 pp., $8.95.*

This book is essentially a series of lessons teaching the development of perception as a skill. As mature adults we have extensive experience in perceptual learning, but that learning is clearly modifiable. Dr. Murphy argues, sometimes quite convincingly, that man is forever at work keeping vast areas of unwelcomed reality out of his view. Thus our perceptual learning has indeed been selective—most often to our disadvantage in terms of mature personality development.

Dr. Murphy's broad base of experience in a psychological climate allows him to draw extensively from mentors such as Bleuler and Murray. Few social scientists have had the broad base of Dr. Murphy's training and experience. The core emphasis of the book seems to be on the development of a feedback theory based on sound psychological principles from the life sciences.

The book is written with a broad readership in mind; it contains relatively little technical terminology and is certainly devoid of psychodynamic jargon. Dr. Murphy's professional maturity is clearly evident in his ability to summarize a considerable body of experimental evidence in a lucid, laymanlike manner. In fact, it borders on a do-it-yourself book for outgrowing self-deception. This manner of approach is graphically demonstrated in one chapter that specifically lists no fewer than 23 devices that may benefit man in coping with self-deception.

The introductory portion of the book lays a rather traditional-experimental groundwork for the subsequent self-improvement guidelines. The latter half of the book describes in detail some of the Freudian, Jungian, and Rogerian approaches to self-awareness and even includes some references to sensitivity training and biofeedback.

Dr. Murphy's style and enthusiasm will no doubt rub off on most readers and give them renewed motivation to pursue the possibility that there still are new psychophysiological horizons. The author indicates there are many ways to overcome the inner "dark spots" and that the best one is the one that works for you. I would submit that this book might well be the one that works for you. Read it.

WENDELL M. SWENSON, PH.D.
Rochester, Minn.

The Adolescent in Group and Family Therapy, *edited by Max Sugar, M.D. New York, N.Y., Brunner/Mazel, 1975, 282 pp., $13.50.*

An adolescent patient with emotional problems can be very difficult to reach in psychotherapy. This book is a collection of papers that explore several aspects of treating adolescent patients in group and family therapy. The book is divided into two parts: the first on group treatment and the second on family therapy. With an obvious emphasis on the psychodynamic forces in therapy, the book in remarkable detail explores the indications and contraindications for both modalities, the structure and setting of the group, group and family therapy dynamics, and a whole range of applications and adaptations of both modalities in treatment of the adolescent patient.

Using a finely balanced mixture of theory and practical application, this work explores a variety of adolescent problems, including descriptions of relevant psychodynamics of the patients involved in treatment. Practical aspects of therapy, including actual techniques of treatment, are included in every chapter. Interestingly, several authors describe not only their therapeutic techniques but also some of their efforts that have gone awry and their evaluation of the errors involved. Although the book emphasizes technique and theory of group and family therapy, many useful insights into the personal psychodynamics of both adolescents and adults are provided.

Dr. Max Sugar has done a remarkable job of editing a book on the relatively broad subjects of group and family therapies. Many edited books are difficult to read because of changes in style and emphasis between chapters. Dr. Sugar has avoided this pitfall with such great skill that it is not apparent to the reader when he has finished with one author and begun the next. Further, in only 282 pages can be found a wealth of information, presented in a concise and very readable format. Often I would find myself rereading passages, not because I did not understand what had been said but because there was even more to be gained.

Although the title might lead one to consider this book to

be of restricted interest, in fact anyone with an interest in group treatment, family therapy, adolescent patients, and even psychodynamic approaches to adult patients may find topics and concepts of interest to them in this work. I can heartily recommend *The Adolescent in Group and Family Therapy* as a book well worth reading.

E. L. LOSCHEN, M.D.
Springfield, Ill.

Self-Attitudes and Deviant Behavior, *by Howard B. Kaplan, Ph.D. Pacific Palisades, Calif., Goodyear Publishing Co., 1975, 181 pp., $8.95.*

The central thesis of this book is that deviant behavior results in improved self-attitudes. This thesis is based on the assumption that self-esteem is a dominant motive and is the normal outcome of the developmental process. Deviant behavior is viewed as a function of a person's membership in his various groups. The theory further states that there are three categories of deviant responses that function to alleviate subjective distress and thus satisfy the self-esteem motive. These functional categories are termed "avoidance of, attacks upon, and substitution for the normative environment."

The author tests his theory in chapters relating to "Dishonesty, Delinquency, and Criminality," "Drug Addiction and Drug Abuse," "Alcoholism and Alcohol Abuse," "Aggressive Behavior," "Suicidal Behavior," and "Mental Illness." In each chapter the relevant literature is examined to find out if the theory is supported. This task seems to be performed objectively, and a relationship is demonstrated in most classifications of deviant behavior.

As each form of deviant behavior is discussed the theory is reviewed, and examples from the literature are discussed and carefully examined. Copious notes are attached to each chapter; the author's style results in the reader's gaining easy understanding of the theoretical approach.

Of special interest to readers of this journal is the chapter on mental illness. Dr. Kaplan distinguishes between "predominantly expressive" and "predominantly instrumental" classes of mental illness in an attempt to relate these to traditional psychiatric diagnostic categories. He states,

> [The predominantly expressive category] here might include such traditionally defined neurotic disorders as anxiety neurosis, neurasthenic neurosis, and depressive neurosis; generally the psychophysiological disorders, and the transient situational disturbances; certain of the behavior disorders of childhood and adolescence such as overanxious reaction; and certain of the psychoses not attributed to physical conditions. . . . The predominantly instrumental category as it is defined here would include other of the traditionally defined neurotic disorders such as hysterical neurosis, phobic neurosis, and obsessive compulsive neurosis; generally the personality disorders; and several of the psychoses not attributed to physical conditions, particularly those disorders of thought and behavior characterized by reality distortion or other apparently self-protective behavior.

The theory does not attempt to explain the predominantly expressive disorders because they are not considered voluntary, motivationally relevant deviant patterns. This obviously leaves out a great many of the clinical issues psychiatrists work with and might be seen as a limitation in Kaplan's approach.

Another area in which more work could be done is in the exploration of the development of the predominance of the self-esteem motive. It would be interesting, for example, to compare this model of infantile development with the psychoanalytic model.

This carefully written book presents a workable approach to deviant behavior and is of interest to psychiatrists as well as to many other social scientists. As the theory is further developed, one hopes that material would emerge that would offer help to criminologists and present new ideas in rehabilitation and even primary prevention of mental disorders.

ALAN M. ELKINS, M.D.
Portland, Me.

The Child as a Work of Art, *by Bennett Olshaker, M.D. New York, N.Y., Reader's Digest Press (E.P. Dutton & Co., distributor), 1975, 198 pp., $7.95.*

This is a book about the difficult art of parenthood. Perhaps even more compelling, it is a book that shows respect for children as unique human creatures who deserve far more than what today's society is willing to commit to its children. The standards that Dr. Olshaker applies to child rearing are very familiar, but they have been discarded by too many modern parents.

The topics discussed in various chapters include self-esteem and mutual respect, the development of independence, discipline, handling anger, and success and failure. A particularly well done chapter deals with the question of who should care for our children. In this chapter the author confronts the difficult subject of day care for children. He concludes,

> Hopefully, parents will remain the principal care takers of their own children whenever possible. This brings me again to what I feel is a very important point—more people should decide not to have children rather than to neglect them once they are born.

In summary, I found this to be a charming book, full of wisdom and truths that many modern parents have lost sight of. I would recommend it wholeheartedly for professionals as well as for young people considering the serious responsibilities of parenthood.

JOHN C. DUFFY, M.D.
Governors Island, N.Y.

This Month's Reviewers

HOWARD P. ROME, M.D., is Senior Consultant, Section on Psychiatry, Mayo Clinic, Rochester, Minn.

DANA L. FARNSWORTH, M.D., is Henry K. Oliver Professor of Hygiene, Emeritus, Harvard University, Cambridge, Mass.

JOHN C. NEMIAH, M.D., is Psychiatrist-in-Chief, Beth Israel Hospital, and Professor of Psychiatry, Harvard Medical School, Boston, Mass.

JOHN M. WHITELEY, M.A.ED., ED.D., is Dean of Students and Associate Professor of Social Ecology, University of California, Irvine, Calif.

JACK C. WESTMAN, M.D., is Professor of Psychiatry, University of Wisconsin Medical School, Madison, Wis.

MERRILL T. EATON, M.D., is Professor and Chairman, Department of Psychiatry, University of Nebraska College of Medicine, and Director, Nebraska Psychiatric Institute, Omaha, Neb.

JOSEPH MICHAELS, M.D., is Medical Director, Shield Institute for Retarded Children and Developmental Evaluation Center, New York, N.Y.

WALTER BROMBERG, M.D., is Adjunct Professor of Legal Medicine, McGeorge School of Law, University of the Pacific, Sacramento, Calif.

GERALD ADLER, M.D., is Professor of Psychiatry and Director of Training in Adult Psychiatry, Tufts-New England Medical Center, Boston, Mass.

THOMAS C. BOND, M.D., is Assistant Psychiatrist, McLean Hospital, Belmont, Mass., and Instructor in Psychiatry, Harvard Medical School, Boston, Mass.

HERBERT F. WALDHORN, M.D., is on the faculty of the New York Psychoanalytic Institute, New York, N.Y.

GERALD R. CLARK, M.D., is President, Elwyn Institute, Elwyn, Pa.

MARVIN ROSEN, PH.D., is Director of Psychology, Elwyn Institute, Elwyn, Pa.

DONALD W. MULDER, M.D., is Professor of Neurology, Mayo Medical School, and Consultant, Department of Neurology, Mayo Clinic, Rochester, Minn.

WENDELL M. SWENSON, PH.D., is Professor and Head of the Section on Psychology, Mayo Clinic, Rochester, Minn.

E. L. LOSCHEN, M.D., is Assistant Professor, Department of Psychiatry, Southern Illinois University School of Medicine, Springfield, Ill.

ALAN M. ELKINS, M.D., is Chief of Psychiatry, Maine Medical Center, Portland, Me.

JOHN C. DUFFY, M.D., is Captain, U.S. Public Health Service, and Chief, Psychiatric Branch, U.S. Coast Guard Support Center, Governors Island, N.Y.

Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

The Behavioral Treatment of Sexual Problems, Vol. 2: Intensive Therapy, *by Jack S. Annon, Ph.D. Honolulu, Hawaii, Enabling Systems, 1975, 576 pp., $13.50 (paper).*

Gaze and Mutual Gaze, *by Michael Argyle and Mark Cook. New York, N.Y., Cambridge University Press, 1976, 201 pp., $18.50.*

For Yourself: The Fulfillment of Female Sexuality, *by Lonnie Garfield Barbach. New York, N.Y., Anchor Books (Anchor Press/Doubleday), 1976, 211 pp., $3.95 (paper).*

The Horrors of the Half-Known Life: Male Attitudes Toward Women and Sexuality in Nineteenth-Century America, *by G.J. Barker-Benfield. New York, N.Y., Harper & Row, 1976, 338 pp., $15.00.*

Decisions Decisions: Game Theory and You, *by Robert Bell and John Coplans. New York, N.Y., W.W. Norton & Co., 1976, 160 pp., $7.95.*

A Primer on School Mental Health Consultation, *by Morton I. Berkowitz, M.D. Springfield, Ill., Charles C Thomas, 1975, 110 pp., $10.00.*

Neuropsychopharmacology. Proceedings of the IX Congress of the Collegium Internationale Neuropsychopharmacologicum, Paris, 7–12 July, 1974, *edited by J.R. Boissier, H. Hippius, and P. Pichot. New York, N.Y., American Elsevier Publishing Co., 1975, 1,022 pp., $108.50.*

Preschool IQ: Prenatal and Early Developmental Correlates, *by Sarah H. Broman, Paul L. Nichols, and Wallace A. Kennedy. Hillsdale, N.J., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1975, 316 pp., no price listed.*

The Gifted Child, *by Cyril Burt. New York, N.Y., Halsted Press (John Wiley & Sons), 1975, 209 pp., $14.75.*

Annual Progress in Child Psychiatry and Child Development 1975, *edited by Stella Chess, M.D., and Alexander Thomas, M.D. New York, N.Y., Brunner/Mazel, 1975, 541 pp., $17.50.*

My Psychiatrist Says—Let Me Make This Perfectly Clear . . . , *by Gertrude Cooper, New York, N.Y., William-Frederick Press, 1975, 119 pp., $4.00.*

The Watcher and the Watched, *by Bruno M. Cormier, M.D. Plattsburgh, N.Y., Tundra Books (New York, N.Y., Charles Scribner's Sons, distributor), 1976, 330 pp., $10.00.*

The Sanctity of Social Life: Physicians' Treatment of Critically Ill Patients, *by Diana Crane. New York, N.Y., Russell Sage Foundation (Basic Books, distributor), 1975, 277 pp., $13.50.*

Season of Birth: A Study of Schizophrenia and Other Mental Disorders, *by Per Dalén. New York, N.Y., American Elsevier Publishing Co., 1975, 152 pp., $13.50 (paper).*

Socialization for Achievement: Essays on the Cultural Psychology of the Japanese, *by George A. De Vos, with contributions by Hiroshi Wagatsuma, William Caudill, and Keiichi Mizushima. Berkeley, Calif., University of California Press, 1976, 582 pp., $8.95 (paper).*

The Relation Between Physical and Mental Illness: The Physical Status of Psychiatric Patients at a Multiphasic Screening Survey, *by Michael Robin Eastwood. Buffalo, N.Y., University of Toronto Press, 1975, 115 pp., $10.00.*

Des idées de Jackson à un modèle organo-dynamique en psychiatrie, *by Henri Ey. Toulouse, France, Edouard Privat, 1975, 301 pp., 51,40 French francs (paper).*

The Assessment of Early Child Development, *by Dorothy Flapan and Peter B. Neubauer. New York, N.Y., Jason Aronson, 1976, 145 pp., $10.00.*

The Unconscious God: Psychotherapy and Theology, *by Viktor E. Frankl. New York, N.Y., Simon & Schuster, 1975, 159 pp., $6.95.*

Cocaine Papers, *by Sigmund Freud; edited by Robert Byck, M.D. New York, N.Y., Stonehill Publishing Co., 1974, 402 pp., $14.95.*

Psychiatry and the Human Condition, *by Iago Galdston, M.D. New York, N.Y., Brunner/Mazel, 1976, 440 pp., $20.00.*

Operating on the Mind: The Psychosurgery Conflict, *edited by Willard M. Gaylin, Joel S. Meister, and Robert C. Neville. New York, N.Y., Basic Books, 1976, 209 pp., $11.95.*

Genesis and Treatment of Psychologic Disorders in the Elderly. Aging, Vol. 2, *edited by Samuel Gershon, M.D., and Allen Raskin, Ph.D. New York, N.Y., Raven Press, 1975, 272 pp., $19.95.*

The Hazards of Being Male: Surviving the Myth of Masculine

Privilege, *by Herb Goldberg, Ph.D. Plainview, N.Y., Nash Publishing, 1976, 198 pp., $8.95.*

The Effect of the Method of Payment on Mental Health Care Practice. Report 95, *by the Group for the Advancement of Psychiatry Committee on Governmental Agencies. New York, N.Y., GAP, 1975, 130 pp., $4.00 (paper).*

The Psychiatrist and Public Welfare Agencies. Report 94, *by the Group for the Advancement of Psychiatry Committee on Psychiatry and the Community. New York, N.Y., GAP, 1975, 49 pp., $2.50 (paper).*

Handbook of Evaluation Research, Vol. 2, *edited by Marcia Guttentag and Elmer L. Struening. Beverly Hills, Calif., SAGE Publications, 1975, 716 pp., no price listed.*

Criminality and Psychiatric Disorders, *by Samuel B. Guze, M.D. New York, N.Y., Oxford University Press, 1976, 177 pp., $9.50.*

Beyond Culture, *by Edward T. Hall. Garden City, N.Y., Anchor Press/Doubleday, 1976, 244 pp., $7.95.*

Textbook of Disturbances of Mental Life, or, Disturbances of the Soul and Their Treatment, Vol. I: Theory; Vol. II: Practice, *by Johann Christian Heinroth. Baltimore, Md., Johns Hopkins University Press, 1975, 455 pp., $17.00.*

Through an Eastern Window, *by Jack Huber. New York, N.Y., St. Martin's Press, 1976, 121 pp., $2.95 (paper).*

Transcultural Aspects of Psychiatric Art. Psychiatry and Art, Vol. 4, *edited by Irene Jakab. New York, N.Y., S. Karger, 1975, 293 pp., $51.75.*

Essays in Freudian Psychoanalysis, *by Samuel Kahn. New York, N.Y., Philosophical Library, 1976, 159 pp., $10.00.*

Psychological Deprivation in Childhood, *by J. Langmeier and Z. Matějček; edited by G.L. Mangan. New York, N.Y., Halsted Press (John Wiley & Sons), 1975, 481 pp., $19.75.*

Knowledge of Illness in a Sepik Society: A Study of the Gnau, New Guinea. London School of Economics Monographs on Social Anthropology 52, *by Gilbert Lewis. London, England, Athlone Press, University of London (Atlantic Highlands, N.J., Humanities Press, distributor), 1975, 370 pp. $33.75.*

Help Yourself to Happiness Through Rational Self-Counseling, *by Maxie C. Maultsby, Jr., M.D. Boston, Mass., Esplanade Books, Marlborough House (Herman Publishing), 1975, 251 pp., $5.95 (paper).*

Power: The Inner Experience, *by David C. McClelland. New York, N.Y., Irvington Publishers (Halsted Press, John Wiley & Sons, distributor), 1975, 421 pp., $15.95.*

Reproductive Physiology of Mammals and Birds: The Comparative Physiology of Domestic and Laboratory Animals and Man, 3rd ed., *by A.V. Nalbandov. San Francisco, Calif., W.H. Freeman and Co., 1976, 323 pp., $14.50.*

Yoga and Psychotherapy: The Evolution of Consciousness, *by Swani Rama, Rudolph Ballentine, M.D., and Swani Ajaya (Allan Weinstock, Ph.D.). Glenview, Ill., Himalayan Institute, 1976, 321 pp., $11.95.*

Coverage and Utilization of Care for Mental Conditions Under Health Insurance—Various Studies, 1973–1974, *by Louis S. Reed, Ph.D. Washington, D.C., American Psychiatric Association, 1975, 80 pp., $4.00 (paper).*

Early Writings, Vol. One, *by Wilhelm Reich; translated by Philip Schmitz. New York, N.Y., Farrar, Straus & Giroux, 1975, 332 pp., $10.00.*

Morita Psychotherapy, *by David K. Reynolds. Berkeley, Calif., University of California Press, 1976, 242 pp., $10.95.*

Time of Transition: The Growth of Families Headed by Women, *by Heather L. Ross and Isabel V. Sawhill, with the assistance of Anita R. MacIntosh. Washington, D.C., Urban Institute, 1975, 216 pp., $10.00.*

Explaining the Brain, *by W. Ritchie Russell, with A.J. Dewar. New York; N.Y., Oxford University Press, 1975, 153 pp., $4.00 (paper).*

Self-Assessment of Current Knowledge in Child Psychiatry, *by G. Pirooz Sholevar, M.D. Flushing, N.Y., Medical Examination Publishing Co., 1975, 280 pp., $12.00 (spiral-bound).*

Death Inside Out: The Hastings Center Report, *edited by Peter Steinfels and Robert M. Veatch. New York, N.Y., Harper & Row, 1975, 149 pp., $7.95.*

Should Trees Have Standing? Toward Legal Rights for Natural Objects, *by Christopher D. Stone. Los Altos, Calif., William Kaufmann, 1974, 94 pp., $3.50 (paper).*

Handbook of Evaluation Research, Vol. 1, *edited by Elmer L. Struening and Marcia Guttentag. Beverly Hills, Calif., SAGE Publications, 1975, 676 pp., no price listed.*

Predictability in Psychopharmacology: Preclinical and Clinical Correlations, *edited by Abraham Sudilovsky, M.D., Samuel Gershon, M.D., and Bernard Beer, Ph.D. New York, N.Y., Raven Press, 1975, 283 pp., $22.50.*

Marijuana and Health Hazards: Methodological Issues in Current Research, *edited by Jared R. Tinklenberg. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1975, 171 pp., $8.50.*

Prevention: A Psychophysiological and Psychodynamic Study, *by Clara Torda, Ph.D., M.D. New York, N.Y., Walters, 1975, 324 pp., no price listed (paper).*

Art Therapy in Theory and Practice, *edited by Elinor Ulman and Penny Dachinger. New York, N.Y., Schocken Books, 1976, 402 pp., $15.00.*

To Each His Farthest Star: University of Rochester Medical Center 1925–1975, *edited by the University of Rochester Medical Center Publications and Publicity Committee. Rochester, N.Y., University of Rochester Medical Center, 1975, 539 pp., no price listed (paper).*

Research in Psychophysiology, *edited by Peter H. Venables and Margaret J. Christie. New York, N.Y., John Wiley & Sons, 1975, 437 pp., $32.00.*

OFFICIAL ACTIONS

1976 Annual Meeting: Registration and Related Information

The 129th annual meeting of the American Psychiatric Association will be held in Miami, Fla., May 10–14, 1976. The information here is what was available as of mid-January 1976. The official program book containing the scientific, business, and social program will be distributed in Miami at the time of registration. (The preliminary scientific program was printed in the February 20 issue of Psychiatric News.)

REGISTRATION

All registration will take place in the North Hall, Miami Beach Convention Center. The registration desk will be open:

Sunday, May 9—1:00 p.m. to 5:00 p.m.

Monday through Thursday, May 10–13—8:00 a.m. to 5:00 p.m.

Friday, May 14—8:00 a.m. to 10 a.m.

A registration fee of $15 per day, or $60 for all five days, is required from all nonmembers with the exception of 1) invited nonmember program participants who are presenting papers or who are session officers, and their immediate families, 2) discussants, 3) participants in morning and evening panels and their immediate families, and 4) the immediate families of APA members. Associate members, members-in-training, and their immediate families are also exempt from registration fees. The fee covers admission to the sessions and includes a badge and a copy of the official program. Badges are required for all sessions (including the opening session) and for the exhibit area. However, a badge does not cover admission to the business session of the Association, which is open only to members.

A $20 registration fee is required of visitors living outside the United States or Canada and of military personnel on active duty.

A $10 registration fee is required of the spouses and dependents of all nonmember registrants (excluding invited nonmember program participants).

A $15 registration fee is required of medical students, interns, residents, chaplains, nursing students, and students in the mental health professions and their spouses. Students must present a letter from their school or training facility instructor certifying their status.

The *Scientific Proceedings in Summary Form* for the 1976 annual meeting will be available throughout the week in the registration area of the Convention Center.

KEY LOCATIONS

All APA staff offices will be located in the Convention Center:

Staff Office	Room 154
Convention Office	North Lobby
Journal Office	Room 157
Press Offices	Rooms 150, 151
Local Arrangements	Room 153
Registration and Membership Information Center	North Hall
Information and Message Center	North Lobby
Spouse Hospitality Center	North Lobby
First Aid	North Lobby

INFORMATION AND MESSAGE CENTER

This center will be located in the North Lobby and will be manned during registration hours. Self-service message racks will be available for pick-up, deposit, and exchange of messages at all times, even when the message center is closed. Extremely urgent messages will receive the personal attention of APA staff members. All registrants are requested to check the message center periodically.

JOURNAL OFFICE

The office of the *American Journal of Psychiatry* will be in Room 157. The office will be open from 9:00 a.m. to 5:00 p.m., Monday through Thursday, and on Friday until 12:00 noon. Staff will be available to discuss any questions that authors who are presenting papers at the meeting may have. They particularly welcome the opportunity to confer with authors about tables and figures to be included in the papers.

Authors of numbered papers are reminded that all such papers are the property of the *Journal*. Permission must be secured from the Editor, Francis J. Braceland, M.D., before a paper may be offered for publication elsewhere. New research papers and papers presented at morning and evening panels do not automatically become the property of the *Journal*; however, authors may submit them for publication if they wish.

It is the policy of the *Journal* to publish only original material that has not been published elsewhere in any form and that is not being considered for publication elsewhere. "Publication" includes recording on videotape or audiotape and appearance in printed form.

WRITERS' WORKSHOP CANCELLED

The workshop on writing for scientific journals, which was scheduled to be held on May 9, has been cancelled.

CONVOCATION

The convocation will take place Wednesday, May 11, at 7:30 p.m. in the Fontainebleau Hotel, and will be followed by a special lecture.

FORUM ROOM

Rooms in the Convention Center are available for discussion of special issues during the course of the meeting. All groups who wish to schedule programs must apply for the use of these rooms by contacting the Convention Manager in the Convention Office, North Lobby. Groups who have secured use of a room and who wish to have a placard prepared for display in the registration area should contact the Convention Manager. Forums scheduled at the time of printing are listed in the official program under APA Activities.

SPOUSE HOSPITALITY CENTER

The Local Arrangements Committee will host a Spouse Hospitality Lounge in the Lobby of the Convention Center. Information on all leisure-time activities will be available Sunday through Thursday in this area. This area will also serve as the assembly point for all tours and special events.

DAY-CARE CENTER

APA has arranged with Ms. Maude Martin, Director of the Children's Corner Nursery School, 7118 Byron Ave., Miami Beach, Fla. 33141, to provide day care for infants and children 12 years and younger. The day-care center will operate from 7:30 a.m. to 5:30 p.m., Monday through Friday, and will charge $4.50 for four hours or less and $8.50 for up to nine hours. Advance reservations are recommended; coupons for this purpose can be found in various issues of *Psychiatric News*.

PROFESSIONAL TOURS

Information on tours to facilities of psychiatric interest in the Miami area may be obtained from members of the Local Arrangements Committee in the Spouse Hospitality Center.

PLACEMENT SERVICE

There will be a register available in the Membership Information Center for prospective employers and for those seeking positions.

MORNING AND EVENING PANELS

Morning panels will be held on Tuesday, Wednesday, and Thursday from 7:00 a.m. to 8:45 a.m. Breakfast may be obtained in regular hotel facilities. Evening panels will be held on Monday and Thursday at 8:00 p.m. All panels will be held at either the Fontainebleau, Eden Roc, Deauville, or Carillon hotels.

VIDEOTAPE SPECIAL SESSIONS

Videotape special sessions will be shown on wide-screen closed-circuit color TV in Room 101, Convention Center, from 9:00 a.m. to 12:00 noon and 2:00 p.m. to 5:00 p.m., Monday through Thursday, and from 9:00 a.m. to 12:00 noon on Friday.

FILMS

Films will be shown in Room 102, Convention Center, from 9:00 a.m. to 12:00 noon and 2:00 p.m. to 5:00 p.m., Monday through Thursday, and from 9:00 a.m. to 12:00 noon on Friday. Additional special features will also be shown.

SPECIAL LECTURES

APA will sponsor nine special lectures this year. Among these will be the Solomon Carter Fuller Lecture (Tuesday, from 10:00 a.m. to 12 noon), the Adolf Meyer Lecture (Tuesday, 1:00 p.m.), the Simon Bolivar Lecture (Wednesday, from 10:00 a.m. to 12:00 noon), and the Benjamin Rush Lecture (Wednesday, from 2:00 p.m. to 3:30 p.m.). In addition, Dr. Francis J. Braceland has been invited to give the Bicentennial Lecture on the theme of the convention, The Continuing Revolution: Benjamin Rush and His Successors (Tuesday, 10:00 a.m.).

SESSIONS ON NEW RESEARCH

These sessions will be held in the Miami Convention Center on Monday, from 2:00 p.m. to 5:00 p.m., Thursday, from 2:00 p.m. to 8:00 p.m., and Friday, from 9:00 a.m. to 12:00 noon. Program leaflets containing all details will be available at the registration desk.

CONTINUING EDUCATION CREDIT

In addition to other formal presentations, AMA Category I accredited courses will be offered for the first time. One hour of credit may be claimed for each hour of participation. Persons who want to participate in a course should consider possible conflicts with other annual meeting activities, including scientific papers. Check the February 20 issue of *Psychiatric News* for a complete program schedule of the annual meeting.

Those interested in participating in the courses MUST preregister, enclosing a $15 fee for each course. Registration closes April 15, 1976. Preregistration coupons and complete course descriptions can be found in various January-February issues of *Psychiatric News*.

ARTS AND CULTURE

A special production of the Broadway play "Equus" will take the place of the Annual Banquet. Performances will be at the Coconut Grove Theater on Sunday, May 9, at 8:00 p.m., and Tuesday, May 11, at 2:00 and 8:00 p.m. Complimentary general admission tickets will be available for APA members (two per member) in the registration area on a first-come, first-served basis. Shuttle bus service will be provided to and from each performance; details will be printed on the back of each ticket. "Question the Expert," a panel discussion with the author of "Equus," will be held on Wednesday, May 12, from 10:00 a.m. to 12 noon, in Room 201 of the Convention Center.

SHUTTLE BUS SERVICE

Bus service will be available between the Convention Center and nearby hotels. Route maps, hours of operation, and fare information will be available at the Information and Message Center.

EXHIBITS

Technical and scientific exhibits will be on display in North Hall, Convention Center, Monday through Thursday, 8:00 a.m. to 5:00 p.m.

SPECIAL BRUNCH

A champagne brunch with a special guest speaker will be held in honor of the spouses of APA officers on Tuesday, May 11, at 10:30 a.m. in the Fontaine Room of the Fontainebleau Hotel. Tickets for the brunch are $13.50 per person and may be purchased in the registration area. Buses will depart at 10:00 a.m. from the North Lobby.

Position Statement on Organizational Structure of State Mental Health Programs

This statement was approved by the Board of Trustees of the American Psychiatric Association at its December 5-6, 1975, meeting upon recommendation of the Council on Mental Health Services. It was prepared by the Council on Mental Health Services.[1]

A STATE MENTAL health program can function most effectively when there is a separate and easily identified organizational component such as a department of mental health with full responsibility for the program.

The director of the mental health program should be a qualified psychiatrist with an appropriate administrative background, and he should have a qualified business administrator as one of his principal assistants. The director requires authority commensurate with the responsibility and accountability of the position.

Direct access to the Governor and to the legislature is necessary for the director to adequately perform the duties of his position.

The organizational placement of state services for the mentally retarded as a separate program or as a program combined with mental health services is most appropriately determined by considerations of the size and complexity of the program for the mentally retarded.

[1]The Council on Mental Health Services included: James C. Johnson, M.D., chairperson, Mildred Mitchell-Bateman, vice-chairperson, Israel Zwerling, M.D., Bruce W. Alspach, M.D., J.M. Stubblebine, M.D., Aubrey Dent, M.D., observer-consultant, and William B. Spriegel, M.D., Assembly of District Branches liaison.

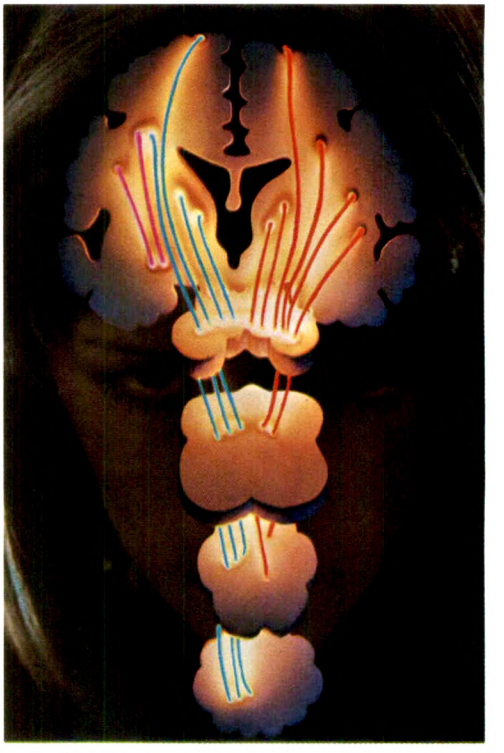
Treating the chemocircuitry of depression



Depression as an organic disease

At one time, depression was thought to be predominantly a behavioristic, nonmetabolic abnormality. But recent work on CNS catecholamine metabolism strongly suggests that biochemical factors are involved.

Theory of biogenic etiology



□ In individual nerve systems originating in the brainstem, impulses are relayed within each network by chemically distinct compounds—the biogenic amines.[1]

□ These compounds "... serve as neurotransmitters in brain sites that probably modulate feeling states, and other mental functions, that are disturbed in psychiatric disease."[2]

□ Amine systems involved in affective disorders[3] are:
norepinephrine (catecholamine)
dopamine (catecholamine)
serotonin (indoleamine)

There is a school of thought that associates catecholamines and affective disorders. According to this hypothesis, "... some, if not all, depressions are associated with an absolute or relative deficiency of catecholamines, particularly norepinephrine, at functionally important adrenergic receptor sites in the brain."[4]

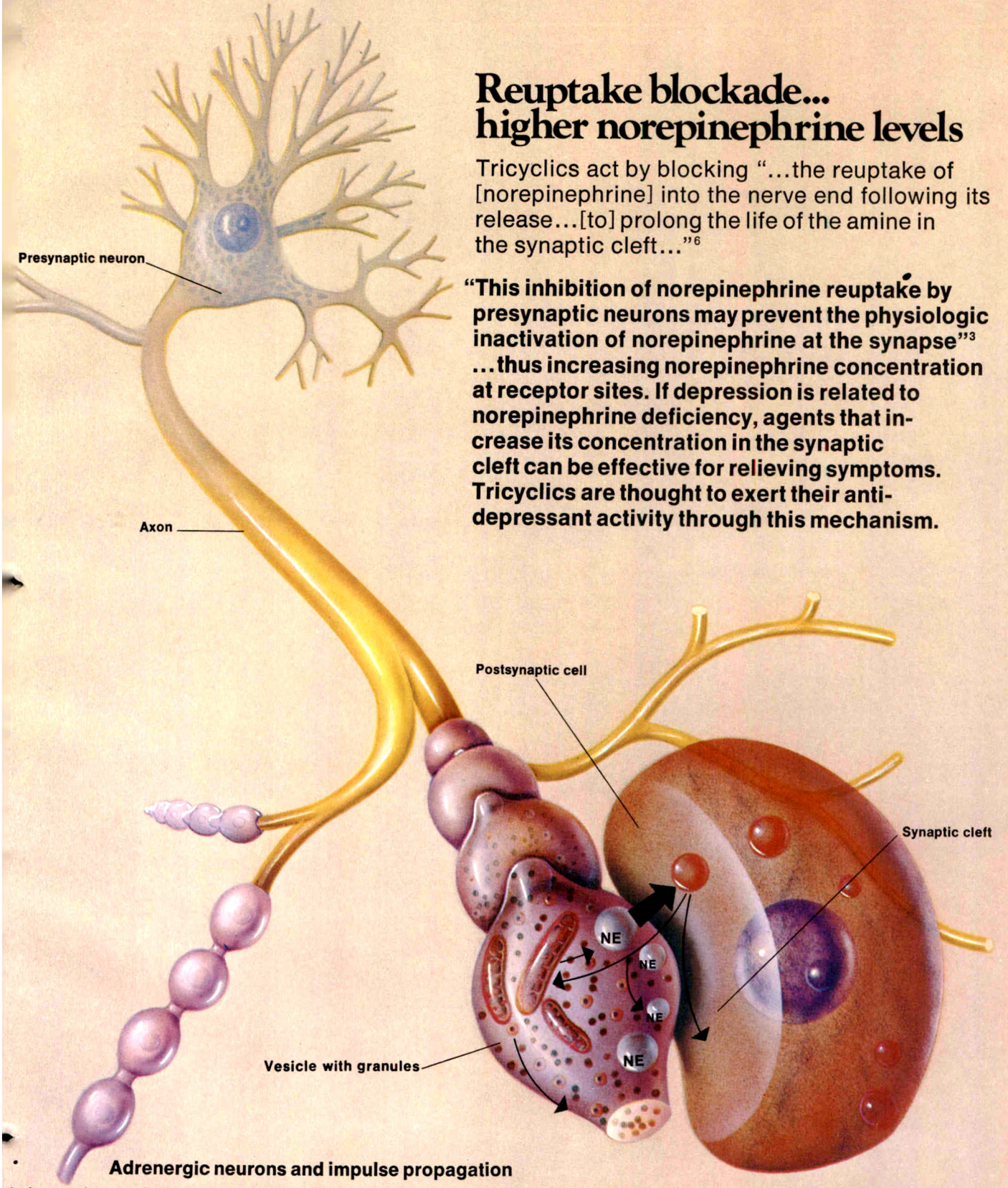
"The possibility that depressed patients may have a functional deficiency of brain norepinephrine has gained considerable currency over the past decade."[5]

.and the efficacy of tricyclics

Reuptake blockade... higher norepinephrine levels

Tricyclics act by blocking "...the reuptake of [norepinephrine] into the nerve end following its release...[to] prolong the life of the amine in the synaptic cleft..."[6]

"This inhibition of norepinephrine reuptake by presynaptic neurons may prevent the physiologic inactivation of norepinephrine at the synapse"[3] ...thus increasing norepinephrine concentration at receptor sites. If depression is related to norepinephrine deficiency, agents that increase its concentration in the synaptic cleft can be effective for relieving symptoms. Tricyclics are thought to exert their antidepressant activity through this mechanism.



Adrenergic neurons and impulse propagation

Information coded as electric impulses arrives synapse via axons of presynaptic neurons.

Impulses cause "release" of norepinephrine ored in vesicles adjacent to the presynaptic embrane.

Neurotransmitter migrates across the synaptic

it either triggers a new impulse or inhibits activity in the postsynaptic cell.

□ Because of the complex nature of brain activity, this action must be terminated rapidly and the cleft primarily cleared through reuptake of the transmitter into the presynaptic cell.

Tricyclics...the right choice in depression

Circuit functions and pharmacologic effects

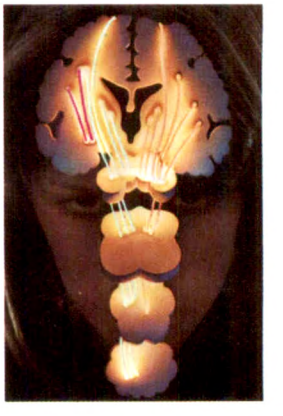
Functioning of the biogenic amine systems, and how they are affected by therapeutic agents, underscore the value of tricyclics in depression.

The serotonin system: sedation/sleep



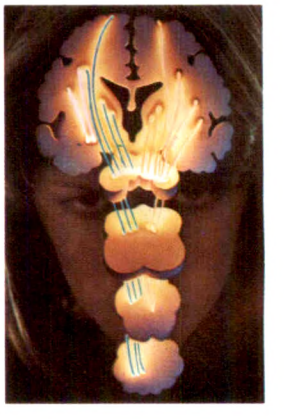
☐ Thought to regulate aspects of the sleep-wakefulness cycle and may be involved in REM sleep or dreaming.[2]
☐ Deficiency in turnover can produce symptomatology also present in depression.
☐ Some tricyclics, however, principally block the reuptake inactivation of serotonin.[6]
☐ **Activation of this system may therefore produce unwanted sedation.**

The dopamine system: motor activity



☐ Associated with normal posture and regulation of motor activity.
☐ Disturbed dopamine levels linked to symptoms of Parkinson's disease.
☐ "A possible relationship between decreased baseline... levels [of a dopamine metabolite] and decreases in motor activity in depressed patients has been suggested by some investigators..."[7]
☐ **Agents that influence this system can thus affect motor activity[6] and produce unwanted side effects.**

The norepinephrine system: mood



☐ Plays a major role in affective states.
☐ Deficiencies of norepinephrine linked to depression.
☐ Norepinephrine levels may be raised by psychomotor stimulants which cause release and block reuptake... by MAO inhibitors which block intraneuronal metabolism ...and by tricyclics through reuptake blockade.[1]
☐ Of these agents, the "most useful are the tricyclics."[6] They are preferred for their greater safety[8] and lower incidence of side effects.[9]
☐ **But tricyclics vary in their degree of interaction with other circuits—not only the catecholamines, but serotonin and acetylcholine as well.[10]**

Pertofrane®...the right choice in tricyclics
(desipramine hydrochloride NF)

Consider the chemistry

'...qualitative and quantitative changes in the biochemical, pharmacological and clinical spectrum...[were produced by] the elimination of one of the *N*-methyl groups in the terminal amino group of imipramine to yield desipramine."[11]

"...monomethylated forms of tricyclic antidepressants...are generally much more potent inhibitors of catecholamine re-uptake than dimethylated forms ...[whereas] dimethylated tricyclic antidepressants are more potent anticholinergics than the monomethylated forms."[12]

Dimethylated drugs are more potent serotonin blockers than monomethylated analogs. "Imipramine slows the turnover rate of [serotonin], an effect not shared by desipramine..."[13]

'...compared to imipramine, [desipramine] is more 'procatechol' than 'proindole.'"[6]

...and the clinical implications

Rapid onset of action

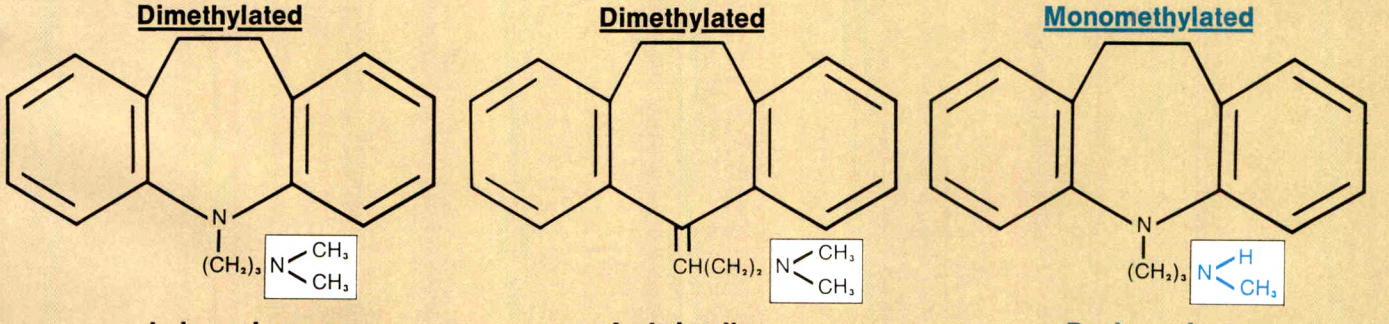
Pertofrane has been found in some studies[14,16,19] to have a more rapid onset of action than imipramine. This relatively rapid onset of action may bring about prompt therapeutic response.

In a recent double-blind study comparing desipramine and amitriptyline, "the activity of desipramine becomes manifest rapidly and its beneficial effects further improve over time."[15]

Less sedative effect

Desipramine HCl shows good to excellent antidepressant effect yet is only mid-range in sedation.[6]

In a recent double-blind study comparing desipramine and amitriptyline in outpatients with depressive neurosis, while desipramine was equal or superior to amitriptyline in improving anxiety/depression and sleep disturbance factors, amitriptyline produced sedation in significantly more patients than did desipramine.[15]



Imipramine **Amitriptyline** **Desipramine**

Selection of the right tricyclic for a particular patient can be based on structural activity relationships. And the profile of activity for a particular agent frequently depends on whether it is dimethylated or monomethylated.

Please see back cover for full prescribing information.

When activity is important



the neurochemistry's right for Pertofrane® (desipramine hydrochloride NF)

Proven clinical efficacy

☐ "...seems ideally suited to use in an active ambulatory patient population...[because of] its significant improvement of the depressed mood, work and activities..."[15]

☐ Effectiveness well documented in over a decade of clinical use.[9,16–18]

Prompt onset of action

☐ Relief may begin in 2 to 5 days—but full therapeutic effect is seldom seen before 2 weeks.

☐ Rapid onset of action[11,14,15,16,19] is particularly useful in patients who must continue to function actively during the course of therapy.

Mid-range sedation[6]

☐ Relieves associated mild anxiety/agitation, yet is only mid-range in sedative effect.

☐ "There is no problem of drowsiness"[16]—especially important when normal levels of daily activity must be maintained during treatment.

Compatible with tranquilizers/sedatives

☐ Where necessary, may be used with antianxiety or hypnotic agents.[16]

☐ Permits individualization of therapy when mild anxiety or agitation accompanies depression.

Provides dosage flexibility

☐ The therapeutic range is from 25 to 200 mg. daily. Patients may start at 25 mg. t.i.d. and dosage may be increased up to 200 mg./day according to patient needs.

Please see back cover for full prescribing information.

Pertofrane® (desipramine hydrochloride NF)

when activity is important

INDICATIONS: PERTOFRANE is indicated for the relief of mental depression (see Note before How Supplied section for additional information on depressive symptomatologies often responsive to this drug).

CONTRAINDICATIONS: The use of PERTOFRANE concomitantly or within two weeks of the administration of MAO inhibitors, is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse reactions can be serious, or even fatal. When it is desired to substitute PERTOFRANE in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation allows, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed.

The drug is contraindicated following recent myocardial infarction.

Patients with a known hypersensitivity to tricyclic antidepressants should not be given PERTOFRANE.

WARNINGS: As with other potent antidepressants, an activation of the psychosis may occasionally be observed in schizophrenic patients.

Due to the drug's atropine-like effects and sympathomimetic potentiation, PERTOFRANE should be used only with the greatest care in patients with narrow-angle glaucoma and with urethral or ureteral spasm.

Likewise, only when patient need outweighs the risk of serious adverse reactions should the drug be used in the presence of any of the following conditions: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (this agent has been shown to lower the seizure threshold).

In some instances, desipramine and the parent compound, imipramine, have been shown to block the pharmacologic action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents.

Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride therapy.

The concurrent use of other central nervous system drugs or alcohol may potentiate the adverse effects of desipramine hydrochloride. Since many such drugs may be used during surgery, it is recommended that desipramine hydrochloride be discontinued for as long as the clinical situation will allow prior to elective surgery.

Patients should be cautioned about the possibility of impaired ability to operate a motor vehicle or other dangerous machinery.

Usage in Pregnancy: Although teratogenic studies in mice, rats and rabbits have revealed no adverse effects, safe use of this drug in women who are or may become pregnant has not been definitely established. Therefore, PERTOFRANE should be withheld from these women unless the clinical situation warrants the potential risk.

In view of the lack of experience in children, the drug is not recommended for use in patients under twelve years of age.

Elderly and adolescent patients can usually be managed on lower dosage than that recommended for other patients and may not tolerate higher doses as well because of an increased incidence of adverse reactions.

PRECAUTIONS: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until remission occurs. These patients require careful supervision and protective measures during therapy.

Anxiety and increased agitation have been reported, particularly where depression is not the primary disorder. A shift to hypomanic or manic excitement may occur during therapy. Such reactions may necessitate discontinuation of the drug.

Although the anticholinergic activity of the drug is weak, in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents), atropine-like effects may be more pronounced (e.g. paralytic ileus).

Caution should be observed in prescribing the drug in hyperthyroid patients and in those receiving thyroid medications. Transient cardiac arrhythmias have occurred in rare instances.

In all patients undergoing extended courses of therapy, periodic blood and liver studies for signs of toxicity should supplement careful clinical observations.

ADVERSE REACTIONS: PERTOFRANE (desipramine hydrochloride) is well tolerated by most patients and severe complications or adverse reactions are infrequent. Those most often reported cause little discomfort and seldom require discontinuation of the drug.

Nervous System: Dizziness, drowsiness, insomnia, headache and disturbed visual accommodation have been reported with the drug. In addition, tremor, unsteadiness, tinnitus and paresthesias have been reported. Occasionally, mild extrapyramidal activity, falling and neuromuscular incoordination have been seen. A confusional state (with such symptoms as hallucinations and disorientation) may be produced, particularly in older patients and at higher dosage, and may require discontinuation of the drug. Changes in EEG patterns have been observed. Epileptiform seizures may occur. A reduction in dosage may help control some of these adverse reactions.

Gastrointestinal Tract: Anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea have been reported in patients receiving PERTOFRANE.

Skin: Skin rashes (including photosensitization), perspiration and flushing sensations have been reported during therapy.

Liver: Transient jaundice, apparently of an obstructive nature, and liver damage have been observed in rare cases. Elevation in transaminase and changes in alkaline phosphatase have occurred and should indicate repeated liver-function profiles. If progressive elevation occurs, the drug should be discontinued.

Blood Elements: Bone-marrow depression, agranulocytosis, thrombocytopenia and purpura have been reported. If these occur, the drug should be discontinued. Transient eosinophilia has been observed in some instances.

Cardiovascular System: Orthostatic hypotension and tachycardia have been observed but seldom require discontinuation of treatment. Patients who require concomitant vasodilating therapy should be carefully supervised, particularly during the initial phases.

Genitourinary System: Urinary frequency or retention and impotence have been reported.

Endocrine System: Occasional hormonal effects, including gynecomastia, galactorrhea and breast enlargement have been reported with PERTOFRANE (desipramine hydrochloride). In addition, decreased libido and estrogenic effect have been noted. The exact relationship of these adverse effects to the administration of the drug has not been established.

Sensitivity: Urticaria and rare instances of drug fever and cross-sensitivity with imipramine have been observed.

DOSAGE AND ADMINISTRATION: *All Patients Except Geriatric and Adolescent:* Optimal results are obtained at a dosage of approximately 150 mg./day. PERTOFRANE may be administered as two 25 mg. capsules or one 50 mg. capsule t.i.d. (150 mg. daily). When necessary, dosage may be increased, after initiation of therapy, up to 200 mg. daily in divided doses. Continued therapy at the optimal dosage level should be maintained during the active phase of the depression. It is recommended that a lower maintenance dosage be continued for at least two months after a satisfactory response has been achieved.

In cases of relapse due to premature withdrawal of the drug, a prompt response may be obtained by immediate resumption of treatment.

Geriatric and Adolescent Patients: Elderly and adolescent patients can usually be managed on lower dosage and may not tolerate higher doses as well as other patients. Therapy in these age groups may be initiated with 25 to 50 mg. daily. Dosage may be increased according to response and tolerance, but it is generally unnecessary to exceed 100 mg. daily in these patients. It is recommended that a lower maintenance dosage be continued for at least two months after a satisfactory response has been achieved.

Mild anxiety and agitation which often accompany cases of depression usually remit as the depression responds to PERTOFRANE. Occasionally, however, when anxiety and agitation are more serious, the adjunctive use of a sedative or tranquilizing agent may be indicated.

NOTE: Depressions which are most responsive to PERTOFRANE may be described in terms of "target symptoms." These include: despondency, sadness and depressed mood; fatigue; lack of interest and emotional response; helplessness, hopelessness, pessimism and despair; feelings of incapacity and inferiority; psychomotor retardation and inhibition; delusions of guilt and unworthiness; psychosomatic complaints; insomnia anorexia and weight loss; depression-related anxiety and agitation; suicidal drive.

It should be borne in mind that these "target symptoms" of depression assume their proper significance only after careful history, physical and mental evaluations and other investigative procedures have confirmed a diagnosis of one of the several types of depression.

HOW SUPPLIED: 25 mg. Capsules (pink), bottles of 100 and 1000 (NSN 6505-00-913-0382A). Also, 50 mg. Capsules (maroon and pink), bottles of 100 and 1000; single-dose blister packs, boxes of 500.

References: 1. Axelrod, J.: Biogenic amines and their impact in psychiatry, Semin. Psychiatry 4:199, 1972. 2. Friedhoff, A.: The biogenic amines: Brain biochemistry, in Mandates Depression (Rome, H. P., ed.), New York, Medcom, 1974. 3. Schildkraut, J. J.: Update: Biochemical pathways of depression, in Mandates Depression (Mead, B. T., ed.), New York, Medcom, 1975, pp. 8,10. 4. Schildkraut, J. J.: Norepinephrine metabolism in subtypes of depressive disorders, Psychopharmacol. Bull. 10:49, 1974. 5. Maas, J. W. et al.: Catecholamine metabolism, depressive illness, and drug response, Arch. Gen. Psychiatry 26:252, 1972. 6. Prange, A. J., Jr.: The use of drugs in depression: Its theoretical and practical basis, Psychiatr. Ann. 3:56, 1973. 7. Schildkraut, J. J.: Biogenic amines and affective disorders, Ann. Rev. Med. 25:333, 1974. 8. Hussar, D. A.: The drug therapy of depression, Therapeutics 2:3, 1974. 9. Krakowski, A. J.: Treatment of severe depression with desipramine, a metabolite of imipramine, Am. J. Psychiatry 120:494, 1963. 10. Lipton, M. A.: Summary and overview, Psychopharmacol. Bull. 10:57, 1974. 11. Biel, J. H.: Non-monamine oxidase inhibitor antidepressants structure-activity relationships, in Principles of Psychopharmacology (Clark, W. G. and del Giudice, J., eds.), New York, Academic Press, 1970, p. 272. 12. Ayd, F. J., Jr.: Int. Drug Ther. Newsletter 10:21, June 1975. 13. Byck, R.: Drugs and the treatment of psychiatric disorders, in The Pharmacological Basis of Therapeutics (Goodman, L. S. and Gilman, A., eds.), ed. 5, New York, Macmillan, 1975, p. 176. 14. Lapolla, A. and Jones, H.: Placebo-control evaluation of desipramine in depression, Am. J. Psychiatry 127:335, 1970. 15. Barnes, R. J.: Clinical Depression, USV, 1975. 16. Barringer, T. J.: Desipramine (Pertofrane) in the treatment of depression, Psychosomatics 6:326, 1965. 17. Cammer, L.: Rx for depression, in Depression: A Practical Approach (Davis, J. M., ed.), New York, Medcom, 1974, p. 26. 18. Lapolla, A.: Clinical trial of a new antidepressant (desipramine hydrochloride) in a hospital setting, Am. J. Psychiatry 121:1206, 1965. 19. Lafave, H. G. et al.: Desipramine and imipramine in an outpatient setting: A comparative study, Am. J. Psychiatry 122:698, 1965.

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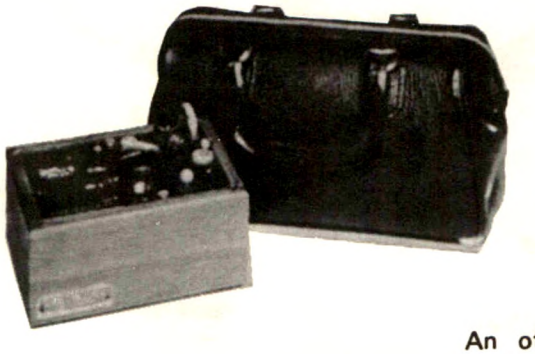
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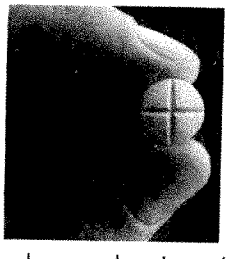
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ANTABUSE: "Social security" for the alcoholic who wants to stop drinking...



The temptation is resisted, ANTABUSE (disulfiram) represents the security alcoholics need in any situation. They are better able to abstain from drinking because they know the consequences of taking "even one."

until psychiatric support can do the rest



Undoubtedly it will take considerable time for psychotherapy to help the chronic alcoholic. As a psychiatrist, you have undoubtedly selected the patients who are best motivated to stay with ANTABUSE while you attempt to deal with the underlying emotional problems. And, of course, you know the importance of reminding patients of the serious consequences of drinking while on ANTABUSE.

Once sober, and well into treatment, the alcoholic, at your discretion, can participate in a total treatment program which makes use of various supportive resources in the community. In the meantime, ANTABUSE can help the chronic alcoholic abstain from drinking.

BRIEF SUMMARY
(For full prescribing information, see package circular.)

ANTABUSE® (disulfiram) in Alcoholism

INDICATION: ANTABUSE is an aid in the management of selected chronic alcoholic patients who want to remain in a state of enforced sobriety so that supportive and psychotherapeutic treatment may be applied to best advantage. (Used alone, without proper motivation and without supportive therapy, ANTABUSE is not a cure for alcoholism, and it is unlikely that it will have more than a brief effect on the drinking pattern of the chronic alcoholic.)

CONTRAINDICATIONS: Patients who are receiving or have recently received metronidazole, paraldehyde, alcohol, or alcohol-containing preparations, e.g. cough syrups, tonics, and the like, should not be given ANTABUSE.

ANTABUSE is contraindicated in the presence of severe myocardial disease or coronary occlusion, psychoses, or hypersensitivity.

WARNINGS: ANTABUSE should never be administered to a patient when he is in a state of alcohol intoxication or without his full knowledge.

The physician should instruct relatives accordingly.

The patient must be fully informed of the ANTABUSE-alcohol reaction. He must be strongly cautioned against surreptitious drinking while taking the drug, and he must be fully aware of possible consequences. He should be warned to avoid alcohol in disguised form, *i.e.* in sauces, vinegars, cough mixtures, and even aftershave lotions and back rubs. He should also be warned that reactions may occur with alcohol up to 14 days after ingesting ANTABUSE.

THE ANTABUSE-ALCOHOL REACTION: ANTABUSE plus alcohol, even small amounts, produces flushing, throbbing in head and neck, throbbing headache, respiratory difficulty, nausea, copious vomiting, sweating, thirst, chest pain, palpitation, dyspnea, hyperventilation, tachycardia, hypotension, syncope, marked uneasiness, weakness, vertigo, blurred vision, and confusion. In severe reactions there may be respiratory depression, cardiovascular collapse, arrhythmias, myocardial infarction, acute congestive heart failure, unconsciousness, convulsions, and death.

The intensity of the reaction varies with each individual, but is generally proportional to the amounts of ANTABUSE (disulfiram) and alcohol ingested. Mild reactions may occur in the sensitive individual when the blood alcohol concentration is increased to as little as 5 to 10 mg. per 100 cc. Symptoms are fully developed at 50 mg. per 100 cc., and unconsciousness usually results when the blood alcohol level reaches 125 to 150 mg.

The duration of the reaction varies from 30 to 60 minutes to several hours in the more severe cases, or as long as there is alcohol in the blood.

DRUG INTERACTIONS: Disulfiram appears to decrease the rate at which certain drugs are metabolized and so may increase the blood levels and the possibility of clinical toxicity of drugs given concomitantly.

Disulfiram should be used with caution in those patients receiving diphenylhydantoin and its congeners, since toxic levels of these antiepileptic agents have been reported during concomitant disulfiram therapy.

It may be necessary to adjust the dosage of oral anticoagulants upon beginning or stopping disulfiram, since disulfiram may prolong prothrombin time.

Patients taking isoniazid when disulfiram is given should be observed for the appearance of unsteady gait or marked changes in mental status and the disulfiram discontinued if such signs appear.

CONCOMITANT CONDITIONS: Because of the possibility of an accidental ANTABUSE-alcohol reaction, ANTABUSE (disulfiram) should be used with extreme caution in patients with any of the following conditions: diabetes mellitus, hypothyroidism, epilepsy, cerebral damage, chronic and acute nephritis, hepatic cirrhosis or insufficiency.

USAGE IN PREGNANCY: The safe use of this drug in pregnancy has not been established. Therefore, ANTABUSE (disulfiram) should be used during pregnancy only when, in the judgment of the physician, the probable benefits outweigh the possible risks.

PRECAUTIONS: It is suggested that every patient under treatment carry an Identification Card, stating that he is receiving ANTABUSE and describing the symptoms most likely to occur as a result of the ANTABUSE-alcohol reaction. In addition, this card should indicate the physician or institution to be contacted in emergency. (Cards may be obtained from Ayerst Laboratories upon request.)

Alcoholism may accompany or be followed by dependence on narcotics or sedatives. Barbiturates have been administered concurrently with ANTABUSE (disulfiram) without untoward effects, but the possibility of initiating a new abuse should be considered.

Base line and follow-up transaminase tests (10-14 days) are suggested to detect any hepatic dysfunction that may result with ANTABUSE therapy. In addition, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

ADVERSE REACTIONS: (See Contraindications, Warnings, and Precautions.)

Occasional skin eruptions are, as a rule, readily controlled by concomitant administration of an antihistaminic drug.

In a small number of patients, a transient mild drowsiness, fatigability, impotence, headache, acneform eruptions, allergic dermatitis, or a metallic or garlic-like aftertaste may be experienced during the first two weeks of therapy. These complaints usually disappear spontaneously with the continuation of therapy or with reduced dosage.

Psychotic reactions have been noted, attributable in most cases to high dosage, combined toxicity (with metronidazole or isoniazid), or to the unmasking of underlying psychoses in patients stressed by the withdrawal of alcohol.

There have been reports of polyneuritis and peripheral neuritis, and rare instances of optic neuritis. One case of cholestatic hepatitis has been reported, but its relationship to ANTABUSE has not been unequivocally established.

DOSAGE AND ADMINISTRATION: ANTABUSE (disulfiram) should never be administered until the patient has abstained from alcohol for at least 12 hours.

INITIAL DOSAGE SCHEDULE: In the first phase of treatment, a maximum of 500 mg. daily is given in a single dose for one to two weeks. Although usually taken in the morning, ANTABUSE may be taken on retiring by patients who experience a sedative effect. Alternatively, to minimize, or eliminate, the sedative effect, dosage may be adjusted downward.

MAINTENANCE REGIMEN: The average maintenance dose is 250 mg. daily (range, 125 to 500 mg.); it should not exceed 500 mg. daily.

NOTE: Occasional patients, while seemingly on adequate maintenance doses of ANTABUSE (disulfiram), report that they are able to drink alcoholic beverages with impunity and without any symptomatology. All appearances to the contrary, such patients must be presumed to be disposing of their tablets in some manner without actually taking them. Until such patients have been observed reliably taking their daily ANTABUSE tablets (preferably crushed and well mixed with liquid), it cannot be concluded that ANTABUSE is ineffective.

DURATION OF THERAPY: The daily, uninterrupted administration of ANTABUSE must be continued until the patient is fully recovered socially and a basis for permanent self-control is established. Depending on the individual patient, maintenance therapy may be required for months or even years.

TRIAL WITH ALCOHOL: During early experience with ANTABUSE, it was thought advisable for each patient to have at least one supervised alcohol-drug reaction. More recently, the test reaction has been largely abandoned. Furthermore, such a test reaction should never be administered to a patient over 50 years of age. A clear, detailed, and convincing description of the reaction is felt to be sufficient in most cases.

However, where a test reaction is deemed necessary, the suggested procedure is as follows:

After the first one to two weeks' therapy with 500 mg. daily, a drink of 15 cc. (½ oz.) of 100 proof whiskey or equivalent is taken slowly. This test dose of alcoholic beverage may be repeated once only so that the total dose does not exceed 30 cc. (1 oz.) of whiskey. Once a reaction develops, no more alcohol should be consumed. Such tests should be carried out only when the patient is hospitalized, or comparable supervision and facilities, including oxygen, are available.

MANAGEMENT OF ANTABUSE (DISULFIRAM)-ALCOHOL REACTION: In severe reactions, whether caused by an excessive test dose or by the patient's unsupervised ingestion of alcohol, supportive measures to restore blood pressure and treat shock should be instituted. Other recommendations include: oxygen, carbogen (95 per cent oxygen and 5 per cent carbon dioxide), vitamin C intravenously in massive doses (1 Gm.), and ephedrine sulfate. Antihistamines have also been used intravenously. Potassium levels should be monitored particularly in patients on digitalis since hypokalemia has been reported.

HOW SUPPLIED: No. 809—Each tablet (scored) contains 250 mg. disulfiram, in bottles of 100. No. 810—Each tablet (scored) contains 500 mg. disulfiram, in bottles of 50 and 1,000.

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BRAND OF DISULFIRAM

The TRIAVIL® Potential in the management of moderate to severe anxiety with depression

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The therapist is the primary catalyst for change in the psychotherapeutic relationship. However, when patients suffer from moderate to severe anxiety with depression, there are situations when TRIAVIL can often be a useful adjunct.

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There are three important benefits you may expect when TRIAVIL is part of the treatment program: (1) When symptoms of moderate to severe anxiety or agitation with depression are relieved, the patient may become more accessible and cooperative. (2) As somatic manifestations are controlled, attention may be focused on underlying causative factors. (3) Symptomatic relief may enable the patient to function more effectively in his daily life while your work with the patient progresses.

Tablets TRIAVIL are available in four different combinations affording flexibility and individualized dosage adjustment. Since it is simpler to remember to take one tablet rather than several (particularly in multiple daily doses), your patients on TRIAVIL will be more likely to take proper doses of the medication.

TRIAVIL is contraindicated in CNS depression from drugs; in the presence of evidence of bone marrow depression; and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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TRIAVIL® **containing perphenazine and amitriptyline HCl**

a tranquilizer-antidepressant

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FOR FLEXIBILITY IN ADJUSTING MAINTENANCE THERAPY
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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Do not give concomitantly with MAOI drugs because hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. Allow minimum of 14 days between therapies, then initiate therapy with TRIAVIL cautiously, with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given with guanethidine or similarly acting compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, particularly in high doses, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. Caution patients performing hazardous tasks, such as operating machinery or driving motor vehicles, that drug may impair mental and/or physical abilities. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Side effects may be any of those reported with phenothiazine drugs: extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and that the full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; hypnotic effects; pigmentary retinopathy; corneal and lenticular pigmentation; occasional lassitude, muscle weakness, mild insomnia. Other adverse reactions reported with various phenothiazine compounds include blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); liver damage (jaundice, biliary stasis); grand mal convulsions; cerebral edema; polyphagia; photophobia; skin pigmentation; and failure of ejaculation.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness; weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; jaundice; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

For more detailed information, consult your MSD Representative or see full Prescribing Information.
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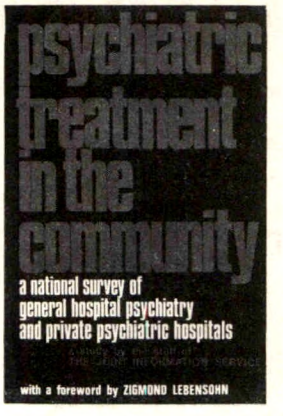
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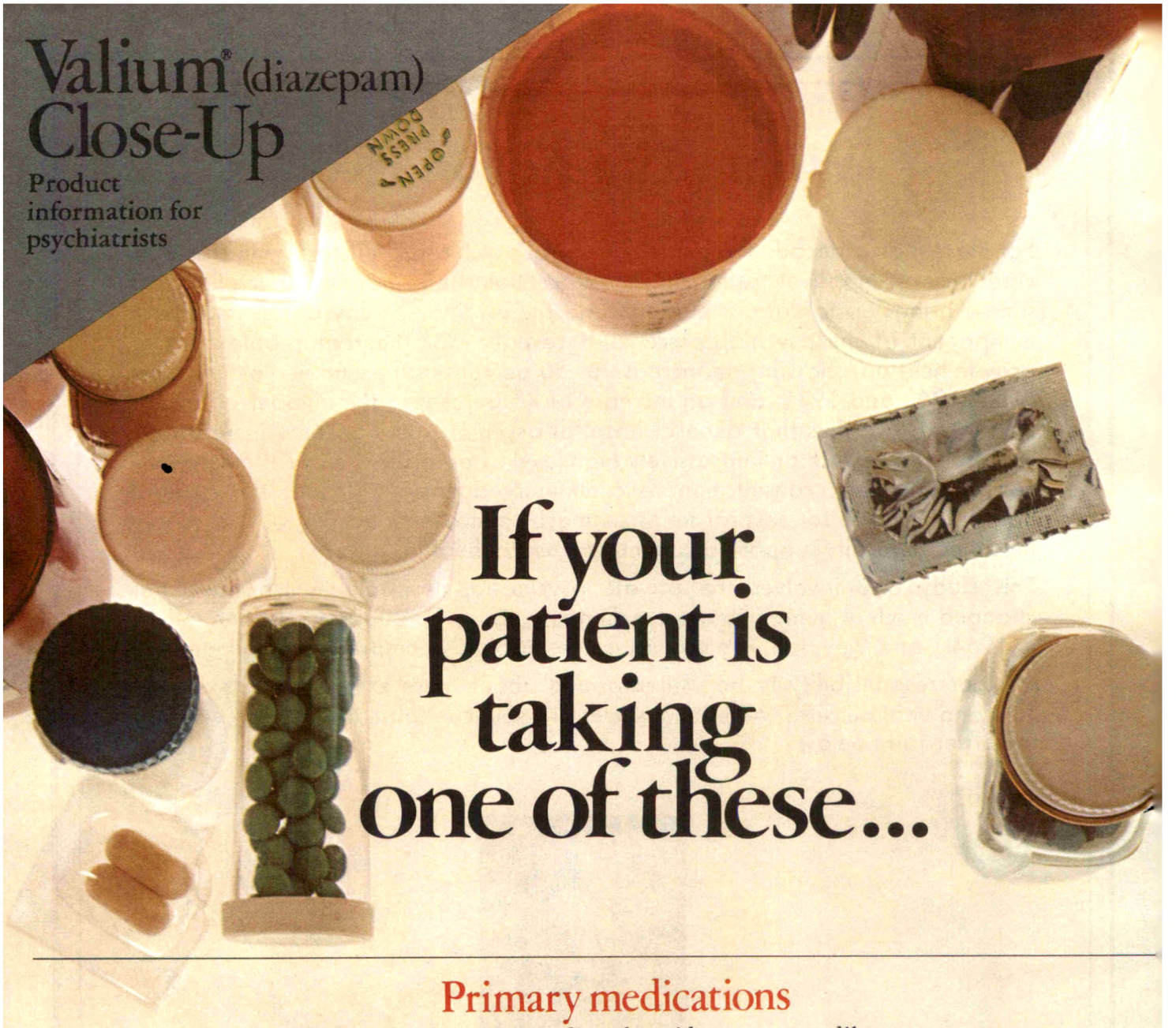
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Anticonvulsants

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Valium® (diazepam) ℞

2-mg, 5-mg, 10-mg scored tablets

Before prescribing, please see following page for a summary of product information.

Valium® (diazepam)

2-mg, 5-mg, 10-mg scored tablets

can relieve psychoneurotic anxiety and its associated depressive symptoms

Prompt, effective action. Valium works rapidly to relieve pronounced psychic tension in patients overreacting to stress and in psychoneurotic patients.

Wide margin of safety. Valium is generally well tolerated and in usual dosages rarely produces significant adverse reactions.

Dosage flexibility. Scored Valium 2-, 5-, 10-mg tablets give you dosage flexibility no tranquilizer capsule can match.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline (primidone) for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards— used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate—As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail— A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

Ayerst®

Mysoline® (primidone)

Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

7538

Mysoline® (primidone)

may be the start of a better life for the epileptic

initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

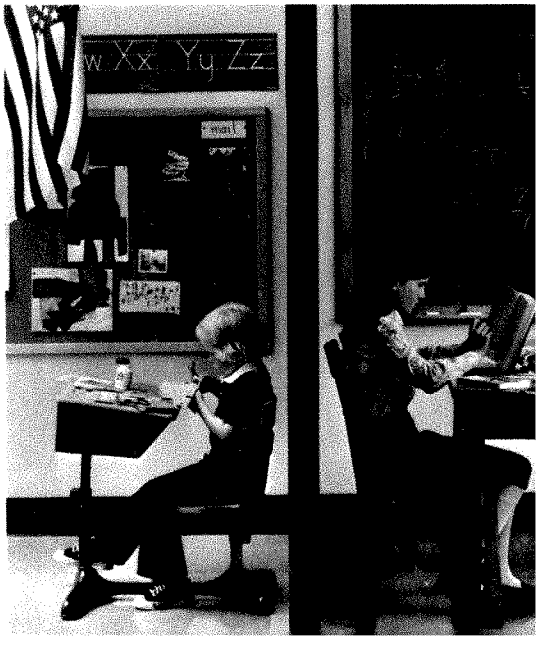
In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (teaspoonful) contains 250 mg. of primidone, in bottles of 8 fluid-ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. *37*:49 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. *47*:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. *68*:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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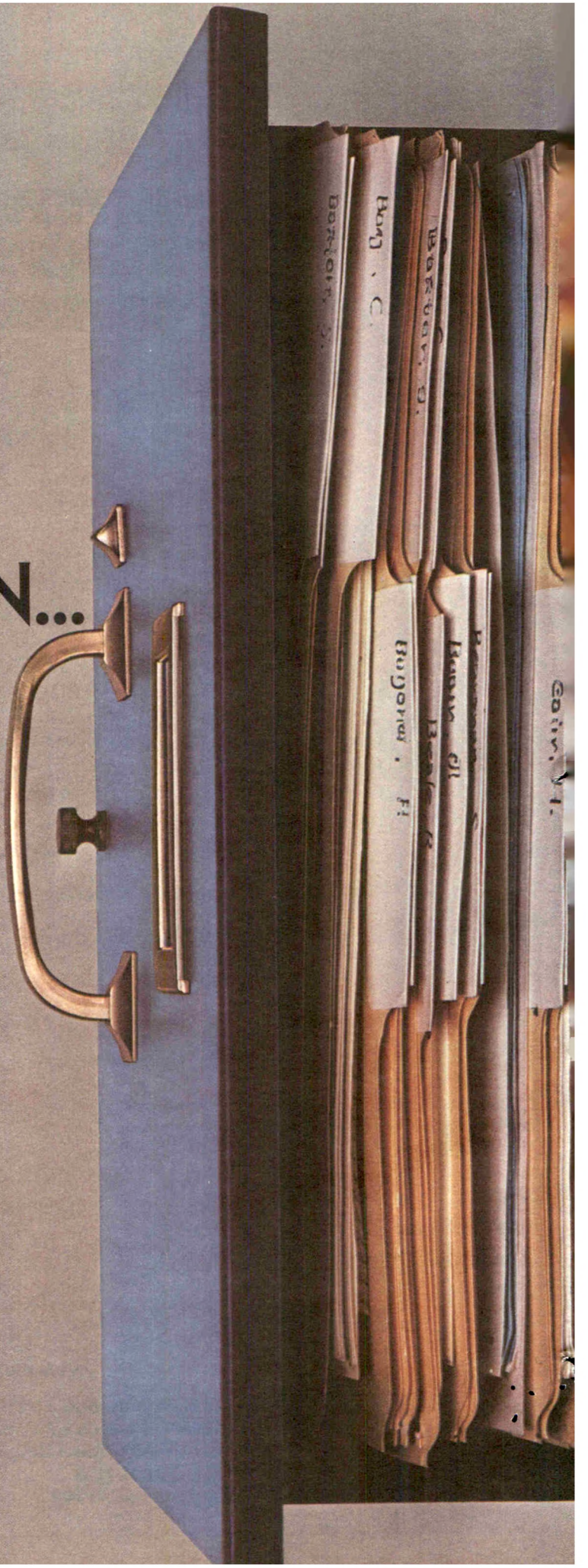
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100-mg., 50-mg., 25-mg. Capsules and ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

MAY BE AN APPROPRIATE CHOICE FOR YOUR NEXT PATIENT WITH CLINICAL DEPRESSION/ANXIETY

See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

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DOXEPIN HCl

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ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

Proved efficacy in the treatment of clinical depression/anxiety

In the five years since its introduction, Sinequan (doxepin HCl) has objectively demonstrated its value in the treatment of clinical depression/anxiety. Clinical studies have shown Sinequan to be unsurpassed in effectiveness when compared to other tricyclic antidepressants.

In addition, Sinequan may offer certain advantages not generally seen with some other drugs of its class:

Marked antianxiety activity

In addition to its significant antidepressant activity, which is usually seen two to three weeks after initiation of therapy, Sinequan possesses marked antianxiety activity.

In double-blind comparisons between doxepin and amitriptyline,[1-3] the antidepressant effects of both drugs were comparable; however, more notable antianxiety effects were seen with doxepin. Superior antianxiety activity was reported among depressed patients treated in a crisis intervention clinic.[4]

Prompt sedative activity

Of the tricyclic antidepressants, Sinequan has been found to have one of the most prominent sedative effects.[5] This sedative property can be taken advantage of by administering the major portion of the daily dose *h.s.* Since clinically depressed/anxious patients frequently have accompanying sleep disturbances, this may be a valuable benefit.

Generally does not affect the activity of guanethidine at usual clinical doses[6]

"A new tricyclic antidepressant, doxepin, was evaluated for its ability to block the amine uptake mechanisms of the peripheral adrenergic neuron and blood platelet in man. At low doses, there was no evidence of inhibition of the amine pump." At clinical dosages up to 150 mg. per day, Sinequan can be given to man concomitantly with guanethidine and related compounds without blocking the antihypertensive effect.[6]

Cardiovascular side effects reported infrequently

In a series of double-blind comparative studies, 1706 doxepin-treated patients were evaluated for side effects. Incidence of tachycardia was associated with only 45 (2.6%) clinically depressed/anxious patients.[3]

REFERENCES:
1. Grof, P. et al.: Curr. Ther. Res. 16:470, May 1974.
2. Hackett, E. and Kline, N. S.: Psychosomatics 10:21, May-June (Section II) 1969.
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4. Kiev, A. and Slavin, J. R.: Scientific Exhibit, Presented at the 126th Annual Meeting of the American Psychiatric Association, Honolulu, Hawaii, May 7-11, 1973.
5. Claghorn, J. L., in Claghorn, J. L. et al.: Patient Care 5:108, Oct. 15, 1971.
6. Fann, W. E. et al.: Psychopharmacologia 22:111, 1971.

See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

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MAY BE AN APPROPRIATE CHOICE FOR YOUR NEXT CLINICALLY DEPRESSED/ANXIOUS PATIENT

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and
ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, paresthesias, ataxia, and extrapyramidal symptoms.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, facial edema, photosensitization, pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, and anorexia have been reported. (See anticholinergic effects).

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice and alopecia have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg. t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg./day to 150 mg./day.

In more severely ill patients an initial dose of 50 mg. t.i.d. may be required with subsequent gradual increase to 300 mg./day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg./day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg./day.

As an alternative regimen, the total daily dosage, up to 150 mg., may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Supply. Available as capsules containing doxepin HCl equivalent to 10 mg., 25 mg., 50 mg., and 100 mg. of doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN (doxepin HCl) 25 mg. and 50 mg. also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg./ml.) is available in 120 ml. bottles with an accompanying dropper calibrated at 5 mg., 10 mg., 15 mg., 20 mg., and 25 mg. SINEQUAN Oral Concentrate should be diluted with approximately 120 ml. of water or orange juice prior to administration.

More detailed professional information available on request.

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In clinically significant depression



TABLETS, 5 mg and 10 mg

Vivactil®*

(Protriptyline HCl | MSD)

***When you want an antidepressant that is characteristically nonsedating for the patient under close medical supervision (Symptoms such as anxiety or agitation may be aggravated.)**

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information please see following page.

In clinically significant depression

TABLETS, 5 mg and 10 mg

Vivactil®

(Protriptyline HCl | MSD)

Contraindications: Known hypersensitivity; acute recovery phase following myocardial infarction. Should not be given concomitantly with an MAOI; hyperpyretic crises, severe convulsions, and deaths have occurred in patients receiving tricyclic antidepressant and MAOI drugs simultaneously. When it is desired to substitute protriptyline HCl for an MAOI, a minimum of 14 days should be allowed to elapse after the latter is discontinued. Protriptyline HCl should then be initiated cautiously with gradual increase in dosage until optimum response is achieved.

Warnings: May block the antihypertensive effect of guanethidine or similarly acting compounds. May impair mental and/or physical abilities required for the performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Should be used with caution in patients with a history of seizures and, because of its autonomic activity, in patients with a tendency to urinary retention or increased intraocular tension.

Tachycardia and postural hypotension may occur more frequently than with other antidepressant drugs. Should be used with caution in elderly patients and patients with cardiovascular disorders; such patients should be observed closely because of the tendency of the drug to produce tachycardia, hypotension, arrhythmias, and prolongation of the conduction time. Myocardial infarction and stroke have occurred with drugs of this class. On rare occasions, hyperthyroid patients or those receiving thyroid medication may develop arrhythmias when this drug is given.

Usage in Children: Not recommended for use in children because safety and effectiveness in the pediatric age group have not been established.

Usage in Pregnancy: Safe use in pregnancy and lactation has not been established; therefore, use in pregnant women, nursing mothers, or women who may become pregnant requires that possible benefits be weighed against possible hazards to mother and child.

Precautions: When protriptyline HCl is used to treat the depressive component of schizophrenia, psychotic symptoms may be aggravated; likewise, in manic-depressive psychosis, depressed patients may experience a shift toward the manic phase; paranoid delusions, with or without associated hostility, may be exaggerated. In any of these circumstances, it may be advisable to reduce the dose of protriptyline HCl or to use a major tranquilizing drug concurrently. Symptoms, such as anxiety or agitation, may be aggravated in overactive or agitated patients.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. May enhance response to alcohol and effects of barbiturates and other CNS depressants. Possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent administration with electroshock therapy may increase hazards of therapy; such treatment should be limited to patients for whom it is essential. Discontinue drug several days before elective surgery, if possible. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions which have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when protriptyline HCl is administered. Protriptyline HCl is more likely to aggravate agitation and anxiety and produce cardiovascular reactions such as tachycardia and hypotension.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions, anxiety, restlessness, agitation; insomnia, panic, and nightmares; hypomania; exacerbation of psychosis.

Neurological: numbness, tingling, and paresthesias of extremities; incoordination, ataxia, tremors, peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns, tinnitus.

Anticholinergic: dry mouth and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of the urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (general, or of face and tongue), drug fever.

Hematologic: bone marrow depression; agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of other tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets, containing 5 mg and 10 mg protriptyline HCl each, in single-unit packages of 100 and bottles of 100 and 1000.

For more detailed information, consult your MSD representative or see full prescribing information.

Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

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Elmira is located in the beautiful Finger Lakes Region of Western New York, an area offering a year-round variety of sports and recreational activities. Ten colleges, including Cornell University, are within commuting distance. Elmira and its neighboring communities take pride in their good schools, excellent shopping areas, and pleasant pastoral surroundings. Syracuse and Rochester are located approximately two hours away.

We invite your participation in this institution. For more information please write to Director
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is seeking a Chairman for the Section of Psychiatry and Human Behavior and Psychiatrist in Chief for the Butler Hospital. Inquiries and applications with curricula vitae should be addressed to Dr. Leo Stern, Chairman, Psychiatry Search Committee, Section of Reproductive and Developmental Medicine, Women & Infants Hospital, 50 Maude Street, Providence, Rhode Island 02908. Both Brown University and Butler Hospital are equal opportunity employers.

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Introducing

The unforgettable antidepressant

Endep™ 75 mg

amitriptyline HCl / Roche

Here's what makes it unforgettable



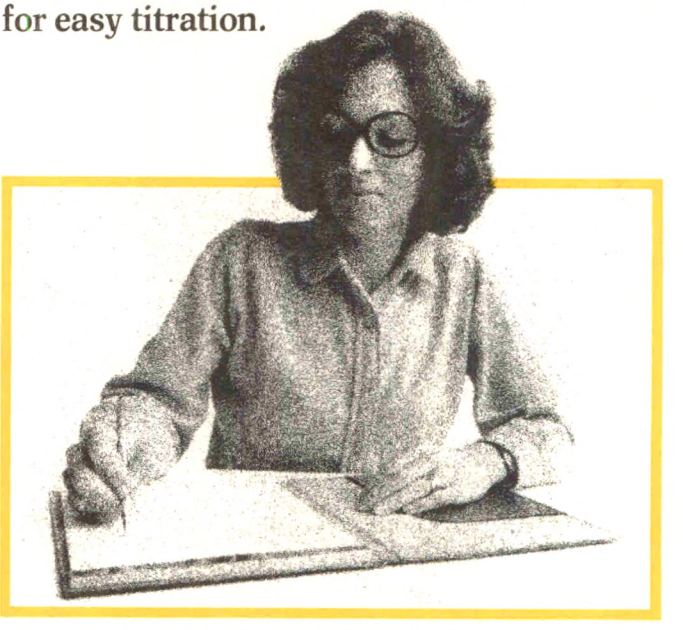
Patients are more likely to remember the single bedtime dosage of Endep (amitriptyline HCl).



You'll remember 1 or 2 tablets *h.s.*, because fewer forgotten doses produce a more consistent clinical effect, and because it is the only tricyclic antidepressant scored for easy titration.



Patients will also remember family/interpersonal relationships that they have forgotten or neglected.



In three or four weeks patients will usually remember how well they used to feel.

Endep™ 75 mg

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day

Before prescribing, please consult complete product information, a summary of which appears on the following page.

Introducing Endep™

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day



10-mg scored tablets | **25-mg** scored tablets | **50-mg** scored tablets | **75-mg** scored tablets

■ Endep (amitriptyline HCl), in responsive patients, lifts mood and relieves symptoms such as anorexia, loss of interest, inability to concentrate, decreased libido or GI disturbances, often within three or four weeks after starting therapy.

■ Endep usually relieves accompanying sleep disturbances, particularly early in the treatment regimen.

■ All four dosage strengths are scored for greater flexibility and convenience in titration.

■ The 75-mg *h.s.* dose minimizes skipped doses, maximizes patient acceptance and compliance.

■ Specifying Endep/Roche assures quality plus potential economy.

Important Considerations

Should not be given concomitantly with an MAO inhibitor.

Not recommended for use during the acute recovery phase following myocardial infarction.

Contraindicated in patients with hypersensitivity to amitriptyline.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh potential benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia. purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels; increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier. *Initial Adult Dosage:* Outpatients – 25 mg t.i.d.; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate – 50 to 100 mg h.s., gradually increasing h.s. dose up to 150 mg/day. Hospitalized Patients – Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day. *Adolescent and Elderly Patients:* In general, 10 mg t.i.d. with 20 mg h.s. may be satisfactory for those who do not tolerate higher doses. *Maintenance Dosage:* With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg b.i.d. to q.i.d., or 10 mg q.i.d. Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg and 75-mg scored tablets – bottles of 100 and 500; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg), available singly and in trays of 10.

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Expanding midwestern community of 100,000 with desirable environment. Varied opportunities for private inpatient and outpatient psychiatry with Mental Health Center consultation, if desired. Clinical appointments available on new four-year medical school faculty. Details as to type of practice, financial arrangement, etc. are open.

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is a thousand bed psychiatric facility located in Bolivar, Tennessee. Positions are available for Hospital Superintendent and staff psychiatrist. Board Certified/Eligible and Tennessee license are required. Salary per qualifications with the following bases:

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Tennessee Department of Mental Health and Mental Retardation
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Nashville, Tennessee 37219
Phone: 615-741-3348

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The Prince George Regional Hospital is requesting applications from PSYCHIATRISTS. The hospital currently expanded to a 370 bed, (including a 27 bed Psychiatric Unit), fully accredited; active treatment Regional referral hospital, located in Central B.C., is situated in the City of Prince George. The present population of Prince George is approximately 70,000, and serves an area of about 150,000. Prince George is one of the fastest growing communities in the province and offers excellent educational and recreational facilities with easy access to Vancouver and other parts of Canada. Please direct enquiries to: Dr. P.J. Konkal, Chairman, Medical Manpower Committee, Prince George Regional Hospital, Prince George, B.C.

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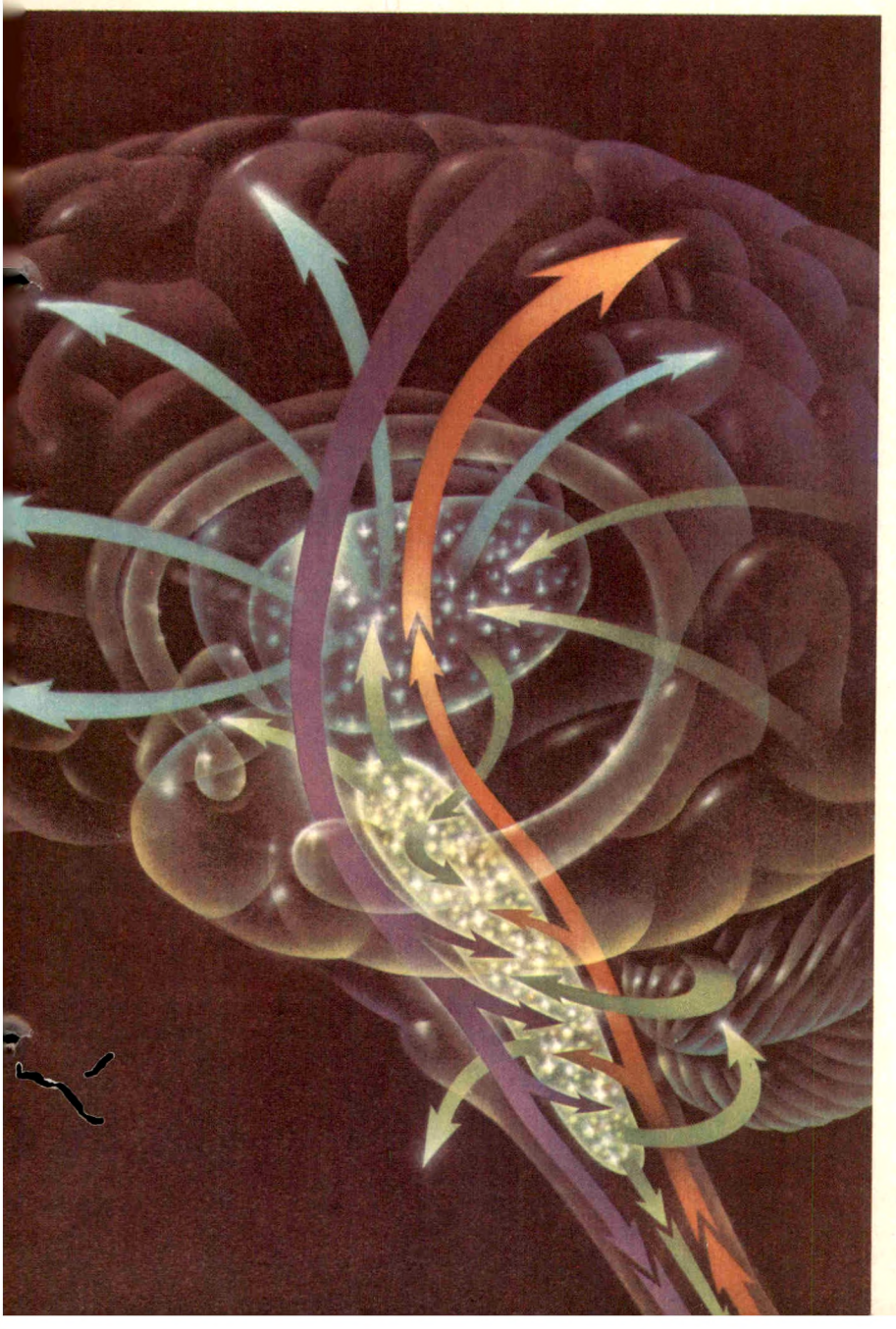
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Whether your schizophrenic patient is hospitalized or ambulatory, he may benefit from a different chemical modality

MOBAN®
(molindone HCl)



acts on the RAS with low incidence of adverse effects

EEG studies show that MOBAN® exerts its effect on the ascending reticular activating system. This often helps to encourage increased social participation, receptivity and communication in schizophrenic patients.

Artist's representation of the reticular activating system, with neuronal activity passing on to cortical and subcortical structures.

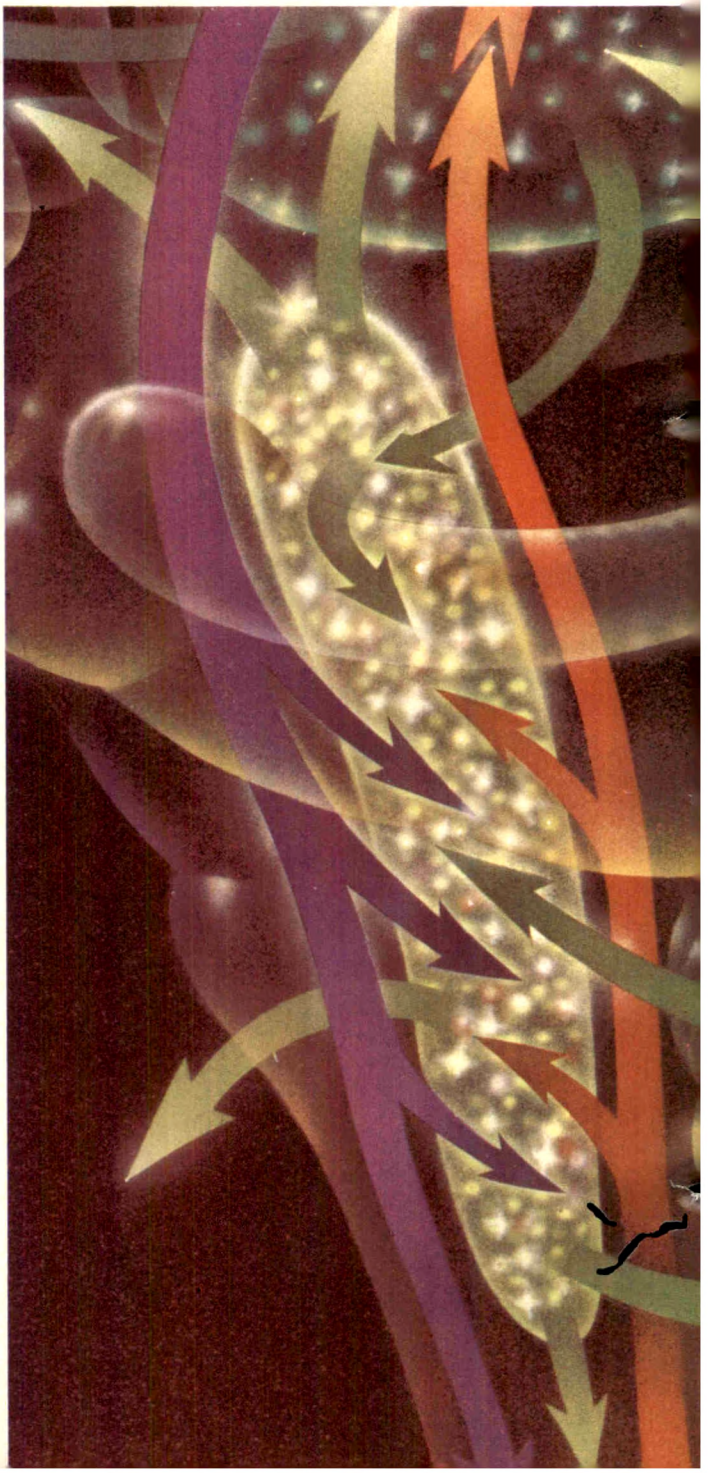
for a chemical difference in treating the schizophrenias

MOBAN®
(molindone HCl)

- generally minimizes drowsiness to help produce improved functioning
  1) to hasten the discharge of hospitalized patients
  2) to keep ambulatory patients at home or on the job

- tranquilizes without muscle relaxant or incoordinating effect

- acts to alleviate target symptoms or reduce their severity
  1) in acute patients, some symptomatic improvement evident in a few days
  2) in chronic patients, symptomatic improvement may not be evident until 2-4 weeks
  3) maximum benefits attained by end of third month (continued maintenance therapy as required)



In early clinical trials, of 425 patients studied, 72% showed overall global improvement in double-blind studies.*

*Data on file, Endo Laboratories

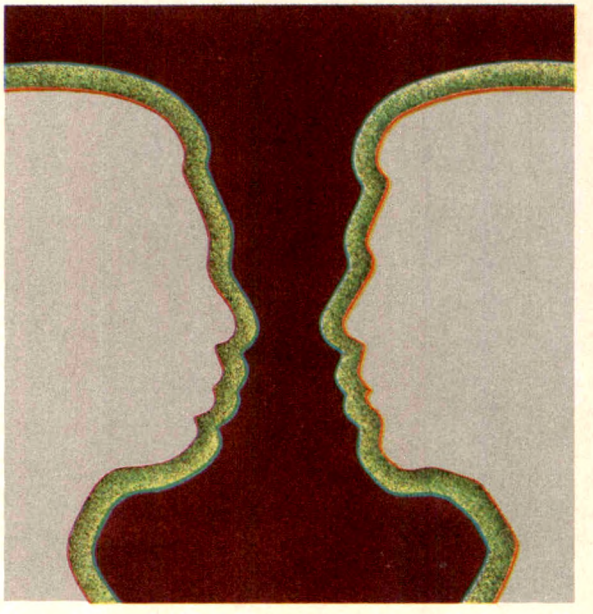
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Subsidiary of E.I. du Pont de Nemours & Co. (Inc.)
Garden City, N.Y. 11530
DUPONT

SOCIAL ADJUSTMENT PROFILE CHART

of 66 hospitalized schizophrenic patients treated with MOBAN® (molindone HCl) for 1 year or more*

Adjustment	Percent
To live on open ward	91%
To function with privileges	89%
To hold hospital job	85%
To leave hospital	70%
To maintain home	33%
To hold employment	32%

*Data on file, Endo Laboratories



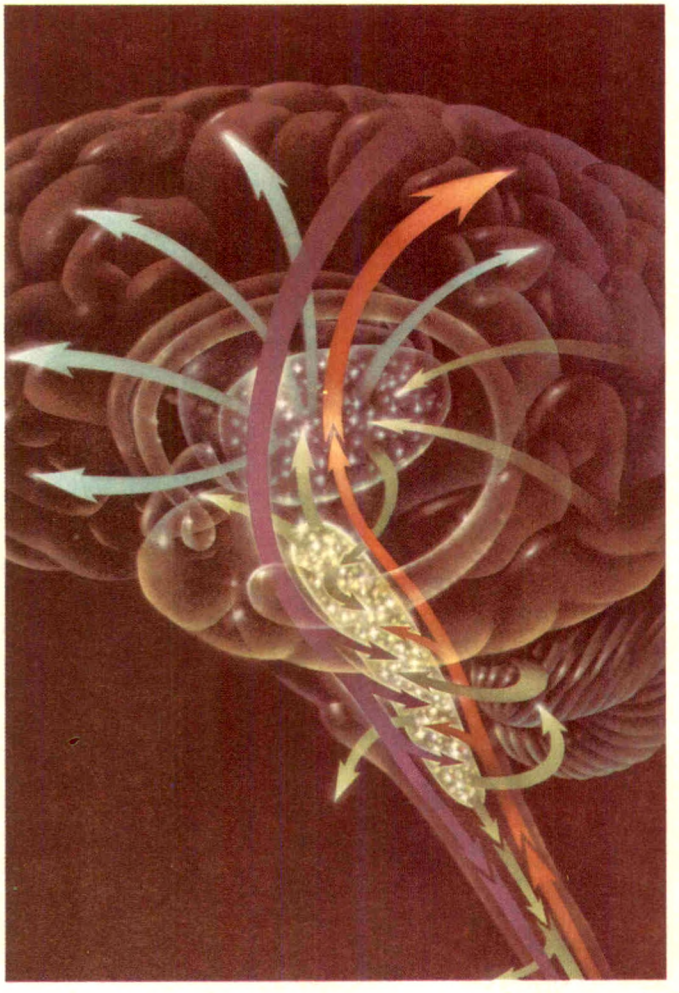
social participation
receptivity
communication

there's something different about

MOBAN®
(molindone HCl)

Please see last page of advertisement for full prescribing information.

Whenever your patient may benefit from less sedation, more activation– MOBAN® (molindone HCl)



CLINICAL EXPECTATIONS FROM MOBAN® (molindone HCl)

The following has been compiled from clinical and field reports, according to the best available information to date.

activation The pattern that has emerged for MOBAN®. This may conflict with usual nursing procedure on the ward at first, but is usually an early indication of improvement.

agitated patient These patients may require a more rapid increase of dose to control symptoms. (Caution should be exercised where increased activity may be harmful.)

amenorrhea Has rarely developed with MOBAN®, as it has with other antipsychotics. Amenorrheic patients have returned to normal menses when switched to MOBAN® from other antipsychotics. Initial periods may be somewhat heavy and prolonged, but soon stabilize to normal.

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dopamine blockage Demonstrated in animals.

drug interaction None reported to date with commonly co-prescribed drugs. Potentiation of drugs administered concurrently with MOBAN® has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs *(i.e.,* barbiturates, chloral hydrate and antiparkinson drugs).

duration of action Exceeding 24 hours.

euphoria Experienced by some patients.

extrapyramidal symptoms Similar to other antipsychotics, but easily controlled. Akathisia most frequently reported, dystonia rare.

insomnia If it occurs, decrease daytime dosage, increase evening dosage.

laboratory values Within normal limits.

lactation A rarity with MOBAN®

lenticular opacity None reported to date. A decrease noted after switching to MOBAN®.

libido No decrease in libido. An increase noted in some patients.

liver dysfunction None reported to date.

peak blood levels Unmetabolized drug peaks at 1 hour, pharmacologic effect persists for 24-36 hours.

relative milligram equivalency MOBAN® is to chlorpromazine 1:6.

sedation MOBAN® may produce transient sedation (1-2 weeks) and has seldom had a prolonged sedative effect.

side effects Seldom does MOBAN® cause the peripheral and endocrine effects produced by the aliphatic and piperidine phenothiazines. MOBAN® may cause extrapyramidal reactions, transient initial drowsiness, fatigue, dizziness, mild hypotension, constipation, weight changes. Other potential adverse reactions described in the accompanying prescribing information.

skin pigmentation None reported to date.

switching Allow time for the previous neuroleptic's effect to wear off and the difference in side effects; *e.g.,* amenorrhea versus normal flow. Also allow for difference in strength until MOBAN® reaches therapeutic dose.

tardive dyskinesia None reported to date.

tolerance None reported to date.

weight change Some obese patients lost large amounts of weight while eating normally. Limited weight gain was noted in other patients.

a dihydroindolone chemically distinct from phenothiazines, thioxanthenes, butyrophenones

MOBAN®
(molindone HCl)

to help control the manifestations of schizophrenia

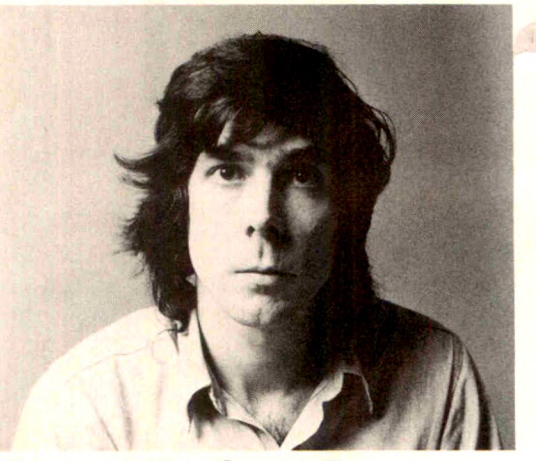
Please see last page of advertisement for full prescribing information.

When to use MOBAN® (molindone HCl)

- for new patients, acute or chronic
- for hospitalized patients, those in hospital clinics and community mental health centers, or those at home or on the job
- when poor results are obtained with other antipsychotics
- when certain undesirable side effects occur with other antipsychotics
- as a replacement for polypharmacy, thus reducing cost, custodial care, risk of side effects
- if patient is refractory to other treatment (after diagnostic reappraisal)

THE IN-HOSPITAL PATIENT.

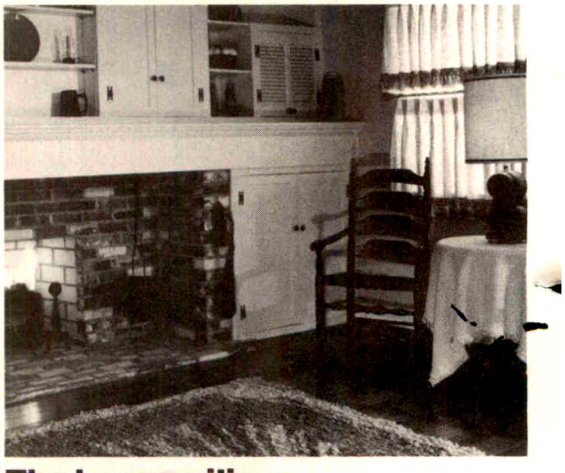
Activation may make the patient more productive. Sedation may increase the patient's baseline anergic vegetative qualities. An activating effect may not benefit every patient.



The neuroleptic syndrome:
anergic vegetative qualities increase.

THE AT-HOME PATIENT.

The hope is that this patient can not only maintain a home but hold a job. That will probably require daytime alertness. For many outpatients, sedation may impair efficiency while activation might improve it.

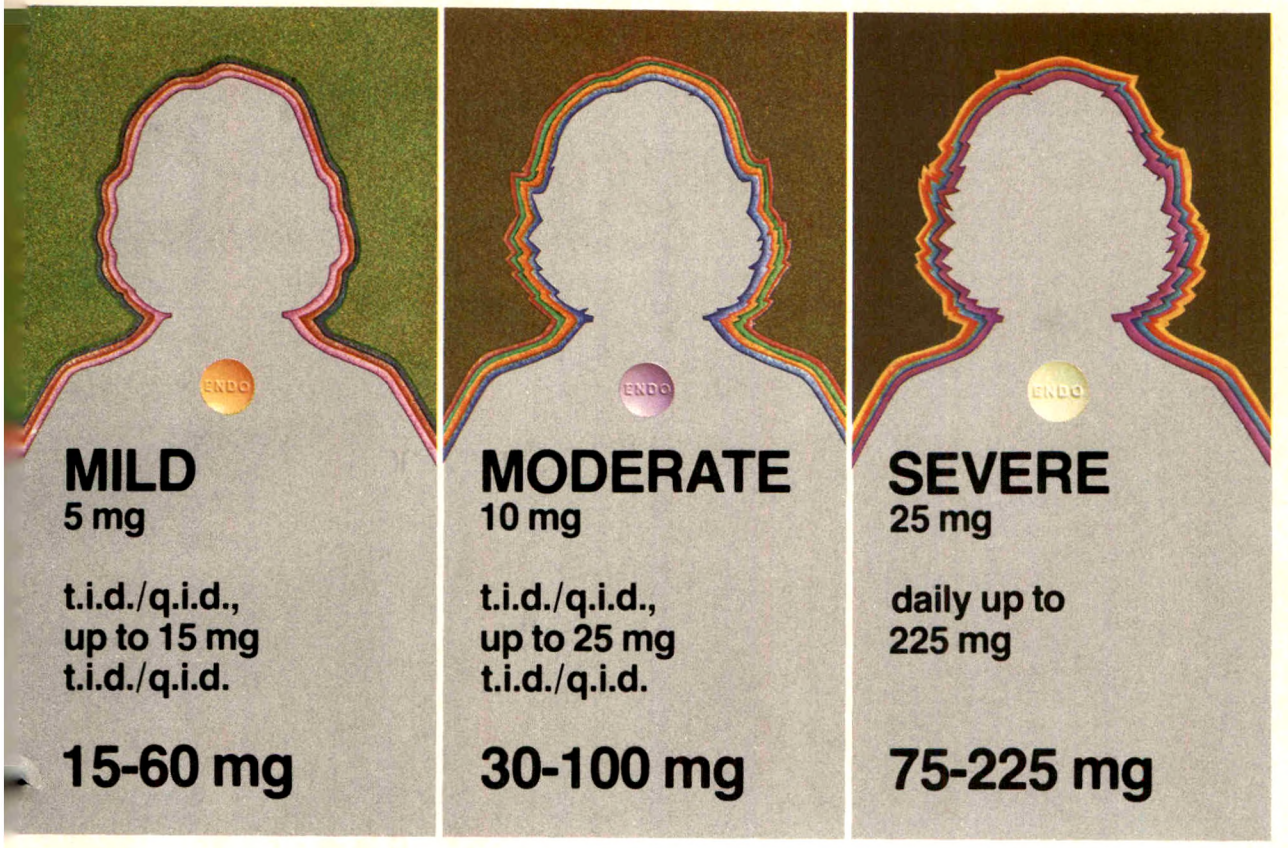


The home milieu:
an end goal of schizophrenic therapy.

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How to use MOBAN® (molindone HCl)

a wide range of dosage strength



MILD
5 mg

t.i.d./q.i.d., up to 15 mg t.i.d./q.i.d.

15-60 mg

MODERATE
10 mg

t.i.d./q.i.d., up to 25 mg t.i.d./q.i.d.

30-100 mg

SEVERE
25 mg

daily up to 225 mg

75-225 mg

Titrate for 1) therapeutic response and 2) minimal effective maintenance dose.

Use lower doses for the elderly and debilitated.

NOTE: Transient drowsiness may occur during the first 1-2 weeks.

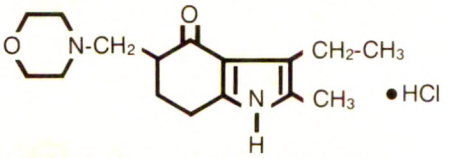
MOBAN®
(molindone HCl)

a chemically distinct treatment modality for the schizophrenias that helps to encourage greater social participation, receptivity, communication

Please see next page for full prescribing information.

MOBAN®
(molindone HCl)

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.
MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.
In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.
Human metabolic studies show MOBAN® to reach peak blood levels within one hour after oral administration.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy Studies in the pregnant patient have not been carried out. Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in these patients.

Nursing Mothers Data is not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.
Increased activity has been noted in patients receiving MOBAN®. Caution should be exercised where increased activity may be harmful.

ADVERSE REACTIONS Transient initial drowsiness was noted most frequently. Noted less frequently were Parkinsonian reactions (akinesia characterized by rigidity, immobility and reduction of voluntary movement and tremor), akathisia, restlessness, insomnia, depression, feeling of dizziness, blurred vision, hyperactivity, euphoria, dry mouth, headache, nausea and tachycardia. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.
Others which were observed included weight gain, weight loss, suicidal thinking, postural hypotension, initial heavy menses, amenorrhea, lactation, rash, spasticity, dystonia, akinesia, oculogyric crisis, gastrointestinal upset, increased libido, leukopenia, and nonspecific EKG changes.

ADVERSE REACTIONS NOTED WITH OTHER ANTIPSYCHOTIC AGENTS
The following adverse reactions have been observed with phenothiazine antipsychotic drugs. MOBAN® (molindone hydrochloride) differs chemically and to some degree pharmacologically from phenothiazines, but the possibility that similar adverse reactions may occur with MOBAN® (molindone hydrochloride) should be kept in mind.

Drowsiness Usually mild to moderate, may occur, particularly during the first or second week, after which it generally disappears. If troublesome, dosage may be lowered.

Jaundice Over-all incidence has been low, regardless of indication or dosage. Most investigators conclude it is a sensitivity reaction. Most cases occur between the second and fourth weeks of therapy. The clinical picture resembles infectious hepatitis, with laboratory features of obstructive jaundice, rather than those of parenchymal damage. It is usually promptly reversible on withdrawal of the medication; however, chronic jaundice has been reported. There is no conclusive evidence that pre-existing liver disease makes patients more susceptible to jaundice. Alcoholics with cirrhosis have been successfully treated with phenothiazine, without complications. Nevertheless, the medication should be used cautiously in patients with liver disease. Patients who have experienced jaundice with a phenothiazine should not, if possible, be re-exposed to phenothiazines.
If fever with grippe-like symptoms occurs, test for increased bilirubin or for bile in urine. If tests are positive, stop treatment.
Liver function tests in jaundice induced by the drug may mimic extrahepatic obstruction; withhold exploratory laparotomy until extrahepatic obstruction is confirmed.

Hematological Disorders Including agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura, and pancytopenia, though rare, have been reported.

Agranulocytosis Observe patients regularly for sudden appearance of sore throat or other signs of infection. If white blood counts and differential smears indicate cellular depression, stop treatment and start antibiotic and other suitable therapy.
Most cases have occurred between the 4th and 10th weeks of therapy; patients should be watched closely during that period.
Moderate suppression of white blood cells is not an indication for stopping treatment unless accompanied by the symptoms described above.

Cardiovascular: Hypotensive Effects Postural hypotension, simple tachycardia, momentary fainting and dizziness may occur after the first injection; occasionally after subsequent injections; rarely after the first oral dose. Usually recovery is spontaneous and symptoms disappear within ½ to 2 hours. Occasionally, these effects may be more severe and prolonged, producing a shock-like condition.

EKG Changes Particularly nonspecific, usually reversible Q and T wave distortions—have been observed in some patients receiving phenothiazine tranquilizers. Their relationship to myocardial damage has not been confirmed.
Note: sudden death, apparently due to cardiac arrest, has been reported, but there is not sufficient evidence to establish a relationship between such deaths and the administration of the drug.

C.N.S. Effects: Neuromuscular (Extrapyramidal) Reactions Closely resembling parkinsonism, and motor restlessness have occurred most frequently in psychiatric patients receiving high dosages. Dystonic reactions have been reported occasionally. Such symptoms usually disappear soon after dosage is lowered, the drug temporarily withdrawn, and/or concomitant administration of an antiparkinsonism agent. (Note: Levodopa has not been found effective in relief of these neuromuscular reactions.) In severe cases suitable supportive measures such as maintaining a clear airway and adequate hydration should be used. When phenothiazine is reinstituted, it should be at a lower dosage.
Hyperreflexia has been reported in the newborn.

Persistent Tardive Dyskinesia As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.
There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.
It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

Adverse Behavioral Effects Psychotic symptoms and catatonic-like states have been reported rarely.

Other C.N.S. Effects Cerebral edema has been reported.
Convulsive seizures (petit mal and grand mal) have been reported, particularly in patients with EEG abnormalities or history of such disorders.
Abnormality of the cerebrospinal fluid proteins has also been reported.

Allergic Reactions Of a mild urticarial type of photosensitivity are seen. Avoid undue exposure to sun. More severe reactions, including exfoliative dermatitis, have been reported occasionally.

Endocrine Disorders Lactation and moderate breast engorgement may occur in females on large doses. If persistent, lower dosage or withdraw drug. False positive pregnancy tests have been reported, but are less likely to occur when a serum test is used. Amenorrhea and gynecomastia have also been reported. Hyperglycemia, hypoglycemia and glycosuria have been reported.

Autonomic Reactions Occasional dry mouth; nasal congestion; constipation; adynamic ileus; urinary retention; miosis and mydriasis.

Special Considerations in Long-Term Therapy Skin pigmentation and ocular changes have occurred in some patients taking substantial doses of phenothiazines, for prolonged periods.

Skin Pigmentation Rare instances of skin pigmentation have been observed in hospitalized mental patients, primarily females who have received the drug usually for three years or more in higher dosages. The pigmentary changes, restricted to exposed areas of the body, range from an almost imperceptible darkening of the skin to a slate gray color, sometimes with a violet hue. Histological examination reveals a pigment, chiefly in the dermis, which is probably a melanin-like complex. The pigmentation may fade following discontinuance of the drug.

Ocular Changes Ocular changes have occurred more frequently than skin pigmentation and have been observed both in pigmented and nonpigmented patients receiving phenothiazines, usually for two years or more. Eye changes are characterized by deposition of fine particulate matter in the lens and cornea. In more advanced cases, star-shaped opacities have also been observed in the anterior portion of the lens. The nature of the eye deposits has not yet been determined. A small number of patients with more severe ocular changes have had some visual impairment. In addition to these corneal and lenticular changes, epithelial keratopathy and pigmentary retinopathy have been reported. Reports suggest that the eye lesions may regress after withdrawal of the drug.
Since the occurrence of eye changes seems to be related to dosage levels and/or duration of therapy, it is suggested that long-term patients on moderate to high dosage levels have periodic ocular examinations.

Etiology The etiology of both of these reactions is not clear, but exposure to light, along with dosage/duration of therapy, appears to be the most significant factor. If either of these reactions is observed, the physician should weigh the benefits of continued therapy against the possible risks and, on the merits of the individual case, determine whether or not to continue present therapy, lower the dosage, or withdraw the drug.

Other Adverse Reactions Mild fever may occur after large I.M. doses. Hyperpyrexia has been reported. Increases in appetite and weight sometimes occur. Peripheral edema and a systemic lupus erythematosus-like syndrome have been reported.
Note: There have been occasional reports of sudden death in patients receiving phenothiazines. In some cases, the cause appeared to be asphyxia due to failure of the cough reflex. In others, the cause could not be determined. There is not sufficient evidence to establish a relationship between such deaths and the administration of phenothiazines.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized, and the minimal effective dose should be employed. Elderly and debilitated patients should be started on lower dosage.

Dosage schedule, based on severity of symptomatology
1. Mild—5 mg three or four times a day; an increase to 15 mg three or four times a day may be required.
2. Moderate—10 mg three or four times a day; an increase to 25 mg three or four times a day may be required.
3. Severe—daily dosage as high as 225 mg may be required.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.
Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.
Since the absorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.
Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN® in animals, this blocking effect has not been determined in humans.
A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would **not** be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN® in feces is less than 1%.
Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents, (i.e., Artane*, Cogentin*, Akineton*).

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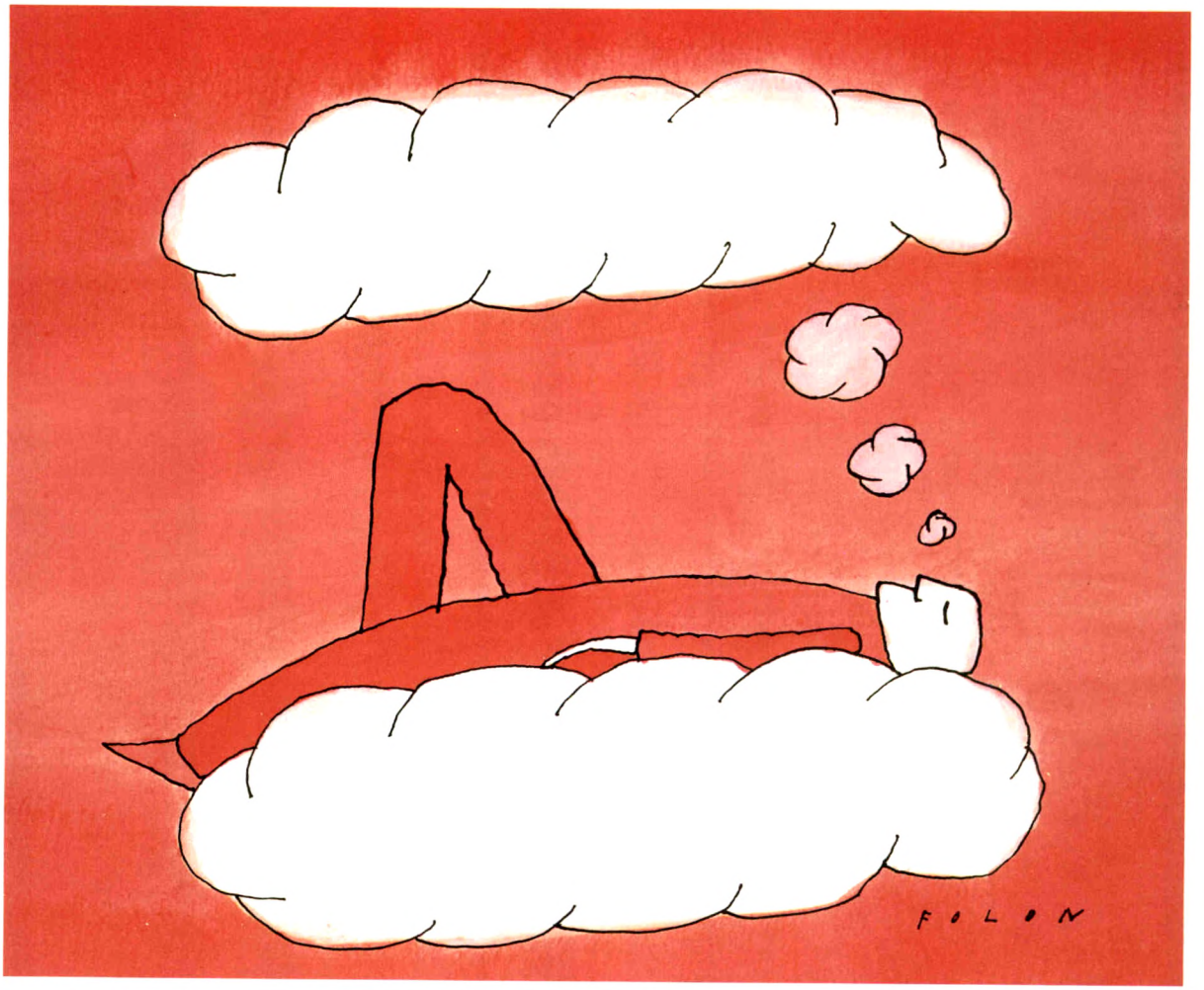
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INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F Co.) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968, pp 1–12

Tables and figures. Titles and headings of any tables and figures should be sufficiently clear that the meaning of the data is understandable without reference to the text. See recent issues of the *Journal* for the general requirements of style.

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Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg, of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

More detailed professional information available on request.

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Valium® (diazepam) Close-Up

Product information for psychiatrists



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cardiac glycosides
diuretics
vasodilators
anticholinergics

Psychotropics/CNS depressants

alcohol
phenothiazines
narcotics
barbiturates
hypnotics
MAO inhibitors
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Anticonvulsants

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you should know this about Valium® (diazepam)

Valium is frequently used with most classes of these primary medications. Clinically significant drug interactions have not been reported to date.

Potentiation may occur with other CNS depressants. Careful consideration should be given to the pharmacology of these compounds when used concomitantly with Valium.

As with other agents which have anticonvulsant activity, the possibility of increase in frequency and/or severity of grand mal seizures may require an increase in dosage of standard anticonvulsants.

Valium® (diazepam) ℞

2-mg, 5-mg, 10-mg scored tablets

Before prescribing, please see following page for a summary of product information

Valium® (diazepam)

2-mg, 5-mg, 10-mg scored tablets

can relieve psychoneurotic anxiety and its associated depressive symptoms

Prompt, effective action. Valium works rapidly to relieve pronounced psychic tension in patients overreacting to stress and in psychoneurotic patients.

Wide margin of safety. Valium is generally well tolerated and in usual dosages rarely produces significant adverse reactions.

Dosage flexibility. Scored Valium 2-, 5-, 10-mg tablets give you dosage flexibility no tranquilizer capsule can match.

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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

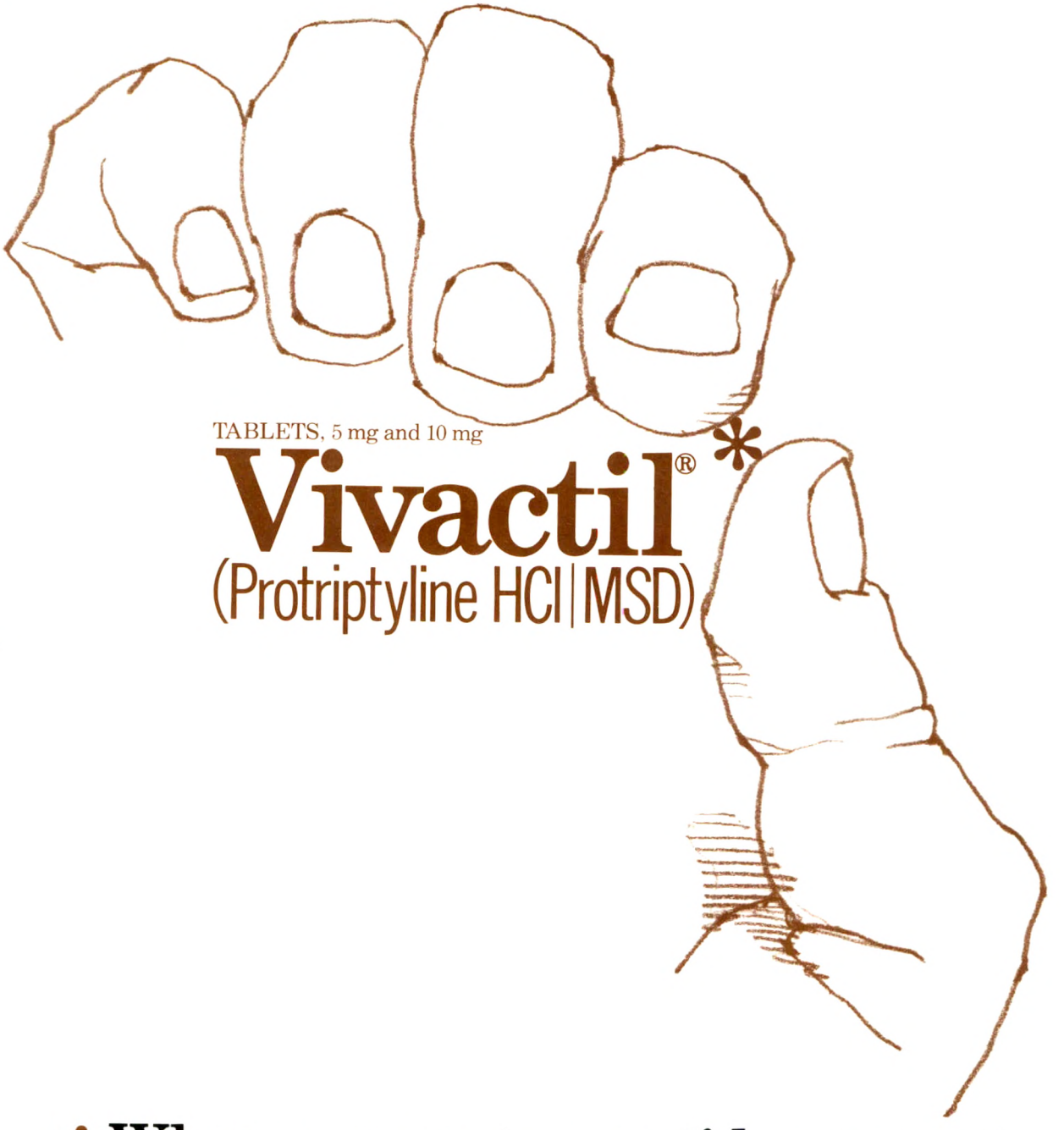
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

ROCHE

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Nutley, New Jersey 07110

In clinically significant depression



TABLETS, 5 mg and 10 mg

Vivactil®*

(Protriptyline HCl | MSD)

***When you want an antidepressant that is characteristically nonsedating for the patient under close medical supervision (Symptoms such as anxiety or agitation may be aggravated.)**

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information please see following page.

In clinically significant depression

TABLETS, 5 mg and 10 mg

Vivactil®

(Protriptyline HCl | MSD)

Contraindications: Known hypersensitivity; acute recovery phase following myocardial infarction. Should not be given concomitantly with an MAOI; hyperpyretic crises, severe convulsions, and deaths have occurred in patients receiving tricyclic antidepressant and MAOI drugs simultaneously. When it is desired to substitute protriptyline HCl for an MAOI, a minimum of 14 days should be allowed to elapse after the latter is discontinued. Protriptyline HCl should then be initiated cautiously with gradual increase in dosage until optimum response is achieved.

Warnings: May block the antihypertensive effect of guanethidine or similarly acting compounds. May impair mental and/or physical abilities required for the performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Should be used with caution in patients with a history of seizures and, because of its autonomic activity, in patients with a tendency to urinary retention or increased intraocular tension.

Tachycardia and postural hypotension may occur more frequently than with other antidepressant drugs. Should be used with caution in elderly patients and patients with cardiovascular disorders; such patients should be observed closely because of the tendency of the drug to produce tachycardia, hypotension, arrhythmias, and prolongation of the conduction time. Myocardial infarction and stroke have occurred with drugs of this class. On rare occasions, hyperthyroid patients or those receiving thyroid medication may develop arrhythmias when this drug is given.

Usage in Children: Not recommended for use in children because safety and effectiveness in the pediatric age group have not been established.

Usage in Pregnancy: Safe use in pregnancy and lactation has not been established; therefore, use in pregnant women, nursing mothers, or women who may become pregnant requires that possible benefits be weighed against possible hazards to mother and child.

Precautions: When protriptyline HCl is used to treat the depressive component of schizophrenia, psychotic symptoms may be aggravated; likewise, in manic-depressive psychosis, depressed patients may experience a shift toward the manic phase; paranoid delusions, with or without associated hostility, may be exaggerated. In any of these circumstances, it may be advisable to reduce the dose of protriptyline HCl or to use a major tranquilizing drug concurrently. Symptoms, such as anxiety or agitation, may be aggravated in overactive or agitated patients.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. May enhance response to alcohol and effects of barbiturates and other CNS depressants. Possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent administration with electroshock therapy may increase hazards of therapy; such treatment should be limited to patients for whom it is essential. Discontinue drug several days before elective surgery, if possible. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions which have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when protriptyline HCl is administered. Protriptyline HCl is more likely to aggravate agitation and anxiety and produce cardiovascular reactions such as tachycardia and hypotension.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions, anxiety, restlessness, agitation; insomnia, panic, and nightmares; hypomania; exacerbation of psychosis.

Neurological: numbness, tingling, and paresthesias of extremities; incoordination, ataxia, tremors, peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns, tinnitus.

Anticholinergic: dry mouth and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of the urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (general, or of face and tongue), drug fever.

Hematologic: bone marrow depression; agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of other tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

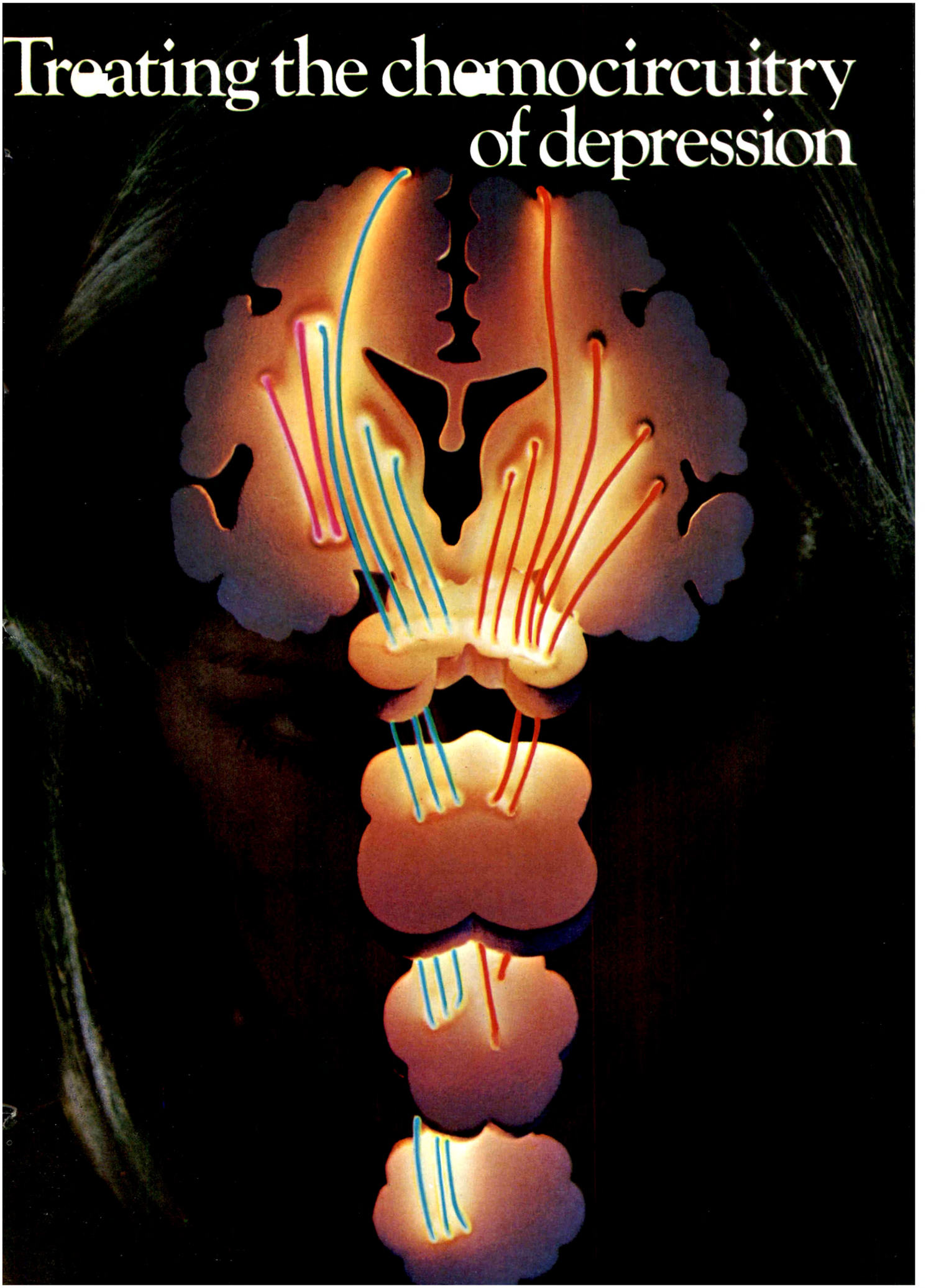
How Supplied: Tablets, containing 5 mg and 10 mg protriptyline HCl each, in single-unit packages of 100 and bottles of 100 and 1000.

For more detailed information, consult your MSD representative or see full prescribing information.

Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

MSD
MERCK SHARP & DOHME

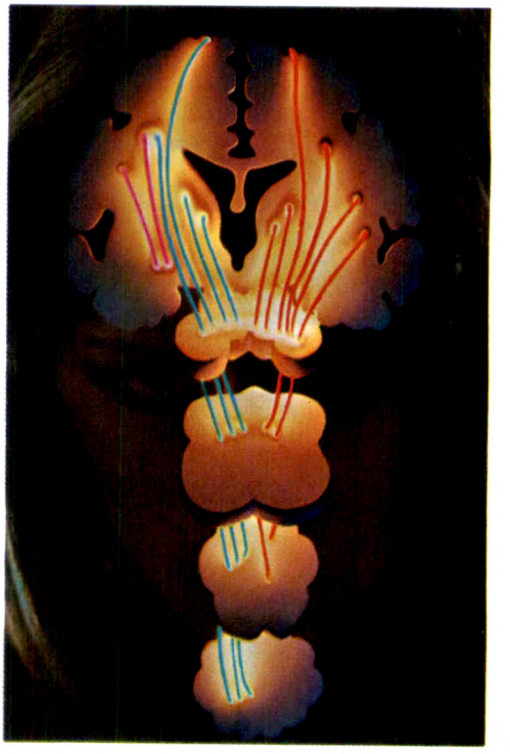
Treating the chemocircuitry of depression



Depression as an organic disease

At one time, depression was thought to be predominantly a behavioristic, nonmetabolic abnormality. But recent work on CNS catecholamine metabolism strongly suggests that biochemical factors are involved.

Theory of biogenic etiology



□ In individual nerve systems originating in the brainstem, impulses are relayed within each network by chemically distinct compounds—the biogenic amines.[1]

□ These compounds "... serve as neurotransmitters in brain sites that probably modulate feeling states, and other mental functions, that are disturbed in psychiatric disease."[2]

□ Amine systems involved in affective disorders[3] are:
norepinephrine (catecholamine)
dopamine (catecholamine)
serotonin (indoleamine)

There is a school of thought that associates catecholamines and affective disorders. According to this hypothesis, "... some, if not all, depressions are associated with an absolute or relative deficiency of catecholamines, particularly norepinephrine, at functionally important adrenergic receptor sites in the brain."[4]

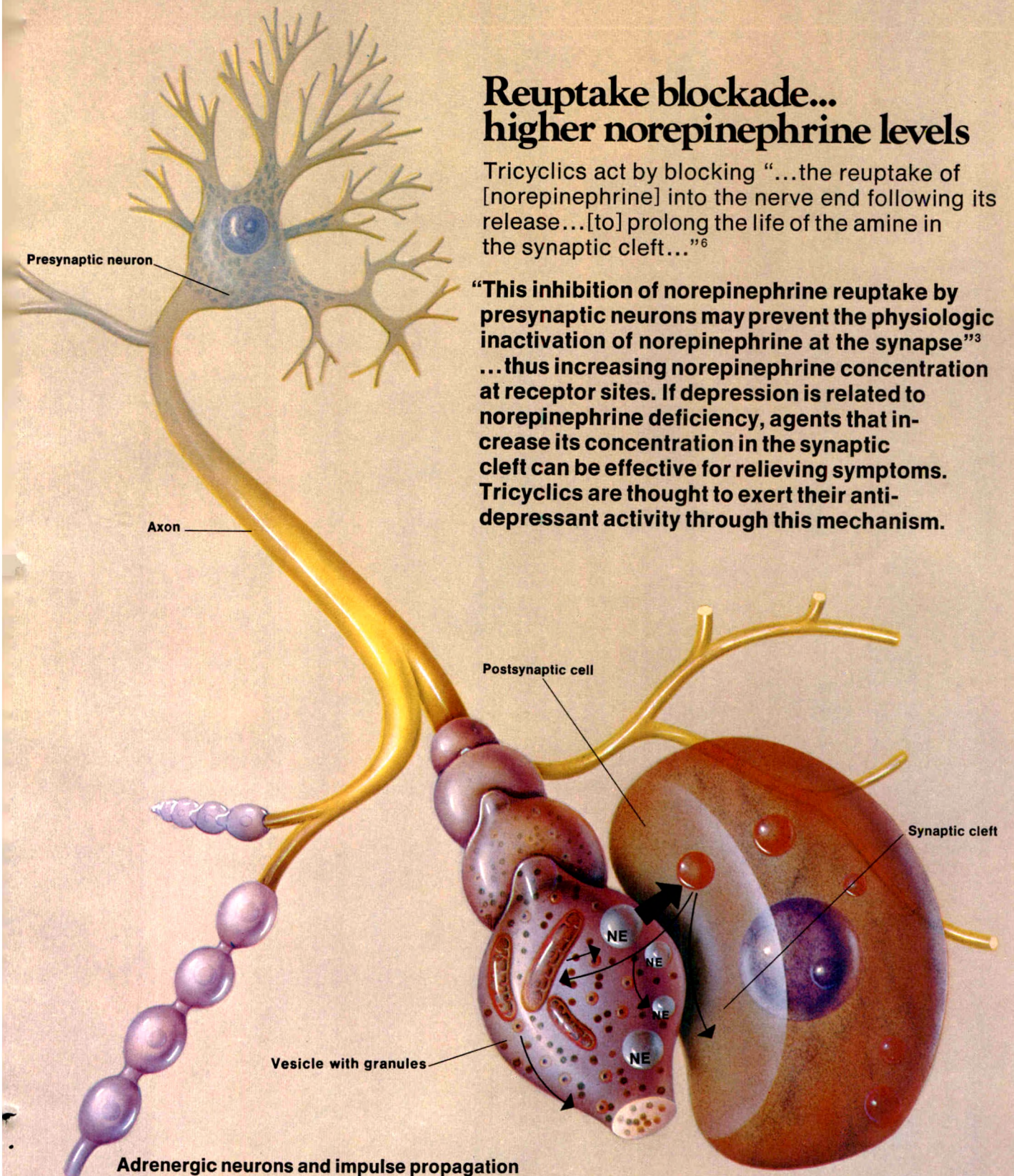
"The possibility that depressed patients may have a functional deficiency of brain norepinephrine has gained considerable currency over the past decade."[5]

..and the efficacy of tricyclics

Reuptake blockade... higher norepinephrine levels

Tricyclics act by blocking "...the reuptake of [norepinephrine] into the nerve end following its release...[to] prolong the life of the amine in the synaptic cleft..."[6]

"This inhibition of norepinephrine reuptake by presynaptic neurons may prevent the physiologic inactivation of norepinephrine at the synapse"[3] ...thus increasing norepinephrine concentration at receptor sites. If depression is related to norepinephrine deficiency, agents that increase its concentration in the synaptic cleft can be effective for relieving symptoms. Tricyclics are thought to exert their antidepressant activity through this mechanism.



Adrenergic neurons and impulse propagation

□ Information coded as electric impulses arrives at synapse via axons of presynaptic neurons.

□ Impulses cause "release" of norepinephrine stored in vesicles adjacent to the presynaptic membrane.

□ Neurotransmitter migrates across the synaptic cleft and binds to receptors on postsynaptic cell membrane; it either triggers a new impulse or inhibits activity in the postsynaptic cell.

□ Because of the complex nature of brain activity, this action must be terminated rapidly and the cleft primarily cleared through reuptake of the transmitter into the presynaptic cell.

Tricyclics...the right choice in depression

Circuit functions and pharmacologic effects

Functioning of the biogenic amine systems, and how they are affected by therapeutic agents, underscore the value of tricyclics in depression.

The serotonin system: sedation/sleep



☐ Thought to regulate aspects of the sleep-wakefulness cycle and may be involved in REM sleep or dreaming.[2]
☐ Deficiency in turnover can produce symptomatology also present in depression.
☐ Some tricyclics, however, principally block the reuptake inactivation of serotonin.[6]
☐ **Activation of this system may therefore produce unwanted sedation.**

The dopamine system: motor activity



☐ Associated with normal posture and regulation of motor activity.
☐ Disturbed dopamine levels linked to symptoms of Parkinson's disease.
☐ "A possible relationship between decreased baseline... levels [of a dopamine metabolite] and decreases in motor activity in depressed patients has been suggested by some investigators..."[7]
☐ **Agents that influence this system can thus affect motor activity[6] and produce unwanted side effects.**

The norepinephrine system: mood



☐ Plays a major role in affective states.
☐ Deficiencies of norepinephrine linked to depression.
☐ Norepinephrine levels may be raised by psychomotor stimulants which cause release and block reuptake... by MAO inhibitors which block intraneuronal metabolism ...and by tricyclics through reuptake blockade.[1]
☐ Of these agents, the "most useful are the tricyclics."[6] They are preferred for their greater safety[8] and lower incidence of side effects.[9]
☐ **But tricyclics vary in their degree of interaction with other circuits—not only the catecholamines, but serotonin and acetylcholine as well.[10]**

Pertofrane®...the right choice in tricyclics
(desipramine hydrochloride NF)

Consider the chemistry

..qualitative and quantitative changes in the biochemical, pharmacological and clinical spectrum...[were produced by] the elimination of one of the N-methyl groups in the terminal amino group of imipramine to yield desipramine."[11]

..monomethylated forms of tricyclic antidepressants...are generally much more potent inhibitors of catecholamine re-uptake than dimethylated forms ..[whereas] dimethylated tricyclic antidepressants are more potent anticholinergics than the monomethylated forms."[12]

Dimethylated drugs are more potent serotonin blockers than monomethylated analogs. "Imipramine slows the turnover rate of [serotonin], an effect not shared by desipramine..."[13]

...compared to imipramine, [desipramine] is more 'procatechol' than 'proindole.' "[6]

...and the clinical implications

Rapid onset of action

Pertofrane has been found in some studies[14,16,19] to have a more rapid onset of action than imipramine. This relatively rapid onset of action may bring about prompt therapeutic response.

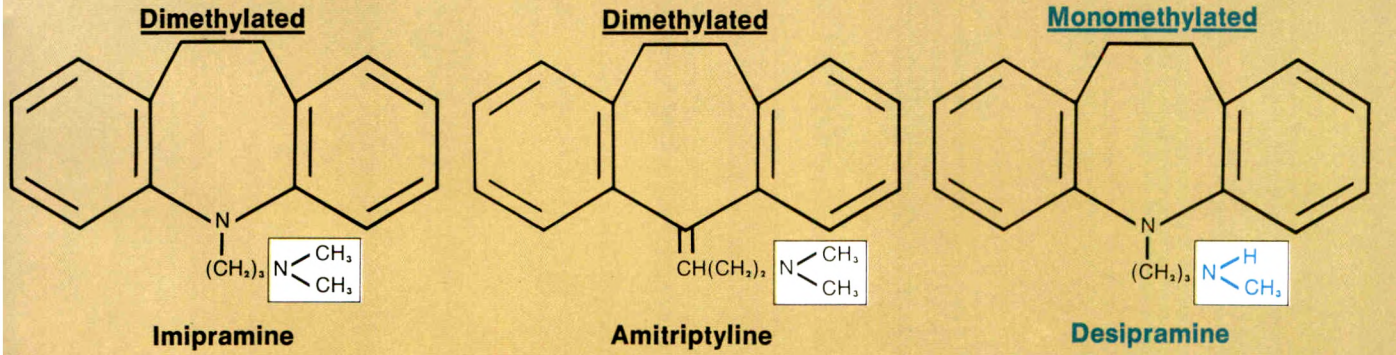
In a recent double-blind study comparing desipramine and amitriptyline, "the activity of desipramine becomes manifest rapidly and its beneficial effects further improve over time."[15]

Less sedative effect

Desipramine HCl shows good to excellent antidepressant effect yet is only mid-range in sedation.[6]

In a recent double-blind study comparing desipramine and amitriptyline in outpatients with depressive neurosis, while desipramine was equal or superior to amitriptyline in improving anxiety/depression and sleep disturbance factors, amitriptyline produced sedation in significantly more patients than did desipramine.[15]

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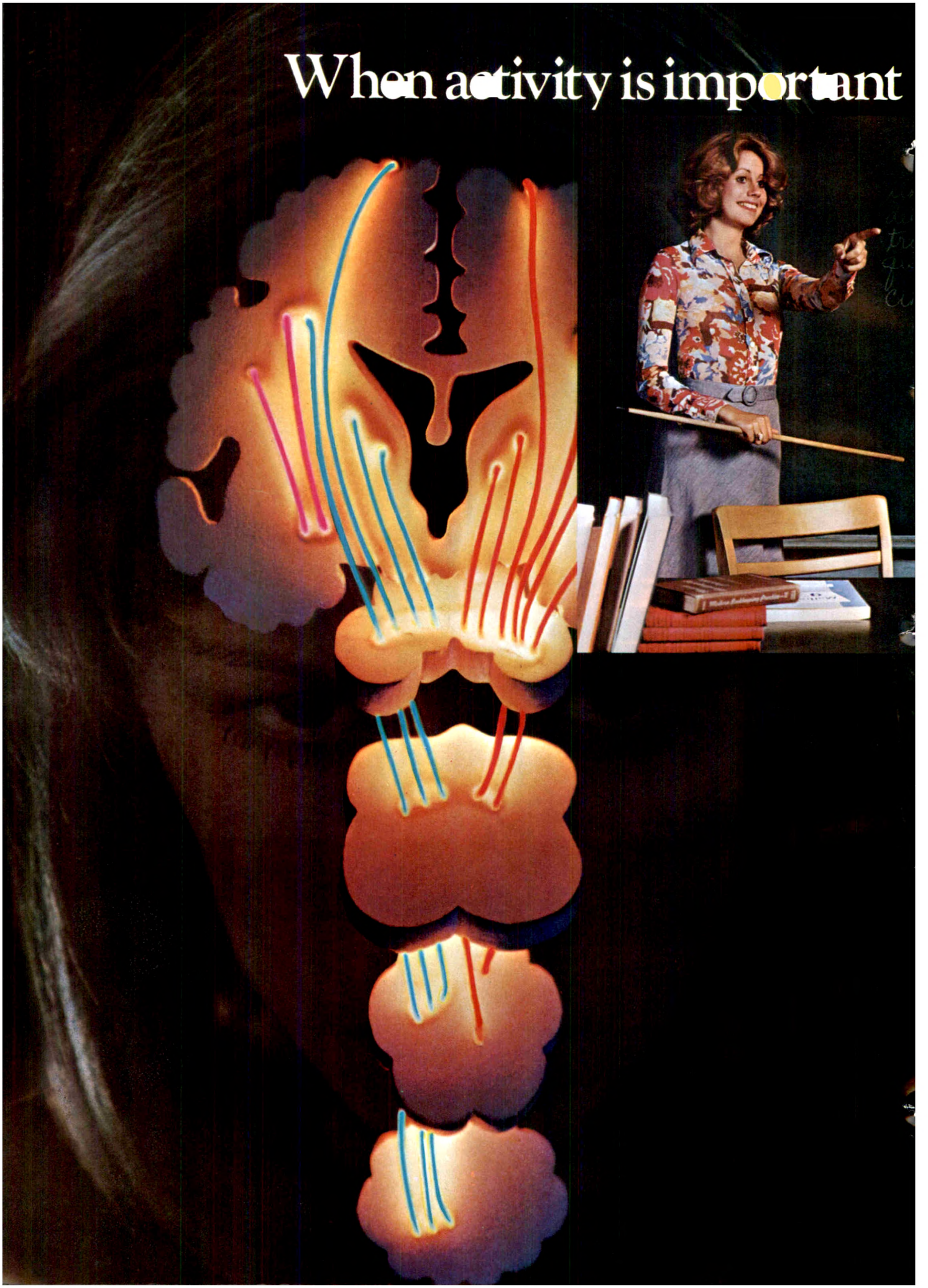


Imipramine **Amitriptyline** **Desipramine**

Selection of the right tricyclic for a particular patient can be based on structural activity relationships. And the profile of activity for a particular agent frequently depends on whether it is dimethylated or monomethylated.

Please see back cover for full prescribing information.

When activity is important



the neurochemistry's right for

Pertofrane®
(desipramine hydrochloride NF)

Proven clinical efficacy

☐ "...seems ideally suited to use in an active ambulatory patient population...[because of] its significant improvement of the depressed mood, work and activities..."[15]

☐ Effectiveness well documented in over a decade of clinical use.[9,16–18]

Prompt onset of action

☐ Relief may begin in 2 to 5 days—but full therapeutic effect is seldom seen before 2 weeks.

☐ Rapid onset of action[11,14,15,16,19] is particularly useful in patients who must continue to function actively during the course of therapy.

Mid-range sedation[6]

☐ Relieves associated mild anxiety/agitation, yet is only mid-range in sedative effect.

☐ "There is no problem of drowsiness"[16]—especially important when normal levels of daily activity must be maintained during treatment.

Compatible with tranquilizers/sedatives

☐ Where necessary, may be used with antianxiety or hypnotic agents.[16]

☐ Permits individualization of therapy when mild anxiety or agitation accompanies depression.

Provides dosage flexibility

☐ The therapeutic range is from 25 to 200 mg. daily. Patients may start at 25 mg. t.i.d. and dosage may be increased up to 200 mg./day according to patient needs.

Please see back cover for full prescribing information.

Pertofrane® (desipramine hydrochloride NF)

when activity is important

INDICATIONS: PERTOFRANE is indicated for the relief of mental depression (see Note before How Supplied section for additional information on depressive symptomatologies often responsive to this drug).

CONTRAINDICATIONS: The use of PERTOFRANE concomitantly or within two weeks of the administration of MAO inhibitors, is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse reactions can be serious, or even fatal. When it is desired to substitute PERTOFRANE in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation allows, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed.

The drug is contraindicated following recent myocardial infarction.

Patients with a known hypersensitivity to tricyclic antidepressants should not be given PERTOFRANE.

WARNINGS: As with other potent antidepressants, an activation of the psychosis may occasionally be observed in schizophrenic patients.

Due to the drug's atropine-like effects and sympathomimetic potentiation, PERTOFRANE should be used only with the greatest care in patients with narrow-angle glaucoma and with urethral or ureteral spasm.

Likewise, only when patient need outweighs the risk of serious adverse reactions should the drug be used in the presence of any of the following conditions: severe coronary heart disease with EKG abnormalities, progressive heart failure, angina pectoris, paroxysmal tachycardia and active seizure disorder (this agent has been shown to lower the seizure threshold).

In some instances, desipramine and the parent compound, imipramine, have been shown to block the pharmacologic action of the antihypertensive, guanethidine, and related adrenergic neuron-blocking agents.

Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride therapy.

The concurrent use of other central nervous system drugs or alcohol may potentiate the adverse effects of desipramine hydrochloride. Since many such drugs may be used during surgery, it is recommended that desipramine hydrochloride be discontinued for as long as the clinical situation will allow prior to elective surgery.

Patients should be cautioned about the possibility of impaired ability to operate a motor vehicle or other dangerous machinery.

Usage in Pregnancy: Although teratogenic studies in mice, rats and rabbits have revealed no adverse effects, safe use of this drug in women who are or may become pregnant has not been definitely established. Therefore, PERTOFRANE should be withheld from these women unless the clinical situation warrants the potential risk.

In view of the lack of experience in children, the drug is not recommended for use in patients under twelve years of age.

Elderly and adolescent patients can usually be managed on lower dosage than that recommended for other patients and may not tolerate higher doses as well because of an increased incidence of adverse reactions.

PRECAUTIONS: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until remission occurs. These patients require careful supervision and protective measures during therapy.

Anxiety and increased agitation have been reported, particularly where depression is not the primary disorder. A shift to hypomanic or manic excitement may occur during therapy. Such reactions may necessitate discontinuation of the drug.

Although the anticholinergic activity of the drug is weak, in susceptible patients and in those receiving anticholinergic drugs (including antiparkinsonism agents), atropine-like effects may be more pronounced (e.g. paralytic ileus).

Caution should be observed in prescribing the drug in hyperthyroid patients and in those receiving thyroid medications. Transient cardiac arrhythmias have occurred in rare instances.

In all patients undergoing extended courses of therapy, periodic blood and liver studies for signs of toxicity should supplement careful clinical observations.

ADVERSE REACTIONS: PERTOFRANE (desipramine hydrochloride) is well tolerated by most patients and severe complications or adverse reactions are infrequent. Those most often reported cause little discomfort and seldom require discontinuation of the drug.

Nervous System: Dizziness, drowsiness, insomnia, headache and disturbed visual accommodation have been reported with the drug. In addition, tremor, unsteadiness, tinnitus and paresthesias have been reported. Occasionally, mild extrapyramidal activity, falling and neuromuscular incoordination have been seen. A confusional state (with such symptoms as hallucinations and disorientation) may be produced, particularly in older patients and at higher dosage, and may require discontinuation of the drug. Changes in EEG patterns have been observed. Epileptiform seizures may occur. A reduction in dosage may help control some of these adverse reactions.

Gastrointestinal Tract: Anorexia, dryness of the mouth, nausea, epigastric distress, constipation and diarrhea have been reported in patients receiving PERTOFRANE.

Skin: Skin rashes (including photosensitization), perspiration and flushing sensations have been reported during therapy.

Liver: Transient jaundice, apparently of an obstructive nature, and liver damage have been observed in rare cases. Elevation in transaminase and changes in alkaline phosphatase have occurred and should indicate repeated liver-function profiles. If progressive elevation occurs, the drug should be discontinued.

Blood Elements: Bone-marrow depression, agranulocytosis, thrombocytopenia and purpura have been reported. If these occur, the drug should be discontinued. Transient eosinophilia has been observed in some instances.

Cardiovascular System: Orthostatic hypotension and tachycardia have been observed but seldom require discontinuation of treatment. Patients who require concomitant vasodilating therapy should be carefully supervised, particularly during the initial phas

Genitourinary System: Urinary frequency or retention and impotence have been repor

Endocrine System: Occasional hormonal effects, including gynecomastia, galactorrhe and breast enlargement have been reported with PERTOFRANE (desipramine hydrochloride). In addition, decreased libido and estrogenic effect have been noted. T exact relationship of these adverse effects to the administration of the drug has not be established.

Sensitivity: Urticaria and rare instances of drug fever and cross-sensitivity with imipramine have been observed.

DOSAGE AND ADMINISTRATION: *All Patients Except Geriatric and Adolescent:* Optimal results are obtained at a dosage of approximately 150 mg./day. PERTOFRAN may be administered as two 25 mg. capsules or one 50 mg. capsule t.i.d. (150 mg. da When necessary, dosage may be increased, after initiation of therapy, up to 200 mg. o in divided doses. Continued therapy at the optimal dosage level should be maintained during the active phase of the depression. It is recommended that a lower maintenanc dosage be continued for at least two months after a satisfactory response has been achieved.

In cases of relapse due to premature withdrawal of the drug, a prompt response may b obtained by immediate resumption of treatment.

Geriatric and Adolescent Patients: Elderly and adolescent patients can usually be managed on lower dosage and may not tolerate higher doses as well as other patients Therapy in these age groups may be initiated with 25 to 50 mg. daily. Dosage may be increased according to response and tolerance, but it is generally unnecessary to exce 100 mg. daily in these patients. It is recommended that a lower maintenance dosage b continued for at least two months after a satisfactory response has been achieved.

Mild anxiety and agitation which often accompany cases of depression usually remit as the depression responds to PERTOFRANE. Occasionally, however, when anxiety an agitation are more serious, the adjunctive use of a sedative or tranquilizing agent may b indicated.

NOTE: Depressions which are most responsive to PERTOFRANE may be described i terms of "target symptoms." These include: despondency, sadness and depressed mood; fatigue; lack of interest and emotional response; helplessness, hopelessness, pessimism and despair; feelings of incapacity and inferiority; psychomotor retardation and inhibition; delusions of guilt and unworthiness; psychosomatic complaints; insomni anorexia and weight loss; depression-related anxiety and agitation; suicidal drive.

It should be borne in mind that these "target symptoms" of depression assume their proper significance only after careful history, physical and mental evaluations and other investigative procedures have confirmed a diagnosis of one of the several types of depression.

HOW SUPPLIED: 25 mg. Capsules (pink), bottles of 100 and 1000 (NSN 6505-00-913 0382A). Also, 50 mg. Capsules (maroon and pink), bottles of 100 and 1000; single-dose blister packs, boxes of 500.

References: 1. Axelrod, J.: Biogenic amines and their impact in psychiatry, Semin. Psychiatry 4:199, 1972. 2. Friedhoff, A.: The biogenic amines: Brain biochemistry, in Mandates Depression (Rome, H. P., ed.), New York, Medcom, 1974. 3. Schildkraut, J. J.: Update: Biochemical pathways of depression, in Mandates Depression (Mead, B. T., ed.), New York, Medcom, 1975, pp. 8,10. 4. Schildkraut, J. J.: Norepinephrine metabolism in subtypes of depressive disorders, Psychopharmacol. Bull. 10:49, 1974 5. Maas, J. W. et al.: Catecholamine metabolism, depressive illness, and drug response, Arch. Gen. Psychiatry 26:252, 1972. 6. Prange, A. J., Jr.: The use of drugs in depression: Its theoretical and practical basis, Psychiatr. Ann. 3:56, 1973. 7. Schildkraut, J. J.: Biogenic amines and affective disorders, Ann. Rev. Med. 25:333, 1974. 8. Hussar, D. A.: The drug therapy of depression, Therapeutics 2:3, 1974. 9. Krakowski, A. J.: Treatment of severe depression with desipramine, a metabolite of imipramine, Am. J. Psychiatry 120:494, 1963. 10. Lipton, M. A.: Summary and overview, Psychopharmacol. Bull. 10:57, 1974. 11. Biel, J. H.: Non-monamine oxidase inhibitor antidepressants structure-activity relationships, in Principles of Psychopharmacolog (Clark, W. G. and del Giudice, J., eds.), New York, Academic Press, 1970, p. 272. 12. Ayd, F. J., Jr.: Int. Drug Ther. Newsletter 10:21, June 1975. 13. Byck, R.: Drugs and the treatment of psychiatric disorders, in The Pharmacological Basis of Therapeutics (Goodman, L. S. and Gilman, A., eds.), ed. 5, New York, Macmillan, 1975, p. 176. 14. Lapolla, A. and Jones, H.: Placebo-control evaluation of desipramine in depression, Am. J. Psychiatry 127:335, 1970. 15. Barnes, R. J.: Clinical Depression, USV, 1975. 16. Barringer, T. J.: Desipramine (Pertofrane) in the treatment of depression. Psychosomatics 6:326, 1965. 17. Cammer, L.: Rx for depression, in Depression: A Practical Approach (Davis, J. M., ed.), New York, Medcom, 1974, p. 26. 18. Lapolla, A.: Clinical trial of a new antidepressant (desipramine hydrochloride) in a hospital setting, Am. J. Psychiatry 121:1206, 1965. 19. Lafave, H. G. et al.: Desipramine and imipramine in an outpatient setting: A comparative study, Am. J. Psychiatry 122:698, 1965.

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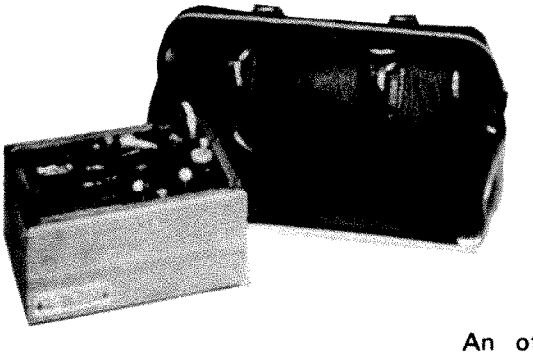
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An officially approved, extremely durable instrument which has earned world-wide professional acceptance, the MOL-AC II is economically priced with a fine, genuine leather physician's bag included.

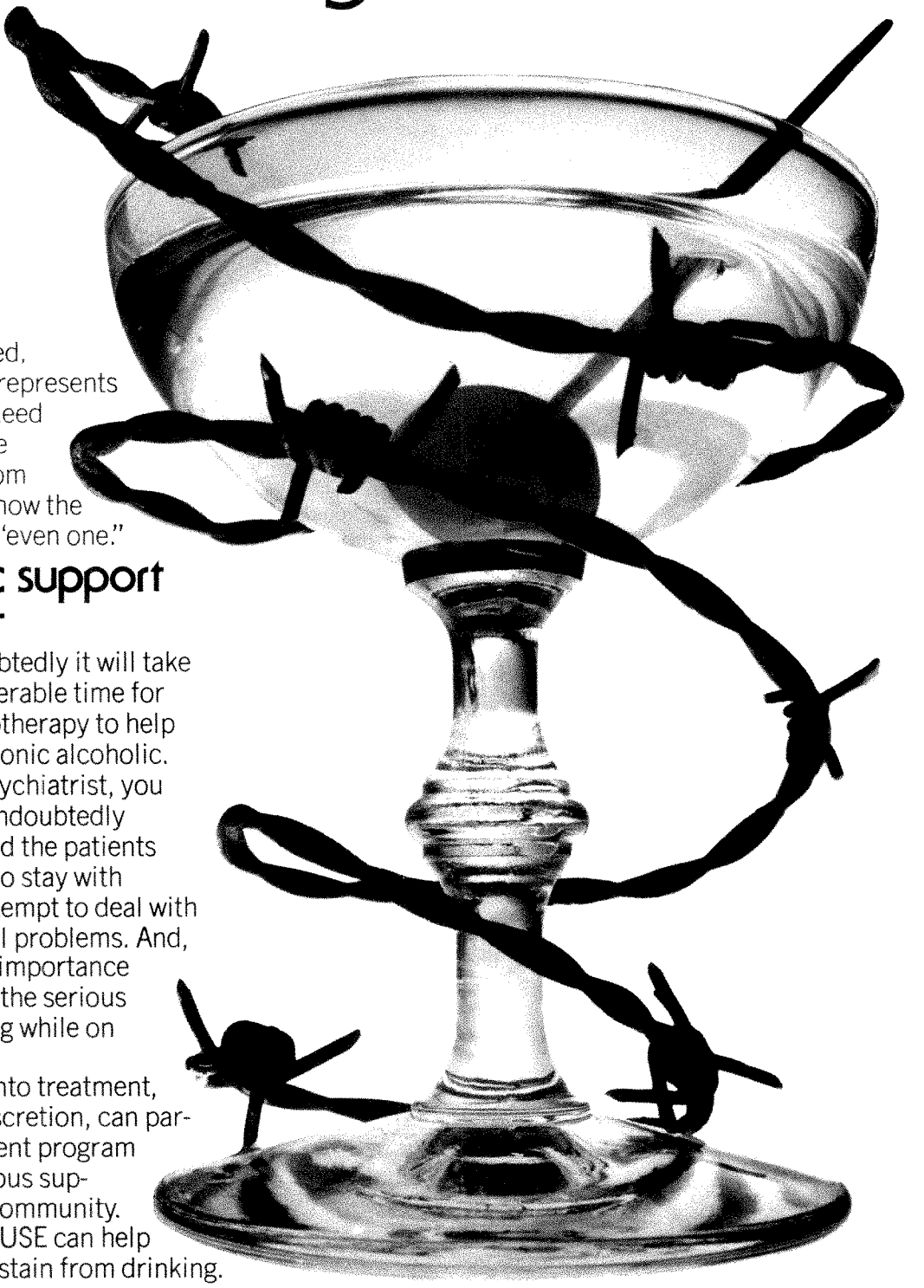
Other Models Still Available: The CW47, original therapeutic cerebral stimulator with only 3 of the 9 treatment current selections available on Model SOS.

For more detailed information, and bibliography of over 200 references, write to:

REUBEN REITER, Sc.D., INC. 187 Sylvan Ave., Leonia, N.J. 07605 — Tel. (201) 944-1211

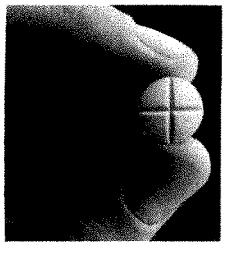
See us at Booth F5 at the Annual Meeting

ANTABUSE: "Social security" for the alcoholic who wants to stop drinking...



The temptation is resisted, ANTABUSE (disulfiram) represents the security alcoholics need in any situation. They are better able to abstain from drinking because they know the consequences of taking "even one."

until psychiatric support can do the rest



Undoubtedly it will take considerable time for psychotherapy to help the chronic alcoholic. As a psychiatrist, you have undoubtedly selected the patients who are best motivated to stay with ANTABUSE while you attempt to deal with the underlying emotional problems. And, of course, you know the importance of reminding patients of the serious consequences of drinking while on ANTABUSE.

Once sober, and well into treatment, the alcoholic, at your discretion, can participate in a total treatment program which makes use of various supportive resources in the community. In the meantime, ANTABUSE can help the chronic alcoholic abstain from drinking.

BRIEF SUMMARY
(For full prescribing information, see package circular.)

ANTABUSE® (disulfiram) in Alcoholism

INDICATION: ANTABUSE is an aid in the management of selected chronic alcoholic patients who want to remain in a state of enforced sobriety so that supportive and psychotherapeutic treatment may be applied to best advantage. (Used alone, without proper motivation and without supportive therapy, ANTABUSE is not a cure for alcoholism, and it is unlikely that it will have more than a brief effect on the drinking pattern of the chronic alcoholic.)

CONTRAINDICATIONS: Patients who are receiving or have recently received metronidazole, paraldehyde, alcohol, or alcohol-containing preparations, e.g. cough syrups, tonics, and the like, should not be given ANTABUSE.

ANTABUSE is contraindicated in the presence of severe myocardial disease or coronary occlusion, psychoses, or hypersensitivity.

WARNINGS: ANTABUSE should never be administered to a patient when he is in a state of alcohol intoxication or without his full knowledge.

The physician should instruct relatives accordingly.

The patient must be fully informed of the ANTABUSE-alcohol reaction. He must be strongly cautioned against surreptitious drinking while taking the drug, and he must be fully aware of possible consequences. He should be warned to avoid alcohol in disguised form, *i.e.* in sauces, vinegars, cough mixtures, and even aftershave lotions and back rubs. He should also be warned that reactions may occur with alcohol up to 14 days after ingesting ANTABUSE.

THE ANTABUSE-ALCOHOL REACTION: ANTABUSE plus alcohol, even small amounts, produces flushing, throbbing in head and neck, throbbing headache, respiratory difficulty, nausea, copious vomiting, sweating, thirst, chest pain, palpitation, dyspnea, hyperventilation, tachycardia, hypotension, syncope, marked uneasiness, weakness, vertigo, blurred vision, and confusion. In severe reactions there may be respiratory depression, cardiovascular collapse, arrhythmias, myocardial infarction, acute congestive heart failure, unconsciousness, convulsions, and death.

The intensity of the reaction varies with each individual, but is generally proportional to the amounts of ANTABUSE (disulfiram) and alcohol ingested. Mild reactions may occur in the sensitive individual when the blood alcohol concentration is increased to as little as 5 to 10 mg. per 100 cc. Symptoms are fully developed at 50 mg. per 100 cc., and unconsciousness usually results when the blood alcohol level reaches 125 to 150 mg.

The duration of the reaction varies from 30 to 60 minutes to several hours in the more severe cases, or as long as there is alcohol in the blood.

DRUG INTERACTIONS: Disulfiram appears to decrease the rate at which certain drugs are metabolized and so may increase the blood levels and the possibility of clinical toxicity of drugs given concomitantly.

Disulfiram should be used with caution in those patients receiving diphenylhydantoin and its congeners, since toxic levels of these antiepileptic agents have been reported during concomitant disulfiram therapy.

It may be necessary to adjust the dosage of oral anticoagulants upon beginning or stopping disulfiram, since disulfiram may prolong prothrombin time.

Patients taking isoniazid when disulfiram is given should be observed for the appearance of unsteady gait or marked changes in mental status and the disulfiram discontinued if such signs appear.

CONCOMITANT CONDITIONS: Because of the possibility of an accidental ANTABUSE-alcohol reaction, ANTABUSE (disulfiram) should be used with extreme caution in patients with any of the following conditions: diabetes mellitus, hypothyroidism, epilepsy, cerebral damage, chronic and acute nephritis, hepatic cirrhosis or insufficiency.

USAGE IN PREGNANCY: The safe use of this drug in pregnancy has not been established. Therefore, ANTABUSE (disulfiram) should be used during pregnancy only when, in the judgment of the physician, the probable benefits outweigh the possible risks.

PRECAUTIONS: It is suggested that every patient under treatment carry an Identification Card, stating that he is receiving ANTABUSE and describing the symptoms most likely to occur as a result of the ANTABUSE-alcohol reaction. In addition, this card should indicate the physician or institution to be contacted in emergency. (Cards may be obtained from Ayerst Laboratories upon request.)

Alcoholism may accompany or be followed by dependence on narcotics or sedatives. Barbiturates have been administered concurrently with ANTABUSE (disulfiram) without untoward effects, but the possibility of initiating a new abuse should be considered.

Base line and follow-up transaminase tests (10-14 days) are suggested to detect any hepatic dysfunction that may result with ANTABUSE therapy. In addition, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

ADVERSE REACTIONS: (See Contraindications, Warnings, and Precautions.)

Occasional skin eruptions are, as a rule, readily controlled by concomitant administration of an antihistaminic drug.

In a small number of patients, a transient mild drowsiness, fatigability, impotence, headache, acneform eruptions, allergic dermatitis, or a metallic or garlic-like aftertaste may be experienced during the first two weeks of therapy. These complaints usually disappear spontaneously with the continuation of therapy or with reduced dosage.

Psychotic reactions have been noted, attributable in most cases to high dosage, combined toxicity (with metronidazole or isoniazid), or to the unmasking of underlying psychoses in patients stressed by the withdrawal of alcohol.

There have been reports of polyneuritis and peripheral neuritis, and rare instances of optic neuritis. One case of cholestatic hepatitis has been reported, but its relationship to ANTABUSE has not been unequivocally established.

DOSAGE AND ADMINISTRATION: ANTABUSE (disulfiram) should never be administered until the patient has abstained from alcohol for at least 12 hours.

INITIAL DOSAGE SCHEDULE: In the first phase of treatment, a maximum of 500 mg. daily is given in a single dose for one to two weeks. Although usually taken in the morning, ANTABUSE may be taken on retiring by patients who experience a sedative effect. Alternatively, to minimize, or eliminate, the sedative effect, dosage may be adjusted downward.

MAINTENANCE REGIMEN: The average maintenance dose is 250 mg. daily (range, 125 to 500 mg.); it should not exceed 500 mg. daily.

NOTE: Occasional patients, while seemingly on adequate maintenance doses of ANTABUSE (disulfiram), report that they are able to drink alcoholic beverages with impunity and without any symptomatology. All appearances to the contrary, such patients must be presumed to be disposing of their tablets in some manner without actually taking them. Until such patients have been observed reliably taking their daily ANTABUSE tablets (preferably crushed and well mixed with liquid), it cannot be concluded that ANTABUSE is ineffective.

DURATION OF THERAPY: The daily, uninterrupted administration of ANTABUSE must be continued until the patient is fully recovered socially and a basis for permanent self-control is established. Depending on the individual patient, maintenance therapy may be required for months or even years.

TRIAL WITH ALCOHOL: During early experience with ANTABUSE, it was thought advisable for each patient to have at least one supervised alcohol-drug reaction. More recently, the test reaction has been largely abandoned. Furthermore, such a test reaction should never be administered to a patient over 50 years of age. A clear, detailed, and convincing description of the reaction is felt to be sufficient in most cases.

However, where a test reaction is deemed necessary, the suggested procedure is as follows:

After the first one to two weeks' therapy with 500 mg. daily, a drink of 15 cc. (½ oz.) of 100 proof whiskey or equivalent is taken slowly. This test dose of alcoholic beverage may be repeated once only so that the total dose does not exceed 30 cc. (1 oz.) of whiskey. Once a reaction develops, no more alcohol should be consumed. Such tests should be carried out only when the patient is hospitalized, or comparable supervision and facilities, including oxygen, are available.

MANAGEMENT OF ANTABUSE (DISULFIRAM)-ALCOHOL REACTION: In severe reactions, whether caused by an excessive test dose or by the patient's unsupervised ingestion of alcohol, supportive measures to restore blood pressure and treat shock should be instituted. Other recommendations include: oxygen, carbogen (95 per cent oxygen and 5 per cent carbon dioxide), vitamin C intravenously in massive doses (1 Gm.), and ephedrine sulfate. Antihistamines have also been used intravenously. Potassium levels should be monitored particularly in patients on digitalis since hypokalemia has been reported.

HOW SUPPLIED: No. 809—Each tablet (scored) contains 250 mg. disulfiram, in bottles of 100. No. 810—Each tablet (scored) contains 500 mg. disulfiram, in bottles of 50 and 1,000.

AYERST LABORATORIES
New York, N.Y. 10017
Ayerst®

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ANTABUSE®

BRAND OF DISULFIRAM

ROCHE

Introducing

The unforgettable antidepressant

Endep™ 75 mg

amitriptyline HCl/Roche

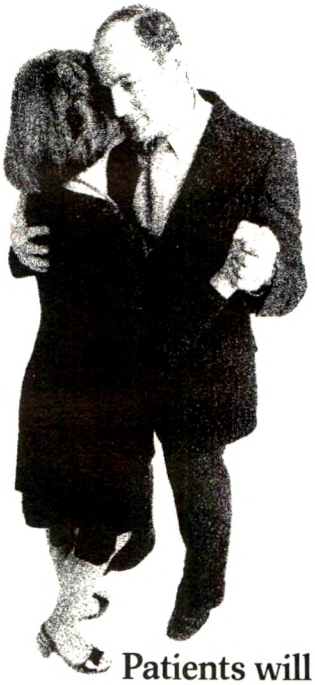
Here's what makes it unforgettable



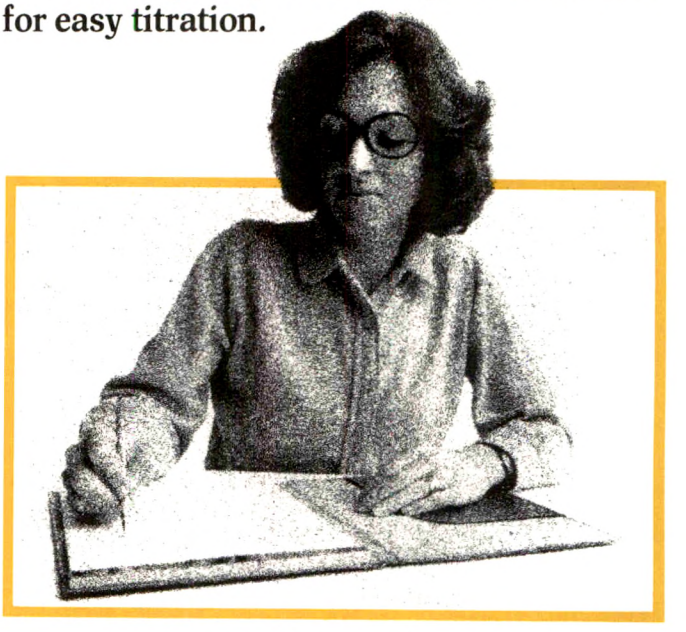
Patients are more likely to remember the single bedtime dosage of Endep (amitriptyline HCl).



You'll remember 1 or 2 tablets *h.s.*, because fewer forgotten doses produce a more consistent clinical effect, and because it is the only tricyclic antidepressant scored for easy titration.



Patients will also remember family/interpersonal relationships that they have forgotten or neglected.



In three or four weeks patients will usually remember how well they used to feel.

Endep™ 75 mg

amitriptyline HCl/Roche

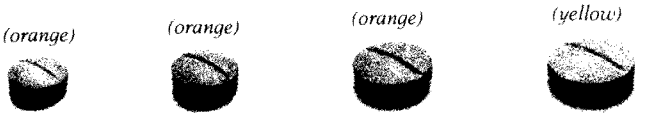
The antidepressant you can prescribe at night to work all day

Before prescribing, please consult complete product information, a summary of which appears on the following page.

Introducing Endep™

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day



10-mg scored tablets | **25-mg** scored tablets | **50-mg** scored tablets | **75-mg** scored tablets

■ Endep (amitriptyline HCl), in responsive patients, lifts mood and relieves symptoms such as anorexia, loss of interest, inability to concentrate, decreased libido or GI disturbances, often within three or four weeks after starting therapy.

■ Endep usually relieves accompanying sleep disturbances, particularly early in the treatment regimen.

■ All four dosage strengths are scored for greater flexibility and convenience in titration.

■ The 75-mg *h.s.* dose minimizes skipped doses, maximizes patient acceptance and compliance.

■ Specifying Endep/Roche assures quality plus potential economy.

Important Considerations

Should not be given concomitantly with an MAO inhibitor.

Not recommended for use during the acute recovery phase following myocardial infarction.

Contraindicated in patients with hypersensitivity to amitriptyline.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh potential benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels; increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier. *Initial Adult Dosage:* Outpatients – 25 mg t.i.d.; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate – 50 to 100 mg h.s., gradually increasing h.s. dose up to 150 mg/day. Hospitalized Patients – Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day. *Adolescent and Elderly Patients:* In general, 10 mg t.i.d. with 20 mg h.s. may be satisfactory for those who do not tolerate higher doses. *Maintenance Dosage:* With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg b.i.d. to q.i.d., or 10 mg q.i.d. Continue maintenance therapy 3 months or longer to avoid relapse.

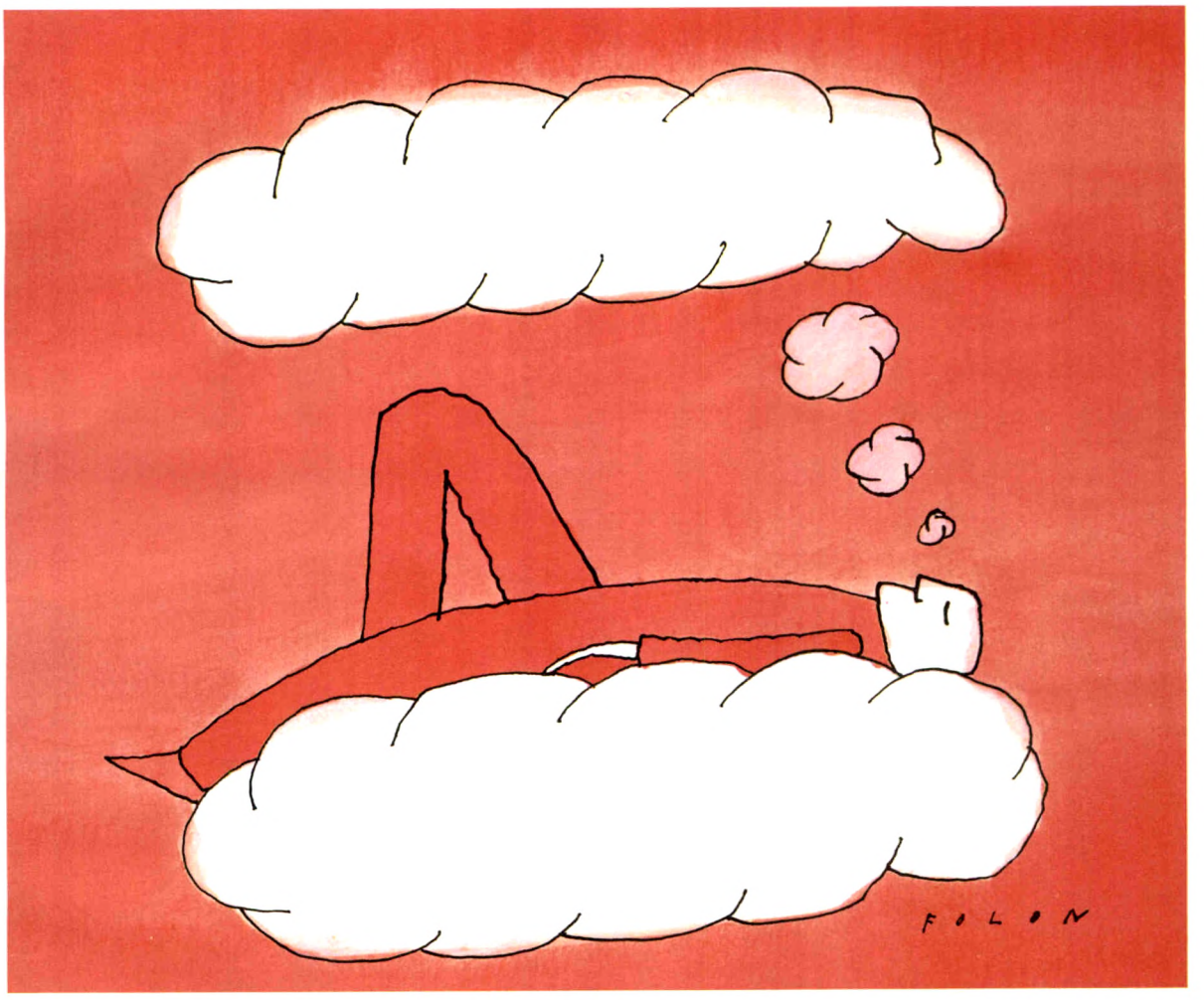
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg and 75-mg scored tablets – bottles of 100 and 500; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg), available singly and in trays of 10.

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Roche Laboratories
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Nutley, New Jersey 07110

Doctor, I dreamed last night you saved me lots of money!



Prescribe low-priced

SK-Pramine™

(imipramine HCl, SK&F)

Tablets 10 mg., 25 mg., 50 mg.

SK-LINE®
Smith Kline & French Laboratories
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The directory contains two major sections: the ALPHABETIC SECTION, containing the name, address, membership class, year joined and board certification for each member; and the GEOGRAPHIC SECTION, containing name, membership class, and board for each member arranged as follows: United States, alphabetically by state and city; Canada, alphabetically by province and city; and Foreign, alphabetically by country and city.

In addition, the directory contains a listing of officers, committees, councils and historical resumes.

197 pages

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Prize competition

announced by the

Anna Monika Foundation

for the investigation of the physical substrate and functional disturbances of endogenous depressions by approval of the Minister of the Interior of Nordrhein-Westfalen, Düsseldorf, on 9th June, 1965.

The Foundation announces the following prizes:

FIRST PRIZE	**10.000, - US Dollars**
SECOND PRIZE	**5.000, - US Dollars**
THIRD PRIZE	**2.500, - US Dollars**

preferably for studies of biochemical, histological, neurophysiological, neuropathological, psychopharmacological, psychiatric or psychosomatic nature. The studies should be carried out in close co-operation with a psychiatric clinic, a university institute or an equivalent scientific institution. So far as possible, *the papers should give information about recent advances* in knowledge that would be helpful in promoting treatment and would open up new paths of progress. The papers may be written in German, French or English and should be submitted to the chairman of the committee, Professor Dr. Kielholz, Basle, Switzerland. Besides hitherto unpublished studies, papers published in the past two years in a German or international professional journal may also be submitted. *Deadline for submission to the Committee is 30th September, 1976.* To help the Committee to come to a speedy decision it is requested that at least four copies including an abstract covering the content of all papers should be submitted. Prizes will be awarded on the 30th June, 1977. If, in the opinion of the Committee, no papers of sufficient merit are submitted, it reserves the right to make no award. Prizes and their amount will be awarded according to the merits of the study in question. Subject to the Committee's decision, each prize can be divided between two papers.

The Committee of Judges Includes:

Chairman: Professor Dr. P. Kielholz, Director of the Psychiatric Clinic of the University of Basle, CH-4056 Basle, Switzerland, Wilhelm-Klein-Strasse 27.

Professor Dr. Karl Bernhard, Institute for Physiological Chemistry, Basle, Switzerland

Professor Dr. Otto Creutzfeldt, Director of the Max-Planck-Institute of Biophysical Chemistry, Göttingen, Germany

Professor Dr. H.J. Dengler, Director of the Medical Clinic of University Bonn, Germany

Professor Dr. Fritz A. Freyhan, M.D., F.A.P.A. Washington, D.C. 20009, USA

Professor Dr. K. Heinrich, Director of the Psychiatric Clinic of the University of Düsseldorf, Germany

Professor Dr. H. Hippius, Director of the Psychiatric Clinic and Out-patient Clinic Munich, Germany

Professor Dr. Horst Jatzkewitz, Director of the Max-Planck-Institute for Psychiatry, Neurochemical Department, Munich, Germany

Professor Dr. D. Palm, Director of the Pharmalogical Institute of the University of Frankfurt/ Main, Germany

Professor Pierre Pichot, Head of Medical Psychology, Centre Psychiatrique Sainte-Anne, Paris, France

Professor Dr. Wilhelm Stoffel, Director of the Institute of Physiological Chemistry, University of Cologne, Germany.

Director of the Foundation: PETER REHME, D-4600 DORTMUND, TÖLLNERSTR. 9/11

The familiar refrain of depression: morning fatigue... sadness... anorexia... insomnia

Now, Merrell offers Norpramin (desipramine hydrochloride tablets N.F.) to effectively relieve these common manifestations of depression.

Norpramin also provides additional benefits in treatment of your patients.

- ☐ effectively relieves physical, psychological and emotional symptoms of depression
- ☐ minimal daytime sedation — important for patients who must be alert to perform daytime activities
- ☐ relief that may begin in 2 to 5 days — but full therapeutic effect is seldom seen before 2 weeks
- ☐ side effects rarely require discontinuation of therapy
- ☐ increases interpersonal responsiveness
- ☐ can help facilitate psychotherapy

Prescribe Norpramin to change the familiar refrain of depression in your practice.

Norpramin®
(desipramine hydrochloride tablets N.F.)

Brief Summary

Indications: Norpramin (desipramine hydrochloride tablets N.F.) is indicated for the relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Desipramine hydrochloride should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Contraindications include the acute recovery period following myocardial infarction and hypersensitivity to the drug. Cross sensitivity with other dibenzazepines is a possibility.

Warnings: 1. Extreme caution should be used in patients: (a) with cardiovascular disease. (b) with a history of urinary retention or glaucoma. (c) with thyroid disease or those on thyroid medication. (d) with a history of seizure disorder. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. *Use in Pregnancy:* Safe use during pregnancy and lactation has not been established. 4. *Use in Children:* Norpramin is not recommended for use in children. 5. This drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. Therefore, the patient should be cautioned accordingly.

Precautions: This drug should be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with drugs of this class. If possible, dispense in child-resistant containers. It should be kept out of reach of children. Reduce dosage, or alter treatment, if serious adverse effects occur. Norpramin therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates and may cause exacerbation of psychosis in schizophrenic patients. Use cautiously with anticholinergic or sympathomimetic drugs. Response to alcoholic beverages may be exaggerated. In the concurrent administration of ECT and antidepressant drugs one should consider the possibility of increased risk relative to benefits. Discontinue as soon as possible prior to elective surgery because of possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy: the drug should be discontinued if there is neutropenia.

Adverse Reactions: *Cardiovascular:* hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke. *Psychiatric:* confusional states (especially in the elderly), hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis. *Neurological:* numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus. *Anticholinergic:* dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, hypotonic bladder. *Allergic:* skin rash, petechiae, urticaria, itching, photosensitization, edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs. *Hematologic:* bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue. *Endocrine:* gynecomastia, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels. *Other:* jaundice (simulating obstructive), altered liver function; weight gain or loss, perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia. *Withdrawal Symptoms:* Though not indicative of addiction, abrupt cessation after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: *The usual adult dose:* 50 mg. three times daily; increase if necessary after 7 to 10 days to maximum of 200 mg. daily. Dosages above 200 mg. per day are not recommended. *Maintenance:* At a lower dose adequate to maintain remission. *Adolescent and geriatric patient dose:* 25 to 50 mg. daily; increase to 100 mg. daily if necessary.

Overdosage: There is no specific antidote for desipramine, nor are there specific phenomena of diagnostic value characterizing poisoning by the drug. The principles of management of coma and shock by means of the mechanical respirator, cardiac pacemaker, monitoring of central venous pressure and regulation of fluid and acid-base balance are well known in most medical centers. If heart failure is imminent, digitalize promptly.

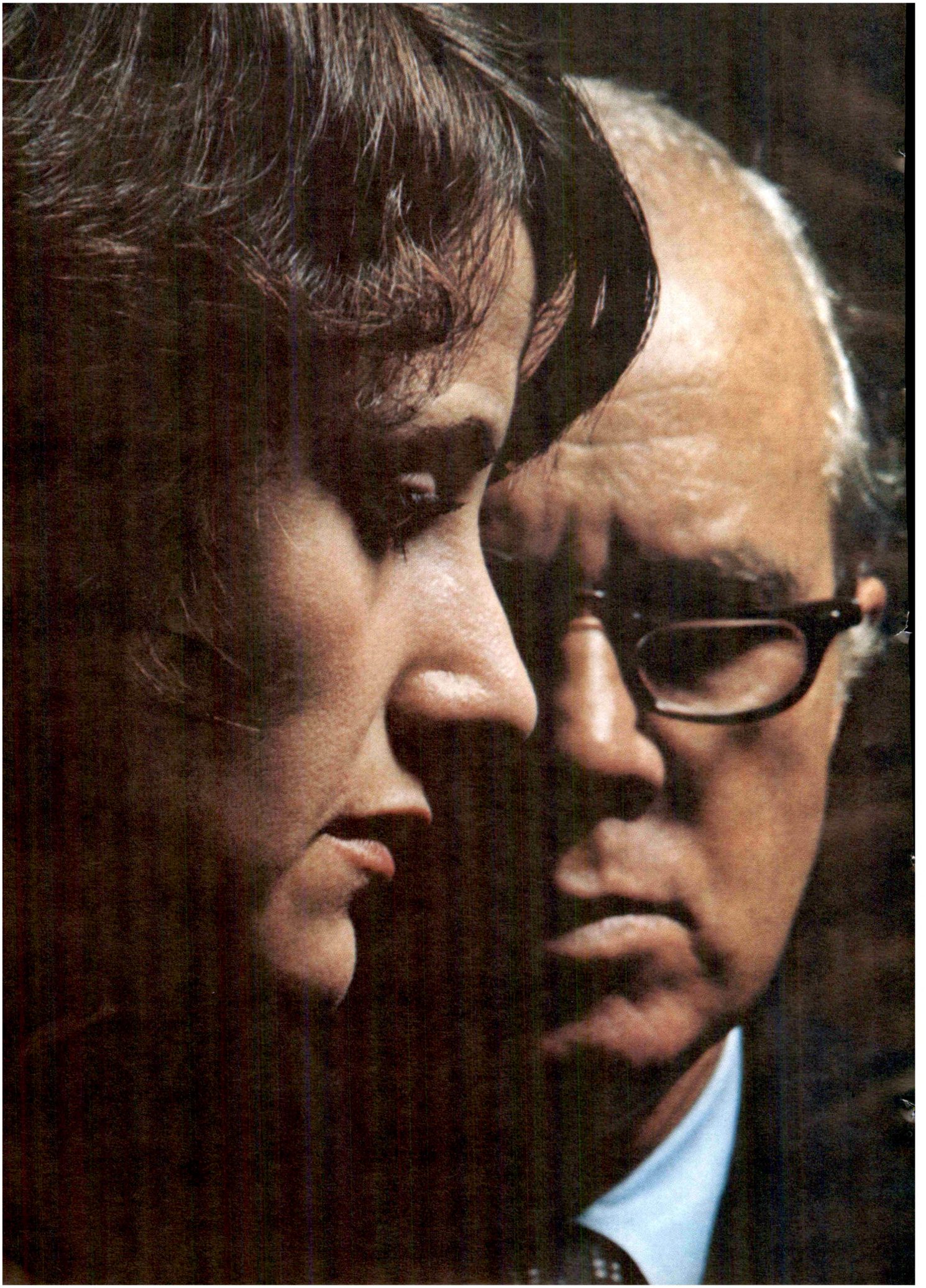
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Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215
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Norpramin®
(desipramine hydrochloride)
25 mg., 50 mg. tablets

lightens and brightens
the days of your
depressed patients

Merrell



The TRIAVIL® Potential in the management of moderate to severe anxiety with depression

When time and talk are not enough...
The therapist is the primary catalyst for change in the psychotherapeutic relationship. However, when patients suffer from moderate to severe anxiety with depression, there are situations when TRIAVIL can often be a useful adjunct.

...TRIAVIL may help
There are three important benefits you may expect when TRIAVIL is part of the treatment program: (1) When symptoms of moderate to severe anxiety or agitation with depression are relieved, the patient may become more accessible and cooperative. (2) As somatic manifestations are controlled, attention may be focused on underlying causative factors. (3) Symptomatic relief may enable the patient to function more effectively in his daily life while your work with the patient progresses.

Tablets TRIAVIL are available in four different combinations affording flexibility and individualized dosage adjustment. Since it is simpler to remember to take one tablet rather than several (particularly in multiple daily doses), your patients on TRIAVIL will be more likely to take proper doses of the medication.

TRIAVIL is contraindicated in CNS depression from drugs; in the presence of evidence of bone marrow depression; and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. Not recommended in children or during pregnancy. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please turn to the following page.

when patients exhibit moderate to marked anxiety or agitation with symptoms of depression

TRIAVIL® **containing perphenazine and amitriptyline HCl**

a tranquilizer-antidepressant

for highly effective relief of depression with moderate anxiety

TRIAVIL®

containing perphenazine and amitriptyline HCl a tranquilizer-antidepressant

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WARNINGS: TRIAVIL should not be given with guanethidine or similarly acting compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, particularly in high doses, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. Caution patients performing hazardous tasks, such as operating machinery or driving motor vehicles, that drug may impair mental and/or physical abilities. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Side effects may be any of those reported with phenothiazine drugs: extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased or another drug substituted, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and that the full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; hypnotic effects; pigmentary retinopathy; corneal and lenticular pigmentation; occasional lassitude, muscle weakness, mild insomnia. Other adverse reactions reported with various phenothiazine compounds include blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); liver damage (jaundice, biliary stasis); grand mal convulsions; cerebral edema; polyphagia; photophobia; skin pigmentation; and failure of ejaculation.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness; weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; jaundice; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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I CLI ICALLY SIG IFICA T DEPRESSIVE NEUROSIS...



For Brief Summary, please see following page.

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RESULTS OFTEN SEEN IN A WEEK



Because the somatic and psychic complaints that accompany depressive neurosis can interfere with the treatment of the underlying psychopathology—rapid relief of these symptoms is often crucial to therapy.

In 14 double-blind studies of four weeks duration, 339 patients with depressive neurosis received Mellaril. In these studies, 55% of the overall improvement was observed by the end of the first week, and a total of 293 patients (86%) improved during the four weeks.*

Mellaril usually does not cause euphoria or undue sedation and is nonaddicting. Patients, therefore, generally remain alert and better able to respond to therapy. (The physician should, however, caution patients against participating in activities which require complete mental alertness, e.g., driving.)

*Data on file at Sandoz Pharmaceuticals.

MELLARIL®
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TABLETS: 25 mg and 50 mg thioridazine HCl, U.S.P.

for the short-term treatment of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling. The following reactions have occurred with phenothiazines and should be considered: *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Women in Psychiatry: A Review

BY NANCY A. ROESKE, M.D.

Throughout the world, psychiatry is in a period of accelerated transition. In each nation the role of women in psychiatry, and the role of psychiatry in the lives of women, reflect one aspect of the development of egalitarianism. Women psychiatrists have perennially been confronted with conflicts between their professional and private life responsibilities. Internationally, the external and internal factors that prohibit or limit women's personal development and vocational participation are being scrutinized. American psychiatry is proving to be a pioneer among medical specialties in this area.

THE UNITED NATIONS designated 1975 as International Women's Year. This action called attention to the universal right of women to have an equal opportunity for self-development and self-expression. It has prompted many articles reviewing the current involvement of women in their country's educational, vocational, social, and political life (1, 2). As might be expected, there is evidence that cultural attitudes are changing slowly, even in countries where laws make women the equal of men. In addition to cultural attitudes, other key factors affecting the role of women include 1) each nation's decisions about its economic requirements, and 2) the health care needs of its people.

Thus the presence and role of women psychiatrists reflect the complex interaction of historical, cultural, religious, economic, educational, political, and legal factors in each country. The role of women psychiatrists must be understood within the context of the role of psychiatry, the role of women physicians, and the evolving role of all women within each nation.

Dr. Roeske is Professor and Director of Undergraduate Curriculum, Department of Psychiatry, Indiana University School of Medicine, 1100 West Michigan St., Indianapolis, Ind. 46202.

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VARYING CONCEPTS OF PSYCHIATRY AND WOMEN PSYCHIATRISTS

The number and role of women physicians, and the teaching and practice of psychiatry, vary remarkably throughout the world (3, 4). The German Federal Republic, Poland, the Union of Soviet Socialist Republics, and the United States of America report the largest number of women physicians (see table 1). Data are currently unavailable from China. Viewed in terms of percentages, however, in the 1960s the United States, Spain, and Japan were cited as having the lowest percentages of women physicians (United States, 6.8%; Spain, 9.1%; Japan, 8%) (5). Cultural attitudes antithetical to women in medicine were described as major deterrents. The percentages of women physicians in western European countries and other English-speaking countries, notably Canada and Australia, have been reported as varying between 15 and 30% (5).

The most significant reasons given for the higher percentages of European women seeking professional training include 1) the European tradition of family continuity in careers (so that a daughter may be encouraged to enter her father's profession), and 2) the loss of men during World War II (5, 6). This latter factor has been particularly important in the United Kingdom, Germany, Poland, Finland, Czechoslovakia, Austria, Yugoslavia, and the U.S.S.R. (3). In Russia, the following factors have been described as contributing to a woman's choice of medicine as a career: a general necessity for self-support because of the acute shortage of husbands due to wars and purges; a necessity for lifelong work, even if one is married, because of the low family income; and limited options for women who want to rise out of the lowest working class, since men are given preference in the nonmedical sciences and in engineering. Yet despite the large number of women physicians in the Soviet Union, less than 5% of them achieve high-level medical posts in administration, academia, and research (7, 8).

The 1970 *World Health Statistics Annual* (3) gives a reliable but limited review of the worldwide distribu-

TABLE 1
Countries with Comparable Numbers of Women Physicians, 1970*

Country	Total	Men	Women
United States			
Physicians	351,623	325,820	25,803
Psychiatrists	23,240	20,335	2,905
West Germany			
Physicians	105,976	86,960	19,016
Neurologists/psychiatrists	3,009	2,369	640
Poland			
Physicians	49,283	25,806	23,477
Psychiatrists	1,139	—	—
U.S.S.R.			
Physicians	577,330	138,559	438,771
Psychiatrists	14,500	—	—

* Based on data from the World Health Organization (3).

tion of physicians by sex, according to their medical specialty. Although every country in the world was asked to submit data, only 69 complied. The variability of completeness of the data, the differences in training patterns, and the different concepts of a psychiatrist's identity and function all pose problems in making international comparisons of men and women psychiatrists.

For countries reporting similar numbers of women physicians, it is interesting to review the number of psychiatrists in general, and of women psychiatrists in particular (see table 1). Although it is not necessarily a reflection of the role and importance of psychiatry in medicine, psychiatrists are present in appreciable numbers in only the United States and the U.S.S.R. (3). The exceptionally large number of psychiatrists in the United States attests to the impact of pre- and post-World War II immigration. Although Russia reports fewer psychiatrists than the United States it may have more women psychiatrists, since three-fourths of all its physicians are women. (Statistics that would confirm this possibility are unavailable.)

Mental health specialists, including psychiatrists, are in very short supply in nearly all of the developing countries (3). It is understandable that the few practicing physicians in these countries are primarily involved in child and maternal health, general medicine, public health, parasitology, and virology. This is not to say that these countries are without mental illness; epidemiological studies of mental disorders have shown a similarity between morbidity rates for the more severe disorders in both developed and developing countries (the United States vs. many countries in Africa, for example) (9). Nonetheless, psychiatry is a seriously neglected subject in most medical schools in developing countries. If courses are offered at all, they consist mainly of presentations of hospitalized patients by part-time faculty (4). Mentally ill persons are usually treated by male village healers, who may work in collaboration with local physicians (9).

Because of the limited availability of medical training programs in developing nations, their psychiatrists are usually educated in the United States or Europe. Yet the cultural dissonance between the country where the psychiatrist is educated and the country where she or he practices presents problems of professional adaptation.

As might be anticipated, there are few women psychiatrists in the developing nations. This situation is due to the following factors: cultural attitudes toward higher education and professional occupations for women, social attitudes about women traveling and living alone for educational and professional reasons, the expense of an education abroad, and dual wife-husband career problems and family conflicts. Nonetheless, the foreign woman medical school graduate frequently comes to the United States and later plays an important role in American psychiatry. Forty percent of the women psychiatrists in the United States are foreign medical graduates (10); about 36% of the rest of the women physicians in the United States are also foreign medical graduates (11). In the past 5 years an appreciable number of these women have probably come from Asian countries (11), although specific data regarding country of origin are currently unavailable. These women are confronted with the problems of language, acculturation, and a less than adequate medical school education in psychiatry.

FACTORS INFLUENCING WOMEN'S PROFESSIONAL LIVES

When examined in the light of their effect on women in psychiatry, the statistical reports from the World Health Organization (3) can be viewed as a commentary on the impact of such factors as each nation's culture and its health care needs. Unfortunately, however, fewer than 40 studies in the English literature have made a detailed investigation of the factors influencing the professional lives of women physicians, including psychiatrists.

Still, the results of research in the United States (12–22), West Germany (23), England and Scotland (24–31), Canada (32–34), Australia (35), and New Zealand (36) indicate remarkable similarities in women physicians' experiences and personality characteristics. These similarities include family background, the effect of marriage and children on the woman's career, the effect of the availability of professional education and experience on the woman's career, the results of personality assessment tests, and the woman's self-perception. Hence, a fairly valid composite profile of women psychiatrists can be derived from these studies.

It should be kept in mind, however, that statistics are as much a point of view as they are a method; they are a way of asking questions and examining evidence. Therefore the reader should be aware of a number of methodological problems that become apparent upon detailed comparison of research projects. These in-

clude variations in the following areas: the size of populations; composition of populations (age, specialty training, and geographical location, including specific characteristics of educational institutions); focus of the study (longitudinal vs. cross-sectional); number of research topics; the questions concerning a given topic; and the types of tests used to assess personality characteristics. A final methodological problem is the frequent absence of a comparable group of men.

It is noteworthy that the vast majority of the studies of practicing women physicians have dealt with a population sample that was educated prior to 1965, although many of these projects have been published in the literature since 1970. Therefore there is a need for current research to clarify the factors that facilitate and impede a woman psychiatrist's career.

These studies demonstrate that a woman's choice of a medical specialty is not directly related to her degree of professional involvement (14, 15, 17, 22). Contrary to popular opinion, women do not seem to choose psychiatry in order to work full-time. Women with children are just as likely to work part-time in psychiatry as in any other field of medicine.

Composite Profile of the Woman Psychiatrist

The composite picture presented by studies of the practicing woman psychiatrist (12–36) is one of a highly self-reliant woman who seeks to balance the demands upon her private life with the available options for professional education and career. Women psychiatrists are more likely than nonphysician women to have been raised in an urban community family, where the father had a university education and was a professional person and the mother was educated beyond secondary school and worked during the psychiatrist's childhood. Both parents stimulated and supported their daughter's interest in higher education and a profession. The decision to become a physician was usually made before or during early adolescence. Women psychiatrists have usually had a high interest in biological sciences and a desire to help people. They are different from the majority of other university women in their persistence to achieve self-expression. Outstanding achievement in medical school and in specialty training is unrelated to later career patterns. Approximately 80% of women psychiatrists marry, and 50% of them marry physicians.

They have the same number of years of postgraduate training as men, although their training and/or professional practice is usually disrupted by child bearing, since flexible schedules in training programs and jobs in conjunction with day-care centers are rarely available. Women psychiatrists are more likely to work part-time or not at all, depending upon the number and ages of their children. (The amount of time for a career decreases rapidly when there are 3 or more children and/or the children are less than 5 years old.) Women are more likely than men to work in institutional settings with fixed employment hours, and on the average, married women have a more varied practice pattern (full-time, part-time, or not at all) than men do. Overall, women practice 30% fewer hours than men, although single women's professional involvement (hours and number of patients) is similar to that of their male counterparts. Approximately 35% of women and 50% of men psychiatrists have specialty certification. Few women psychiatrists (2–3%) are found in each of the following areas: education, research, administration, or the overall policy-making components of psychiatry (10, 11).

Career Development

Thus, for most women psychiatrists, their career pattern is a resolution of the conflicts generated by such factors as 1) familial demands upon their time versus familial support and help, 2) the availability of child care, 3) the availability of a flexible time schedule in training and jobs, and 4) the opportunity for career development in areas of psychiatry where few women are currently involved.

In many ways geography can be another crucial factor in the career development of women psychiatrists. Being located in a densely populated state, such as California or New York, correlates with having a greater degree of choice in terms of variety of jobs, location of education, and husband's career location.

Few articles have explored women's thinking about these factors and their resolution of these conflicts. English researchers Lawrie and associates (25, 26), Flynn and Gardiner (27), Stanley and Last (28), Eskin (29), and Whitfield (31), motivated by the shortage of physicians and the "wastage of woman power," have found that in Great Britain the national health care system limits a woman's career development since promotions are based on the amount of time spent in a career. High taxation, the unavailability and/or high cost of domestic help, geographical distance from educational institutions, and the lack of child care facilities are other factors limiting a woman's professional commitment. Therefore 25–30% of English women physicians state that they wish to work more than they are currently able to do, or that they have chosen to work in a specialty that was not their first choice. Finally, in Ashurst's recent report (37) on the women psychiatrists of southwest England, "lack of mobility" was cited as the major deterrent to career development of married women.

The Scottish researchers Ulyatt and Ulyatt (38, 39) have questioned whether it is only these external factors, realistic as they may be, which are the basis for career decisions. They stated that, while she is in medical school, a woman's anticipation that her marriage and family will take precedence over her career is inversely related to her working full-time. Such factors as her desire to be close to her family and her fear of damaging her children by absence are also inversely related to full-time employment.

Another study of factors influencing a woman's professional involvement was conducted by Heslop and associates (36) in New Zealand. More than half of the

married women with children, whether they worked full-time or part-time, chose the specialty or job that fitted in with their domestic arrangements and their husband's career decisions. For the women who did not work because of children, their loss of confidence and feeling of professional inadequacy were clearly regarded as major difficulties in resuming work. The women explained their absence from the areas of administration and education as being due to lack of interest, limitations of available time, and the unlikelihood of appointment in a predominantly male profession.

Career Conflicts

Women's perception of men's acceptance and support of their careers was experimentally studied by Horner and Walsh (40). In their study of women and men medical students they found that the conflicts between achievement in medical school and acceptance by male peers resulted in women lowering their level of academic achievement. Later, as physicians, women may think they should be more passive than men think is necessary. Steinmann and associates (41, 42) found that eager, ambitious women physicians often thought that men's ideal woman was a "very passive one." On the other hand, the men physicians' ideal woman was in fact active, outgoing, and oriented toward self-realization. In addition, Shapiro (43) explored the feelings of physician-mother psychiatry residents in a pioneer part-time program at New York Medical College (44), the choice of which reflects the women's desire for a lifestyle that incorporates their interests in both family and profession. In the examples given of dual career development conflicts, the husbands were more tolerant of separation for a period of time than the women had anticipated.

Of all the possible solutions to the pressures and demands made on the life of the woman physician, the most negative and self-destructive answer to conflicts in public and private life is suicide. The suicide rate of women physicians in the United States (33.6 per 100,000) is three to four times higher than that expected for women over 25 years old (45, 46). The suicide rate of women physicians does not differ statistically from that of men doctors. However, women physicians differ from other women and from men physicians in their prime age range for suicide. Almost half of the suicides by women physicians occur before age 40, and approximately three-fourths occur before age 50. In comparison, half of the suicides by men physicians occur before age 50. The small sample size of the populations in the two studies cited (17 women in the first study and 41 in the second) does not lend itself to valid statistical analysis of other factors, including choice of specialty and marital status. There is an indication, however, that single women in residency training are the most vulnerable group.

Comment

Our present understanding of women psychiatrists is based upon research of the 1960s. The 1970s have seen an international burgeoning of articles about women in both the popular and scientific literature. Women and men writers have questioned and reviewed the biological, cultural, economic, legal, philosophical, and religious sanctions that, in effect, degrade women. Whether or not this present awareness can modify women's decisions about their careers will also depend upon changes in education, organizations, and institutions. The extent to which changes have occurred, and their effect upon women psychiatrists' careers, have yet to be reported in the literature from other countries.

AMERICAN WOMEN IN PSYCHIATRY—1975

For the past 50 years psychiatry has been the second most frequently chosen specialty by women (pediatrics is the first) (11, 14, 15, 47). Therefore, because of the number of women psychiatrists and the humanistic concerns of the profession, it is not surprising that the American Psychiatric Association appointed a task force on women in 1972. Among the medical specialties, psychiatry is a pioneer in this area. However, when considered within a wider educational and sociological context, the changes in the field of psychiatry are similar to the changes that have occurred in the organization of other branches of higher education (notably in the areas of sociology and psychology) (48). In each instance, a relatively small group of women has become involved in investigation and research. The results of this involvement include greater activity by other women in the profession and recommendations for organizational changes within the professional associations.

Following a caucus at the 1972 APA annual meeting, the Task Force on Women was formed to examine both the role of women in psychiatry and the effect of psychiatry in the lives of women (22, 49–52). At the 1973 and 1974 annual meetings there was a substantial increase in the number of papers (4% to 15%) presented by women, and spouses were invited to attend the professional meetings and were provided with a pamphlet of program excerpts. In 1975, for the first time, child care was provided at the annual meeting.

A number of organizational changes were simultaneously taking place. The number of women in the legislative components of the organization was increased to equal the 12% of women members in the Association. Dr. Rebecca Solomon was the first woman to be appointed to the Editorial Board of the *American Journal of Psychiatry* (1973). Dr. Jeanne Spurlock was the first woman to be appointed as a Deputy Medical Director of the Association (1974). In 1974 the Board of Trustees accepted the recommendation of the APA Task Force on Women for a committee on women. The major purposes of the Committee on Women are as follows:

1. To define and recommend action to meet the mental health needs of women by reassessing the current

theory and practice of psychiatry in terms of its congruence with the realities of women's lives.

2. To promote women psychiatrists' involvement in academics, research, administration, and professional organizations and to develop methods for increasing their participation and leadership.

3. To review and stimulate research and the development of theories necessary to implement the first two purposes.

4. To provide support systems for women colleagues.

The significance of this anticipatory action will be revealed over time. However, the charges given to the committee reflect the awareness of the task force that never before in American history have so many women sought higher education and professional careers. The exigency of the situation is supported by two facts: the rapid rise in the number of women entering medical school and the limited knowledge about women psychiatrists as teachers, researchers, administrators, and therapists.

In the 1975–1976 first-year medical school classes nationwide, women comprised 23.8% of the enrollment. The total enrollment of women medical students was 11,417 or 20.5% (53). The proportion of women psychiatry residents is also rising gradually, from 19.8% in 1972 to 22.4% in 1974 (10). In 1974 there were 878 women residents (421 U.S. and Canadian graduates and 457 foreign graduates) in general psychiatry and 131 women (61 U.S. and Canadian graduates and 70 foreign graduates) in child psychiatry (54, p. 1365).

The majority of women psychiatrists are educated and work in the following 7 states: New York, California, Massachusetts, Pennsylvania, Illinois, Maryland, and New Jersey. (New York with 723 and California with 301 women psychiatrists have one-third of all women psychiatrists in the country.) Montana, North Dakota, South Dakota, Nevada, Wyoming, and Idaho have 4 or fewer women psychiatrists. This pattern is similar to the geographical distribution of women in other specialties (55).

Gender Identity

While there are numerous raw statistics about women medical students and the professional activities of women psychiatrists (see tables 2 and 3), detailed descriptive data are meager. (There may be slight variations in data for a given year depending upon sources used.) The meaning of being a woman to the woman resident and her teachers, to the woman therapist and her patients, to the woman teacher and her students, to the woman researcher and her teachers and colleagues, and to the woman administrator has received scant attention. The few published articles are valuable personal accounts (50, 51, 58–62).

In general, there appears to be repeated denial that a person's sexual identity modifies interpersonal relationships. Women psychiatrists in residency training have noted that they and their male and female supervisors deny that being a woman influences the supervisory experience or the woman's role with her patients (22, 49–52). Yet in the face of all this denial, the woman psychiatrist can find herself expected to fulfill stereotypical feminine roles with and for men colleagues (58, 59). In addition, women psychiatrists may be objects of envy, anger, competition, and identification by nonpsychiatrist professional women, women patients, and nonprofessional women.

As therapists, women psychiatrists may be particularly helpful to the severely disturbed patient who had a distorted mother-child relationship, and to the hysterical woman, who cannot manipulate another woman (60, 63). Many women now seek out a woman therapist in an attempt to clarify their role as women and to remove themselves from the traditional "man in power-woman subservient" model. In therapy, these women focus on their ambivalence about the nurturing capabilities of the therapist, their ingrained belief that women are basically second-rate persons, and their ingrained socially conditioned attitude that women are only rivals for men and are therefore not to be trusted.

It may also be advantageous for some men patients to be seen initially or throughout therapy by a woman psychiatrist. For example, a man may be afraid of

TABLE 2
Distribution of U.S. Women Psychiatrists by Professional Activity and Specialty, 1972*

Professional Activity	General Psychiatry	Child Psychiatry	Psychoanalysis
Office-based practice	1,349	413	38
Hospital-based practice			
Residents	663	114	0
Full-time staff	662	95	3
Other professional activities			
Medical teaching	45	23	1
Administration	127	22	0
Research	48	15	0
Other	127	19	0

* Based on data from the Fisher-Stevens report, American Medical Association (55).

TABLE 3
U.S. Medical School Faculty and Medical Students, by Sex and Ethnic Group*

Item	M.D. Faculty (1975)		Medical Students (1974–1975)	
	Men	Women	Men	Women
Total	23,280	2,527	43,357	9,786
Total psychiatrists	2,417	269	—	—
Whites	1,993	226	39,074	8,243
Blacks	32	8	2,358	1,038
Other**	164	20	1,985	505
Unknown	228	15	0	0

* Based on data from several studies (54, pp. 1334, 1338, 1339; 56; 57).
** This category includes other U.S. ethnic groups and nonwhite foreign graduates.

seeing a man psychiatrist because of a latent wish for passive homosexual surrender. The patient's anxiety may be greatly diminished if he is seen by a woman therapist (64).

Feelings and attitudes about sexuality, nurturance, power, fear of abandonment, competition, and hostility of patients, other psychiatrists, and other professional colleagues are intensified when the woman psychiatrist is pregnant (22, 49–52, 61, 62). Everyone, including the woman herself, is confronted with the realities, as well as the fantasies, of the woman psychiatrist as a person. The combined effect of psychophysiological vulnerability and career-family conflict can complicate the woman therapist's interpersonal relationships with her husband, patients, and colleagues. On the other hand, working through these conflicts can be a remarkably productive experience for therapist, patients, colleagues, and supervisors.

Choice of Professional Activity

The woman who chooses to direct her professional interests toward academia faces numerous problems beyond those confronted by the clinician. Of primary importance is the question of whether women have opportunities for academic careers. This question can be answered statistically. In 1975 there were 269 women psychiatrists and 2,417 men psychiatrists on the faculties in U.S. medical schools. Women comprised 10% of all psychiatry faculty and 9.9% of all medical school faculty (56). Few of these women are found at the higher levels of academia and research. Traditionally, the development of an academician and researcher has involved a continuity of career including research, presentations at meetings, and publications. The demands of family at the time of transition from student to faculty member represent a critical factor for career development along these lines; family pressures often act as significant deterrents.

To perhaps an even greater extent, the career problems of the woman academician and researcher carry over to the careers of women psychiatrists in administration. There is a social stereotypical factor that may be of great significance to the woman administrator. A woman who wishes to be a capable leader must refute the stereotype of being weak, emotionally labile, illogical, and unintelligent, or of being powerful and castrating. If a woman administrator is not authoritative she may be ignored as a passive woman; if she is too authoritative, she may be viewed as a destructive woman. The conflict between nonthreatening passive femininity and the demand for competence, which involves competitiveness, is very difficult for a woman to resolve; it is a constant issue for her. All of these factors may be related to the fact that in 1975 there was no woman who was chairperson of a department of psychiatry.

Role in Psychiatric Training Programs

Until 1974 there had been no reports about the role of women in psychiatric training programs. In that year the APA Task Force on Women conducted a survey of 300 such programs (65). Preliminary returns (N=167) yielded the following data: few programs actively recruited either men or women, although there was more of a tendency to actively recruit men; approximately 1 out of 5 programs reported different interviewing procedures for men and women applicants (such as asking women about their marital or family status or plans). Half of the programs offered part-time or flexible schedules for residents; child care facilities were available at only 1 out of 10 programs.

Seminar work on the psychology and sociology of women, sex roles, and gender identity was given by 1 out of 8 programs, usually on an optional basis. Few of the programs actively recruited women faculty members, and half of the programs used personal contact only in recruiting faculty members; half of the programs considered the sex of the teacher nonrelevant; more than one-third of the programs had no course hours taught by women faculty; and 10% of the supervisory hours were conducted by women. Almost all of the training programs reported that there had been no formal discussion about the status of women psychiatrists, the status of other women employees in the department, or issues regarding the treatment of women patients. Recent data (1974) revealed that 16 men and 33 women held part-time residency appointments (54). Although the reasons for this choice were not stated, the information supports the need for flexibility in residency programs for men and women.

In summary, all of the information to date about women and psychiatry supports the conviction that psychiatric residency training programs should be evaluated and possibly altered in order to increase women's competence and contribution to their field. As is so often the case, our actual behavior often differs markedly from our professed desires. Yet the increased number of women applying to residency programs will continue to stimulate changes in American psychiatric training programs.

Personal Characteristics

The fact that women's personality characteristics are particularly suitable for the role of psychiatrist has been noted by men psychiatrists for many years (66–68). Drs. Daniel Blain (67) and Karl Menninger (68) have written that women's attributes of intuitiveness, empathy, patience, and verbal skill are needed in psychiatry. These comments have been supported by research on the personal attributes of women and men medical students (13, 19, 34, 69) and by Lopate's book *Women in Medicine* (47), which is an excellent compilation of all the research in the United States prior to the mid-1960s. The Medical College Aptitude Test has been one of the standard criteria for admission to medical school. Women have usually scored higher than men in verbal ability (a measure of vocabulary strength and ability to perceive verbal relationships) and general knowledge (a measure of factual knowledge in nonscientific fields including art, eco-

nomics, geography, government, history, literature, music, philosophy, and psychology) (47).

Researchers in the United States (12, 13, 19, 70), Canada (34), and Scotland (69) have compared the personality characteristics of women and men medical students. In spite of the variety of assessment methods used, the studies consistently reported differences between women and men medical students and between women medical students and other university women. Women medical students were reported to be more thoughtful and subdued than their male counterparts, yet more apt to be anxious (a factor that correlated with superior academic performance); they were more likely to view themselves as totally committed to their goals and relatively content with them. They were more willing to express their feelings and to admit weaknesses and were less likely than men to endorse the extremes of dominant and aggressive behavior. They were more alert to moral and ethical issues and less apt to convey their own ethical attitudes and values to patients. They had a greater need for personal involvement with patients, although not to the extent of creating an unprofessional approach. Finally, the women medical students valued independence and individuality to a greater degree than men colleagues or other university women.

CONCLUSIONS

Throughout the world, psychiatry is in a period of accelerated transition. In each nation the role of women in psychiatry, and the role of psychiatry in the lives of women, reflect one aspect of the development of egalitarianism. Food limitations, population control, and economic demands linked with laws governing the equality of human rights are powerful reinforcers for increasing the possibility of self-expression by all women. Internationally, the external and internal factors that prohibit or limit women's personal development and vocational participation are being scrutinized. American psychiatry is proving to be a pioneer among medical specialties in this area.

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Lithium in the Treatment of Depression

BY JOE MENDELS, M.D.

The author reviews the studies of lithium as an antidepressant, evaluates the evidence that there might be a specific subgroup of patients for whom it is effective, and reports a study he and his associates conducted that found lithium to be effective for 13 of 21 depressed patients. He concludes that there is convincing, although not conclusive, evidence for an antidepressant effect of lithium and that only by identifying the subgroup of patients for whom it is effective can the continuing uncertainty surrounding lithium's role in affective disorder be resolved. He also calls for a reevaluation of the relationship between mania and depression.

IT HAS BEEN about 8 years since my colleagues and I started to examine the potential antidepressant effect of lithium carbonate. Although the use of lithium for the treatment of depression remains controversial, I think that there is today a reasonable body of evidence supporting the view that it is an effective antidepressant in a subgroup of patients. The precise definition of this group of patients and the determination of whether the lithium by itself is a sufficient antidepressant for all of them remain unresolved.

Given the history of false claims for the use of lithium salts in medicine—including the nineteenth-century claims for its role in the treatment of epilepsy, insomnia, gout, and as a general tonic or sedative—it is not surprising that new claims for its therapeutic usefulness should be treated with reservation, if not outright suspicion.

The claim for an antimanic effect for lithium was first made in 1949 by Cade (1). It took over 20 years for this idea to gain widespread acceptance in North America. The subsequent observation that lithium exerts a prophylactic effect in patients with recurrent affective disorder has achieved acceptance, at least insofar as manic-depressive or bipolar depressive disorder is concerned, in about 10 years—a substantial reduction in lag time. It remains to be seen how long it will take for lithium's role as an antidepressant to be clarified.

I have reviewed the available reports dealing with the antidepressant effect of lithium in detail elsewhere (2, 3) and need only to summarize them here. I will confine myself primarily to the controlled studies and mention the uncontrolled reports only in passing.

I will then briefly evaluate the evidence that there may be a specific subgroup of patients for whom lithium is a useful antidepressant. It is only by the identification of this subgroup of patients that we can expect to resolve the continuing uncertainty surrounding this question. The definition of such a group of patients obviously has important practical as well as theoretical implications.

Finally, I will discuss the theoretical implications of the findings that lithium has a specific antimanic effect and that it may also be useful as an antidepressant. Clearly, this calls for some reevaluation of our conceptualization of the relationship between depression and mania.

REPORTS OF LITHIUM AS AN ANTIDEPRESSANT

Table 1 summarizes 9 uncontrolled studies (1, 4–11). Although no conclusions should be reached on the basis of these reports, it is of interest to note that 6 of the 9 studies listed in this table claimed some antidepressant effects for lithium, while 2 of the 3 negative reports involved early studies with small numbers of patients for which little information is available.

Including the research reported in this paper, there are 10 controlled studies from 8 centers (5, 12–19) in which the effect of lithium alone was tested. Three of these studies (5, 12, 13) claimed no significant antidepressant effect for lithium, while 7 (14–19 and the study reported below) reported a clear-cut effect. These 10 studies are summarized in table 2.

Negative Studies

Of the 3 studies that concluded that lithium did not have any significant antidepressant effect, 1 is an unpublished study cited in a review of lithium (5) and is therefore difficult to evaluate. The 2 more recent studies concluded that lithium has only a "mild antidepressant effect" (12) or was not statistically significantly

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Dr. Mendels is Professor of Psychiatry and Chief, Depression Research Unit, University of Pennsylvania School of Medicine and Veterans Administration Hospital, Philadelphia, Pa. Address reprint requests to him at the Veterans Administration Hospital (151E), Philadelphia, Pa. 19104.

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TABLE 1
Summary of Uncontrolled Studies of Lithium as an Antidepressant

Study	Number of Patients	Patients' Description	Comment
Cade (1)	3	Chronic depressive psychosis	No effect
Noack and Trautner (4)	—	—	No effect
Vojtechovsky*	14	Depression; ECT failure	8 responded
Andreani and associates (6)	24	Severe depression	10 improved
Dyson and Mendels (7)	31	Heterogeneous group of depressed patients	19 improved, especially those with bipolar/ cyclothymic disorder
Van der Velde (8)	—	Mixed affective disorder (bipolar and unipolar); relapsing bipolar disorder	No effect after 14 days' treatment
Noyes and associates (9)	5	Bipolar affective disorder (N=3); unipolar affective disorder (N=2)	4 improved; 2 relapsed with placebo
Náhunek and associates (10)	98	Endogenous or involutional depression	55 improved
Bennie (11)	14	Depression; no response to ECT and/or tricyclics	14 improved

* Vojtechovsky's work, "Zkusenosti s lecbou solemi lithia," was cited by Schou (5).

superior to placebo (13). Examination of these conclusions shows that there *was* a clear-cut improvement in the depressed patients treated with lithium. The group of patients studied by Fieve and associates (12) appears to have consisted primarily of bipolar patients, but there is no information available to allow for this distinction to be made in the group studied by Stokes and associates (13). Furthermore, insufficient information is provided to allow us to determine if the patients received an adequate dose of lithium (with adequate plasma concentrations) for a sufficiently long period of time so that we could be reasonably certain that they received an adequate therapeutic trial. The patients studied by Stokes and associates (13) appear to have received lithium in increasing doses for only 10 days, probably too short a time to allow for an adequate evaluation.

It is possible that the rating scales used by these investigators may not have been sufficiently sensitive. For example, Stokes and associates (13) divided their patients into three categories: improved, worsened, and no change. They did not provide details of the changes in their patients. Thus the clear trend toward improvement in their patients may have been partially obscured by their rating procedures. Their patients did show a statistically significant reduction in depressive symptoms during the 10 days of lithium administration in comparison with depression ratings on the day before the institution of lithium treatment ($p<.005$ by one-tailed Wilcoxon t test).

Fieve and associates (12) reported that there was no significant difference in the responses to lithium and imipramine in their patients after 2 weeks of treatment. It was after this that they found a superior response to imipramine. However, it is important to note that there was a *continuing* improvement in the patients treated with lithium throughout the entire study period.

It seems possible that the relatively negative conclusions regarding the potential antidepressant effect of lithium carbonate reached by these authors may be subject to reinterpretation. It is clear that there was improvement in the patients who were treated with lithium alone; the authors' conclusions may have been affected by any one of several experimental variables. Thus I think that it would be reasonable to conclude that their findings are equivocal rather than negative.

Report of a Recent Study

I would like to briefly summarize our most recent study in this area; this research has not been previously reported.

My associates and I conducted a placebo-substitution controlled study of lithium carbonate in a group of 21 moderately or severely depressed hospitalized patients (their mean Hamilton score [20] at the time of entry into the study was 28.1). Thirteen of the patients were bipolar, and 8 were unipolar. All of the patients received placebo for 7 to 15 days after their admission to the hospital and after the initial medical and psychiatric evaluation. They were then given lithium carbonate in increasing doses to a point of either clinical improvement, a plasma concentration of 1.5 mEq/L, or the development of troublesome toxicity. The optimal or maximal dose of lithium for each patient was maintained for 21 days. This was followed by 7 to 22 days of placebo substitution; neither the patient nor the medical or nursing staff involved in patient management and evaluation were aware of the times when changes were made from placebo to lithium. It is, of course, possible that the patients and/or the staff became aware of the substitution because of subjective or objective effects from the lithium, e.g., slight tremor.

The primary criterion for response was that the patient have sufficient relief of symptoms with lithium that he did not require any other form of active treatment and appeared able to return to his premorbid occupation. Using this criterion plus changes in several rating scale scores—the Hamilton (20), Beck (21), and Bunney-Hamburg (22) scales—13 of the 21 patients improved (9 bipolar and 4 unipolar). Of these 13 patients,

TABLE 2
Summary of Controlled Studies of Lithium as an Antidepressant

Item	Number of Patients	Patients' Description	Research Design	Comment
Studies that found little or no antidepressant effect				
Hansen and associates*	12	Severe endogenous depression	Crossover with placebo	Several patients improved with lithium; only 1 relapsed with placebo
Fieve and associates (12)	29	Bipolar affective disorder	Comparison with imipramine	Imipramine's effect superior to lithium's "mild antidepressant effect"
Stokes and associates (13)	18**	Bipolar affective disorder	Comparison with placebo	Trend toward improvement with lithium, but not significantly superior to placebo after 10 days' treatment
Studies that found significant antidepressant effect				
Goodwin and associates (14, 15)	52	Mixed affective disorder (bipolar and unipolar)	Placebo substitution	15 complete and 21 partial remissions, especially among bipolar patients
Mendels and associates (16)	24	Mixed affective disorder (bipolar and unipolar)	Control group given desimipramine	Lithium as effective as desimipramine
Noyes and associates (17)	22	Mixed affective disorder (bipolar and unipolar)	Placebo substitution	13 responses to lithium (6 of 6 bipolar and 7 of 16 unipolar patients); 9 relapses with placebo
Johnson (18)	10	Endogenous depression	Placebo substitution	5 "marked" improvements
Baron and associates (19)	23	Primary affective disorder	Placebo substitution	5 unequivocal and 5 equivocal responses, especially among bipolar patients
Mendels***	21	Primary affective disorder	Placebo substitution	13 unequivocal improvements; 7 relapses with placebo, especially among bipolar patients

* Unpublished study cited by Schou (5).
** These 18 patients had 31 episodes of affective illness.
*** Reported in this paper.

7 relapsed with subsequent placebo substitution (6 bipolar and 1 unipolar).

Positive Studies

The reports that found that lithium has a significant antidepressant effect are also summarized in table 2. In all, 152 hospitalized patients with moderate or severe depression were included in these 7 controlled studies (14–19 and the study reported above). In all of these studies except 1 (16), the antidepressant effect of lithium was evaluated by placebo substitution. The patients received placebo before and/or after lithium. Most of these studies required that the patient improve with lithium and then relapse with placebo before a determination of unequivocal response was made. In all of the studies a substantial number of patients showed an unequivocal response, and a number of patients showed an equivocal response, i.e., they improved with lithium but did not relapse with placebo.

It should be noted that patients designated as equivocal responders were not necessarily placebo responders. Failure to relapse with placebo substitution, while suggestive, does not in itself mean that the initial response was independent of the pharmacological action of lithium. Lithium *may* have set in motion a train of events that led to the resolution of the illness so that the patient was indeed "cured" at the time of placebo substitution.

Examination of the results of these studies suggests that there is a clear trend for patients with a diagnosis of bipolar or manic-depressive illness to respond to lithium more frequently than patients diagnosed as having recurrent unipolar depression. However, it is also clear that many patients with a diagnosis of unipolar depressive illness respond to treatment with lithium carbonate and that not all bipolar patients respond to this treatment.

There is no one-to-one correlation between the diagnosis of bipolar illness and the antidepressant effect of lithium carbonate; other variables are presumably of importance here. For example, it is known that some patients diagnosed as unipolar may have manic episodes in the future. Thus, even among patients who have had three separate depressive episodes and have

never been manic and are therefore diagnosed as unipolar, 14% will have a manic episode at some time in the future (23) and will have to be rediagnosed as bipolar depressive.

Another possibility is that some unipolar patients may have a positive family history of bipolar illness and may therefore have the genetic endowment for bipolar illness. These patients may best be regarded as pseudo-unipolar depressive, in that they may have the biological characteristics of bipolar illness with a clinical history of unipolar illness.

The fact that not all bipolar depressive patients are lithium responsive is, of course, in agreement with the well-established findings that not all manic patients are lithium responsive and that lithium is not an effective prophylactic agent for all bipolar patients. Similarly, bipolar patients have not been shown to be a homogeneous group in any biochemical or electrophysiological study to date. This clinical syndrome may be biochemically and therapeutically heterogeneous.

Given the limitations and imperfections of our nosological systems, it may be pragmatic to simply refer to a lithium-responsive affective disorder rather than to try to force an artificial or premature fit between therapeutic response and clinical history. Perhaps the systematic study of lithium-responsive patients will eventually reveal some consistent and specific genetic and biological characteristics of this group, allowing them to be separated from the rest of the heterogeneous group of patients with affective disorders. Some preliminary attempts at this are noted below.

COMBINATION LITHIUM THERAPY

Table 3 summarizes 4 reports (24–27) that claimed that lithium exerts a synergistic effect when combined with a tricyclic antidepressant, a monoamine oxidase (MAO) inhibitor, or ECT. Three of these studies (24–26) were uncontrolled. However, they are worth noting because the authors referred to a number of patients who had previously shown a poor response to conventional antidepressant therapy. These observations, together with that of Lingjaerde and associates (27), who reported that lithium plus tricyclics were superior to tricyclics alone in a controlled study, provide provocative but inconclusive grounds to consider combination treatments for treatment-resistant patients. Clearly, there is a need for additional controlled studies of this treatment approach.

TABLE 3
Summary of Studies in Which Lithium Was Used in Combination with Other Antidepressants

Study	Comment
Hartigan (24)*	ECT plus lithium had synergistic effect
Zall and associates (25)*	Lithium plus MAO inhibitors or tricyclics had synergistic effect
Himmelhoch and associates (26)*	Severely depressed patients with hypersomnia improved with lithium plus tranylcypromine; no response to initial tricyclic treatment
Lingjaerde and associates (27)**	Lithium plus tricyclics had synergistic effect, especially when lithium plus imipramine-*N*-oxide used for endogenous depression

*Uncontrolled study.
**Controlled study.

CHARACTERISTIC FEATURES OF LITHIUM RESPONDERS

The following variables have been reported to be associated with the antidepressant effect of lithium: manic-depressive or bipolar illness (7, 15); family history of bipolar illness (7); "endogenous" symptom pattern (7); increased average evoked responses (19); "high" erythrocyte (RBC) lithium:plasma lithium ratio (28); "high" baseline plasma calcium:magnesium ratio (29); initial increase in plasma magnesium and calcium concentration with lithium treatment (29); and reduced accumulation of 5-hydroxyindoleacetic acid in lumbar spinal fluid (30).

It is important to note that all of these claims (with the possible exception of an association with bipolar illness) must be regarded as preliminary. However, it is of interest to note them in light of the possibility that there may be a specific subgroup of patients with a lithium-responsive disease. These claims may point to further studies that would help define the characteristics of this group of patients.

Of the symptomatic-historical features, the possible association between lithium response and bipolar illness has been discussed above. The reported association with an "endogenous" symptom pattern (7) is probably not specific for lithium-responsive patients because this symptom complex is relatively common and is probably associated with a response to other forms of treatment, including ECT (31) and tricyclic antidepressants (32).

The reported association between baseline plasma calcium:magnesium ratio as well as changes in plasma calcium and magnesium with the initiation of lithium treatment (29) is promising. If this idea is confirmed, it will provide a simple method for selecting patients for lithium treatment. However, only a preliminary report of this finding has appeared to date (29); it is therefore difficult to evaluate. Further investigation is clearly needed.

Frazer and I (28) reported that depressed patients who respond to treatment with lithium carbonate have a significantly higher RBC lithium:plasma lithium ratio than patients who do not respond but who receive a similar or even higher dose of lithium and achieve similar plasma lithium concentrations. There is evidence (33) that RBC lithium concentration has a significantly

higher correlation with brain lithium concentration than does plasma lithium. This suggests that greater accuracy in predicting brain lithium concentration is achieved from a knowledge of RBC lithium concentration than from knowing plasma lithium concentration.

For example, the predictability of brain lithium concentration from a knowledge of RBC levels ranges between 64% and 88%, contrasting with the predictability of brain lithium levels from a knowledge of plasma levels, which ranges from a low of 17% to a maximum of 62%. Thus, if one can extrapolate from our studies with rats (33), it is possible that patients with a higher RBC lithium concentration also have a higher central nervous system concentration of lithium than patients with low RBC lithium concentrations.

The higher RBC lithium:plasma lithium ratio in depressed patients who respond to treatment with lithium is probably a function of a proportionately higher RBC lithium concentration. This suggests that patients who respond to treatment with lithium carbonate may have some difference in cell membrane properties governing the entry of lithium into or out of the cell in comparison with patients who do not respond to treatment with lithium (34). This difference in RBC lithium: plasma lithium ratio is unlikely to be a function of variations in dietary sodium intake or plasma hormonal concentrations (34).

This phenomenon has several other interesting characteristics. The RBC lithium:plasma lithium ratio appears to stabilize fairly early in treatment and to show proportionately little change over time. Thus we rarely see "high ratio" patients become "low ratio" patients or vice versa. Frazer and I (34) also found that there is a statistically significant tendency for the RBC lithium: plasma lithium ratio to be higher in patients with a higher baseline RBC sodium concentration. Although lithium itself appears to cause some increase in RBC sodium concentration, this increase is more marked and more likely to occur in depressed patients who show a response to lithium treatment (34). No significant changes have been noted in potassium or magnesium concentrations.

In related studies (35, 36), my associates and I have found that RBC lithium concentration is at least in part under genetic control and that the factors which regulate the distribution of lithium are similar to those which regulate the sodium flux across the cell membrane. This suggests the possibility that there may be some genetically determined abnormality in sodium and lithium flux in patients who are lithium responsive and that lithium may serve as a useful marker in the identification of differences in cation transport in discrete groups of patients.

RELATIONSHIP BETWEEN DEPRESSION AND MANIA

In my opinion, the evidence for an antidepressant effect of lithium carbonate reviewed in this report is convincing if not yet conclusive. As noted earlier, the fact that a single drug, lithium, exerts both antidepressant and antimanic effects raises important questions about the relationship between depression and mania and suggests that they may not be two distinctly opposite conditions (37–39). The term "bipolar," implying two opposite states, may be a misnomer. Like many terms introduced into psychiatric nosology, it may carry greater implications than were originally intended. Indeed, there are a number of reports (2, 40–56) indicating that depressed and manic patients share important characteristics and are not simply opposite states. Although some of these remain controversial observations, they do indicate the importance of reevaluating the relationship between these two syndromes:

Symptoms. Many manic patients have concurrent symptoms of depression while in the manic state, including sadness, pessimism, guilt, self-doubt, crying, and suicidal preoccupation (40, 41).

Biological changes. Investigators have reported that some depressed and manic patients differ from control subjects in the following systems: reduced CSF concentration of 5-hydroxyindoleacetic acid (42, 43), reduced platelet MAO activity (44), reduced catechol-O-methyltransferase (COMT) activity (45), disrupted pattern of cortisol release into plasma (46), reduced transfer of Na_{22} from plasma to CSF (47), reduced CSF tryptophan concentration (48), increased amplitude response in cortical evoked potential (49), and similar alterations in sleep EEG in the two groups of patients (50).

Treatment response. Some manic and depressed patients may respond to the following: lithium carbonate (2, 51), ECT (52), tryptophan (53), imipramine (54, 55), and chlorpromazine (56).

It is also possible that lithium may exert its therapeutic action through different mechanisms in depression and mania (and perhaps through yet another mechanism when used prophylactically). Alternatively, lithium may act on the same systems by exerting a nonspecific homeostatic effect, bringing an elevated or a reduced activity back to some "normal" range. This would be a parsimonious explanation for its several, apparently different clinical effects.

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A Review of Major Implications of the *O'Connor v. Donaldson* Decision

BY LOUIS E. KOPOLOW, M.D.

Although the Supreme Court's decision in O'Connor v. Donaldson *is narrow from the legal perspective, it will have wide clinical applications for psychiatry if it reflects a trend in future court decisions. The author assesses the impact of this decision on the mental health profession in terms of the issue of dangerousness as grounds for involuntary commitment for psychiatric treatment, the principle of least restrictive alternative to hospitalization, the question of adequacy of treatment in light of the absence of nationally defined standards, and the personal liability of physicians for their professional actions.*

THE O'CONNOR V. DONALDSON DECISION (1, 2) marks the first time that a Supreme Court decision recognized the legitimacy of judicial involvement in activities previously considered to be the exclusive domain of psychiatrists. It also marks the first review by the court of the judiciary process by which an individual is civilly committed to a state hospital. In this article I will attempt to assess both the clinical and forensic implications of the decision and address some possible programmatic outcomes.

HISTORICAL BACKGROUND

Kenneth Donaldson, a 67-year-old ex-patient of the Chattahoochee State Hospital in Florida, successfully waged a legal battle for his freedom in the Supreme Court of the United States. Donaldson was initially committed to the hospital in 1957 at the request of his parents, who were concerned about his mental state. Despite his repeated pleas that he was not dangerous or insane and requests for court intervention, Donaldson remained confined at the hospital for 15 years. Attempts to obtain his release by a friend and by workers at a Minnesota halfway house were blocked by Donaldson's doctor. In 1971, after the retirement of Dr. J.B. O'Connor, clinical director and later superintendent of the Chattahoochee State Hospital during Donaldson's confinement, Donaldson was released from the hospital.

A federal court jury awarded Donaldson $38,000 in damages for the invasion of his civil rights by Dr. O'Connor and Dr. John Gumanis of the hospital staff. This decision was upheld by the U.S. Court of Appeals for the Fifth Circuit and was appealed by O'Connor to the Supreme Court, which heard the case on January 15, 1975. The Supreme Court ruled in favor of Donaldson's claim that his constitutional right to freedom had been denied by O'Connor but remanded the case to the lower court on the issue of damages.

ASPECTS OF THE CASE

The importance of the *O'Connor v. Donaldson* decision is reflected in the holdings of the case (the conclusion of law reached by the court as to the legal effect of the facts of the case), the dicta of the case (opinions of a judge that do not embody the resolution or determinations of the court), and the significance of the issues in law and mental health touched on by the suit. The holdings in a Supreme Court decision have the force of law everywhere in the United States, but the dicta merely indicate the underlying philosophy behind the decision and give some indication of how future suits might be handled by the court.

The holdings of the Supreme Court's decision on June 26, 1975, were narrow in the legal sense; however, the dicta and issues examined have the potential to produce wide-ranging philosophical and programmatic effects on the providers and recipients of mental health care in future litigations. This potential impact on the mental health system can best be understood by examining four important issues: 1) the issue of dangerousness as grounds for involuntary commitment, 2) the principle of least restrictive alternative to hospitalization, 3) the question of adequacy of treatment, and 4) the personal liability of physicians for their actions.

Dangerousness and Commitment

The term "dangerousness" has frequently been used in legal and psychiatric circles to describe the

Dr. Kopolow is Staff Psychiatrist for Patients' Rights and Advocacy Programs, Division of Mental Health Service Programs, National Institute of Mental Health, Alcohol, Drug Abuse, and Mental Health Administration, 5600 Fishers Lane, Rockville, Md. 20852.

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concept of likely or actual harmful acts directed either at oneself or others. The limited holding of the court in *O'Connor v. Donaldson* is that "a State cannot constitutionally confine, without *more* [emphasis added], a non-dangerous individual who is capable of surviving safely in freedom by himself or with the help of willing and responsible family members or friends."

This ruling does not directly apply to those who might be considered dangerous by a psychiatrist, to those who might be dangerous to themselves, or to those who are incapable of surviving without assistance. Even more restrictively, it does not apply to individuals who are confined and given something more, presumably certain forms of treatment, even though they might be quite capable of surviving and present no threat to the safety of others. The court also seemed to sanction a broad definition of dangerousness as applied to civil commitment when it declared the following dicta:

> Of course, even if there is no foreseeable risk of self-injury or suicide, a person is literally dangerous to himself if for physical or other reasons he is helpless to avoid the hazards of freedom either through his own efforts or with the aid of willing family members or friends.

Such a definition might permit the continued hospitalization of organically impaired mentally ill individuals (those with organic brain syndrome) who, although they are not dangerous, are not able to avoid the "hazards of freedom." It remains for future court decisions to clarify this issue.

The Supreme Court's decision addressing the issue of dangerousness and confinement will have a rather limited effect on the mental health system. Despite some early predictions that thousands of patients would be released from state hospitals as a result of the rulings, according to a telephone survey of selected state mental health facilities conducted in August 1975, it is possible that only a very limited number of individuals meet the requirements of 1) being nondangerous, 2) receiving only custodial care, 3) able to survive the hazards of freedom through the aid of family or friends, and 4) held involuntarily in a facility. Patients who meet all of these criteria should be discharged quickly.

From the clinical and forensic psychiatric perspective, the *O'Connor* decision regarding dangerousness poses some very difficult problems. The state of the art is such that psychiatrists and other mental health professionals can describe behavior and offer dynamic theories about the etiology of emotional problems in court reasonably well, but they have considerable difficulty agreeing on diagnoses and predicting such future behavior as dangerousness or even the ability to "survive the hazards of freedom."

Predictions of dangerousness are with few exceptions predictions of rare or infrequent events for which there are no reliable base rates. As noted in the APA task force report *Clinical Aspects of the Violent Individual* (3), even if an index of violence proneness could be developed that correctly identified 50% of the individuals who will commit violent acts, the continued employment of such an index would identify 8 times as many false positives as true positives.

In spite of the difficulty in predicting future behavior, the *O'Connor* decision seems to be moving the courts in the direction of requiring a determination of dangerousness as the sole grounds for involuntary commitment. The potential of this trend raises some very serious philosophic questions about the participation of psychiatrists in the court system. If the psychiatrist's prediction of dangerousness is little more reliable than a layman's judgment of whether an individual will commit a criminal act, the use of criteria for involuntary commitment based on the prediction of dangerousness is as inappropriate in the mental health sphere as preventive detention is in the legal system. The mental health and legal systems must therefore determine what if any grounds are sufficient to involuntarily commit an individual for treatment and what minimum degree of reliability should be required for such commitment (4). If psychiatrists participate in proceedings involving involuntary commitment, they should make known at the outset the problems inherent in any prediction of dangerousness.

Least Restrictive Alternative

Another issue raised by the court's decision in *O'Connor* concerns the nature of the treatment that a patient receives and the location at which he receives it (hospital versus community). The court acknowledged that states have a legitimate interest in providing care and assistance to patients, but it also declared that the patients' preferences should be recognized as well. The court expressed the following dicta:

> The mere presence of mental illness does not disqualify a person from preferring his home to the comforts of an institution. Moreover, while the States may arguably confine a person to save him from harm, incarceration is rarely if ever a necessary condition for raising the living standards of those capable of surviving safely in freedom.

This section of the *O'Connor* decision will very likely produce controversy on two gounds. The first involves the question of whether an individual's mental status is of such importance to the state that his condition justifies intervention and potential deprivation of freedom. The second question is whether the decision has any bearing on the concept of least restrictive alternative to hospitalization.

The court's assertion that a state may not confine an individual for the purpose of raising his living standards reflects a changing philosophy about both mental illness and the practice of psychiatry. For hundreds of years many have believed that mental illness categorically prevents an individual from accurately perceiving reality and making reasoned judgments. Therefore, it was considered the state's duty according to

the principle of parens patriae to take care of such afflicted individuals, treat them for their illnesses, and protect society from their potentially harmful actions (5).

Consistent with this legal view has been the widely accepted psychiatric view that life and health, or physical and emotional well-being, are at the pinnacle of any hierarchy of values and should be maintained at any cost—even if the cost is a considerable loss of liberty for the individual whose health is at stake. A corollary to this position is the belief that madness and other forms of mental illness are diseases or processes that so impair an individual's judgment and capacity for responsible action in relation to himself and others that his statement of his own will is no longer valid. When the patient declares that he refuses hospitalization and treatment his wishes might very well be discounted and viewed as symptoms of his mental illness.

A countervailing philosophy is shared by a growing number of lawyers and mental health professionals concerned with civil rights. This view maintains that although a person's physical and mental health are important, they are not necessarily of the highest value, and that the freedom of the individual to place a higher value on other things should be respected. Those espousing this view maintain that what is called "mental illness" is not a process that necessarily interferes with or invalidates a person's will or lessens his responsibility to himself and others (6). Even a psychotic individual should have his wish to live at home rather than in a state mental hospital taken into consideration by the judges and psychiatrists who determine his fate.

The Supreme Court's decision in *O'Connor* reflected the view that confinement that is not demonstrably necessary and consistent with the principle of using the least drastic means necessary would not be sanctioned. Justice Stewart cited *Shelton v. Tucker* (7), in which it was noted that even when the government has shown that it has a valid reason or purpose for limiting or overriding an individual's constitutional rights, it must still show that the means it has chosen to accomplish that purpose will not limit the individual's rights any more than is absolutely necessary.

The court's movement toward a standard of ability to survive and the expectation that the least drastic means of treatment will be used will have the potential of producing some significant changes in psychiatric practice. The psychiatrist will have to modify his approach to involuntary treatment of nondangerous individuals and seek to encourage rather than coerce patients to follow a recommended regimen. In addition, least restrictive alternatives to hospitalization must now become a major consideration in the development of any proposed treatment plan.

The community-based least restrictive alternative approach to treatment might be given a boost by such court decisions as *O'Connor v. Donaldson* if the phrase "willing and responsible family members or friends" is interpreted as applying to such facilities as community mental health centers. Some legal experts (8) have raised the issue of whether the inadequacy of these centers to meet the demands placed on them or the nonexistence of these centers might be used as an excuse for confining an individual in an inpatient setting because he might be viewed as helpless to avoid the hazards of freedom. Another point affecting community-based treatment that needs further clarification concerns the term "willing." Although the Supreme Court declared in *O'Connor* that "mere public intolerance or animosity cannot constitutionally justify the deprivation of a person's physical liberty," one must wonder if such existing criminal laws as vagrancy or such civil statutes as zoning codes might be used to remove the mentally ill from the community. One example of this possibility is the suit of *Stoner v. Miller* (9) in Long Beach, N.Y.

Adequacy of Treatment

Another issue raised by the *O'Connor* decision that will likely be reflected in future court decisions concerns the definition of what should constitute adequate treatment. The court declared, "There is, accordingly, no occasion in this case to decide whether the provision of treatment, standing alone, can ever constitutionally justify involuntary confinement or if it can, how much or what kind of treatment would suffice for this purpose?"

Although the court thus avoided the question of whether the provision of treatment would be acceptable grounds for commitment, it expressed the dicta that "it is plainly unacceptable to suggest that the courts are powerless to determine whether the asserted grounds [treatment] is present." Some have argued that the court's statement in this instance opens the door for future judicial decisions as to what does or does not constitute adequate treatment. Another view is that the court did not refer to a judicial prerogative of determining whether the treatment given represents the best possible care but only if such treatment is possible (10).

Whichever turns out to be the accurate perception of future court rulings, it is clear that the mental health profession's role will undergo considerable change. No longer can the professional base his decisions as to the most appropriate treatment modality solely on clinical grounds. He must become cognizant of such issues as whether the patient will be viewed as dangerous by a judge or jury, whether the recommended program will be considered adequate treatment or even treatment, and whether he will be considered liable for denying the patient his constitutional rights if he continues to hold him in the hospital because he considers him dangerous if a future court hearing finds the patient not dangerous.

Other concerns for the physician will be whether he has followed due process procedures or obtained truly informed consent from the patient and what his liability will be if he releases an individual because he seems to meet the *O'Connor* criteria but the patient later com-

mits a dangerous act (11). If past trends continue, psychiatrists would rather refuse to treat involuntary patients than face the risks of litigation. State facilities, many of which are already overcrowded and understaffed, may find that mental health professionals, who are already feeling uncomfortable about treating involuntary patients, may be even less willing to work in such situations in the future as a result of recent litigation.

Part of the dissatisfaction of mental health professionals in various state and public hospitals is that the quality of treatment and care provided in these institutions is not as high as it should be. In the absence of nationally accepted standards, each state and sometimes each hospital can determine the nature of the care to be provided. The courts have felt justified in moving into the vacuum of lack of national standards to assure the rights of involuntarily committed mentally ill individuals. In cases such as *Wyatt v. Stickney* (now known as *Wyatt v. Aderholt*) (12–14) and *Welsch v. Likins* (15), the judges have consulted with various professional organizations, taken expert testimony, and come up with what they considered minimum standards. These standards tend to be more of the mortar and bricks and staff:patient ratio variety than to deal with actual quality of treatment.

For example, Judge Johnson in the *Wyatt* case handed down a two-part order on April 13, 1972, that set standards for mental disorder and mental retardation facilities and provided a detailed program for implementation (13). These minimum standards promulgated by the court include, among others, the following: a provision against institutional peonage; a number of protections to ensure a humane psychological environment; minimum staffing standards; provision for a human rights committee at each institution; detailed physical standards; minimum nutritional requirements; a provision for individualized evaluations of residents, habilitation plans, and programs; minimum staff:patient ratios; and a requirement that every mentally impaired person has a right to the least restrictive setting necessary for treatment.

The relative inability of psychiatrists and other mental health professionals to demonstrate the relationships among hypothesized etiologies of illnesses as well as their responses to treatment has impeded the development of agreed-upon standards of quality care and treatment and forced closer assessment of the nature of the activities mental health professionals can perform (16). To the extent that the mental health professions can become accountable and responsible for the assessment of need through prescreening, continuous monitoring of treatment, and evaluation of aftercare programs they can be freed from the restrictions and limitations of externally imposed standards.

The increasing pressures of the courts in such decisions as *O'Connor* will begin to move the mental health field into more intensive review of its activities and the development of acceptable standards (8). Although the initial response of mental health professionals might be to consider total withdrawal from law and mental health (no court involvement, no treatment of involuntary patients, etc.) it is to be hoped that this will be only a momentary reaction.

As a physician, the psychiatrist recognizes that his primary duty is to help the sick. Therefore, although he may resent and disagree with the encroachment of the law on what he has traditionally seen as his domain, he will continue do his job as best he can. The psychiatric profession should clearly explain to the courts what it can and cannot do in diagnosing and treating a patient and avoid the trap of being considered omniscient or omnipotent. The court, on its part, must realize that in the overwhelming number of interactions between patient and doctor the intent is to help, not harm, the recipient of care. Only through a realistic understanding of the goals and capacities of mental health professionals can the courts make appropriate determinations concerning such issues as grounds for commitment, the nature of quality care, and physicians' liability.

Physicians' Liability

One of the issues of the *O'Connor* decision that produced great interest among American psychiatrists is the fact that Donaldson not only sued for release but also sought money damages against his doctors. Due to the inexactness of medical science, the severe clinical repercussions for error in treatment, and the detrimental effect on their practices because of bad publicity, physicians as a group tend to be fearful of lawsuits. Donaldson's suit was particularly frightening for physicians because the nature of his charges (violation of his constitutional right to liberty) would obviate the possibility that malpractice insurance could cover any part of the damages. The charge also introduced a new area of accountability for which psychiatrists are unprepared.

Federal law provides that monetary damages are a proper remedy when a person is deprived of civil rights by someone under "cloak of State law." The Supreme Court did not uphold the assessment of monetary damages against O'Connor, but it declared that the issue should be reexamined by the circuit court in light of the new immunity standards enunciated recently in *Wood v. Strickland* (17):

> Whether O'Connor knew or reasonably should have known that the action he took within his sphere of official responsibility would violate the constitutional rights of Donaldson or if he took the action with the malicious intention to cause a deprivation of constitutional rights or other injury [to Donaldson] (2).

The Supreme Court qualifier "for purposes of this question, an official has, of course, no duty to anticipate unforeseeable constitutional developments," seems to indicate that the court is leaning toward the view that O'Connor should not be held liable. However, it clearly puts physicians on notice that from now

on if they confine or retain in custodial care a person who is not dangerous to himself or others and is capable of living safely in an available community alternative, even if he requires some support, they may be held liable for money damages. This decision is another warning to psychiatrists that they must reexamine their activities in light of evolving constitutional developments and not assume that their previous role as instruments of the state's parens patriae activities will continue unchanged.

CONCLUSIONS

As noted in the beginning of this paper, the *O'Connor v. Donaldson* decision is narrow from the legal perspective, but it will have wide clinical implications if it reflects a trend in future court decisions. The court's recent movement into this area after so many years of all but ignoring involuntarily committed mentally ill patients signals the need for a reexamination of mental health activities and closer cooperation and understanding between the legal and mental health systems. Only when the mental health profession fully understands what the court's expectations are concerning its relationship with patients and the court comprehends the potential and limitations of psychiatry vis-à-vis patients can the needs of both fields be adequately served.

On the issue of dangerousness, the court should be aware that even mental health professionals, who consider themselves students of behavior, have considerable difficulty predicting dangerousness. Therefore, to use dangerousness as the primary grounds for involuntary commitment is to place a heavy burden on a slender reed.

On the issue of least restrictive alternative, most mental health professionals would applaud closing antiquated state hospitals and replacing them with community-based alternatives. However, it remains for the government to consider mental health a sufficient priority to allocate the needed funds. Until such time, "least restrictive alternative" may simply be rhetoric for no treatment at all.

Concerning adequacy of treatment, the mental health profession should not be cowed by the beginning of court intervention in this area, but it should recognize it for what it is—the court moving in to fill the vacuum of inadequate standards of care and treatment created by the mental health profession itself. In the past the courts promulgated standards of care only when such standards were entirely lacking or ignored. Although the courts would prefer that the mental health profession set adequate standards of its own, the trend in the future appears to be increasing involvement of the courts in improving the adequacy of care and treatment.

The concept of the liability of physicians for their actions is an old one, but it has a new twist in that psychiatrists can now be found liable for money damages for doing what they have been encouraged to do for hundreds of years, i.e., confining mentally ill individuals. How the mental health system and the legal system grapple with and attempt to resolve this and other issues will dramatically affect all aspects of the practice of psychiatry.

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Marijuana Effects on Simulated Flying Ability

BY DAVID S. JANOWSKY, M.D., MARTIN P. MEACHAM, M.S., JACK D. BLAINE, M.D., MICHAEL SCHOOR, AND LOUIS P. BOZZETTI, M.D.

The authors studied the effects of marijuana intoxication on the ability of 10 certified airplane pilots to operate a flight simulator. They used a randomized double-blind crossover design to compare the effect of active versus placebo marijuana. They found that all 10 pilots showed a significant decrease in measurements of flying performance 30 minutes after smoking active marijuana. For a group of 6 pilots tested sequentially for 6 hours, a nonsignificant decrease in flying performance continued for 2 hours after smoking the active drug. The authors conclude that the effects of marijuana on flying performance may represent a sensitive indicator of the drug's psychomotor effects.

ALTHOUGH THE USE OF MARIJUANA by the young adult population of the United States has dramatically increased in the last decade, virtually no information is available concerning its use by airplane pilots (1–3). Our informal inquiry has revealed that "social" marijuana smoking is not an uncommon practice among pilots, some of whom reported that they have flown aircraft while "high" on marijuana. For this reason, we felt that it was relevant to study the effects of smoking social doses of marijuana on the ability to operate an airplane flight simulator.

METHOD

We studied the effects of marijuana intoxication on the ability of certified pilots to operate a general aviation model ATC-510 instrument flight simulator (Analog Training Computers, Inc.). Seven professional and 3 private male pilots were recruited for the study. All had smoked marijuana socially for several years. Their ages ranged from 21 to 40. Three could be described as infrequent marijuana users (at the time of the study they were smoking marijuana twice a week or less), and 7 could be described as moderate marijuana users (they were smoking marijuana three or more times a week).

The pilots were familiarized with four consecutive 4-minute "holding pattern" sequences requiring a total of 16 minutes' "flight time," which included maneuvers typically encountered in instrument flight (e.g., straight and level flight, turns, three-dimensional maneuvering, and radio navigation). These tasks require psychomotor coordination as well as such cognitive abilities as short-term memory, concentration, and orientation in time and in three-dimensional space.

As described in appendices 1 and 2, the first and fourth 4-minute flight sequences consisted of a standard holding pattern. The second and third sequences consisted of a standard holding pattern modified by incorporating altitude changes. A low level of "turbulence" was also added to all four sequences so that the pilot would be required to continually manipulate the controls to compensate for the effect of the turbulence. These changes were carefully chosen to demand a high level of flying skill to correctly complete each sequence.

After the 10 pilots had attained proficiency in operating the simulator and in performing the four specific flight sequences, marijuana containing 2.1% Δ-9-tetrahydrocannabinol (Δ-9-THC) was administered in a dose of .09 mg of Δ-9-THC per kg. One week before or after active drug administration, a matched placebo containing a nonpharmacological amount of Δ-9-THC was administered. The pilots smoked the active or placebo marijuana in a pipe for a 10-minute period using a standardized smoking technique that consisted of the following: 10 seconds of inhalation, 20 seconds of holding breath, 5 seconds of exhalation, and 5 seconds of normal breathing. A randomized double-blind crossover design was used for the administration of active and placebo marijuana.

Thirty minutes after smoking active or placebo marijuana the 10 pilots performed the flight sequences. Their pulse rates and their self-ratings of degree of intoxication were recorded at this time. For the self-rating of degree of intoxication, each pilot was asked to

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The authors are all with the Department of Psychiatry, University of California, San Diego, School of Medicine, La Jolla, Calif. Dr. Janowsky is Associate Professor of Psychiatry and Chief of Psychiatric Services, University Hospital, 225 West Dickinson St., San Diego, Calif. 92103. Mr. Meacham and Mr. Schoor are Research Assistants and premedical students, Dr. Blaine is a resident in psychiatry, and Dr. Bozzetti is Clinical Associate Professor.

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The marijuana and placebo used in this study were supplied by the National Institute on Drug Abuse of the Alcohol, Drug Abuse, and Mental Health Administration, Rockville, Md.

rate his "high" on an open-ended scale on which 10 was designated as his usual social high and 0 represented his normal state.

We videotaped each pilot's flight sequence after both active and placebo marijuana smoking as well as just before smoking to establish baseline levels. These videotaped 16-minute flights were analyzed at 10-second intervals for deviation from the standardized flight pattern, altitude deviation, and heading deviation. When appropriate, the course deviation indicator (CDI), a radio navigation instrument, was examined at 5-second intervals. Total heading, CDI, and altitude deviations were calculated for each 16-minute flight sequence for each subject.

In addition, the pilot's "major errors," defined as those which if committed in the actual flight situation would take the airplane out of its designated air space with potentially dire consequences (e.g., getting lost, fuel exhaustion, stalling, and gross altitude or navigational deviations), were transcribed from the videotaped sequence and summed. The pilots' "minor errors," defined as altitude deviations greater than plus or minus 100 feet or heading deviations greater than plus or minus 30°, were also observed on the videotape, transcribed, and totaled.

The performances of 6 of the pilots were also evaluated 2 hours, 4 hours, and 6 hours after smoking. Each of these pilots was used as his own control in analyzing the effects of active versus placebo marijuana in altering flying ability (by sign test).

RESULTS

Eight of the 10 pilots were able to distinguish the placebo from the active marijuana. The 2 who erred in identifying the drugs were the least experienced in using marijuana. Most of the pilots rated their level of intoxication after smoking the active drug as slightly greater than their usual social high.

Average data for the entire group of pilots will be presented rather than data for individual performances because individual performance varied considerably from pilot to pilot and from variable to variable. As shown in table 1, the self-rating of degree of intoxication was significantly higher after smoking active marijuana than after smoking placebo ($p<.01$, sign test). Most of the pilots subjectively felt that the flying task was more challenging when they were intoxicated on active drug and did not believe that they had adequately compensated for any drug-induced deficiencies.This feeling was confirmed experimentally: all of the pilots evidenced a significant decrease ($p<.01$, sign test) in performance on all measurements evaluated during the flight after smoking active marijuana (see table 1).

The average pulse rate observed after placebo smoking was significantly lower than that observed after active marijuana smoking ($p<.01$, sign test) (see table 1). The entire group also demonstrated a statistically significant increase in the average number of major errors ($p<.01$, sign test) and minor errors ($p<.01$, sign test) after smoking active marijuana (see table 1).

TABLE 1
Average Pulse Rates, Self-Ratings of Degree of Intoxication, and Flight Performances of 10 Airplane Pilots 30 Minutes After Smoking Active or Placebo Marijuana

Item	After Smoking Active Marijuana	After Smoking Placebo Marijuana
Pulse rate	107	73
Self-rating of degree of intoxication*	10.5	0.5
Altitude (feet)**	2,615	680
Heading (degrees)**	627	332
CDI (units)**	100	42
Number of major errors	2.9	0.4
Number of minor errors	4.5	0.7

* 10=usual social high; 0=normal state.
** Values indicate total deviation from assigned flight path during entire 16-minute flight sequence.

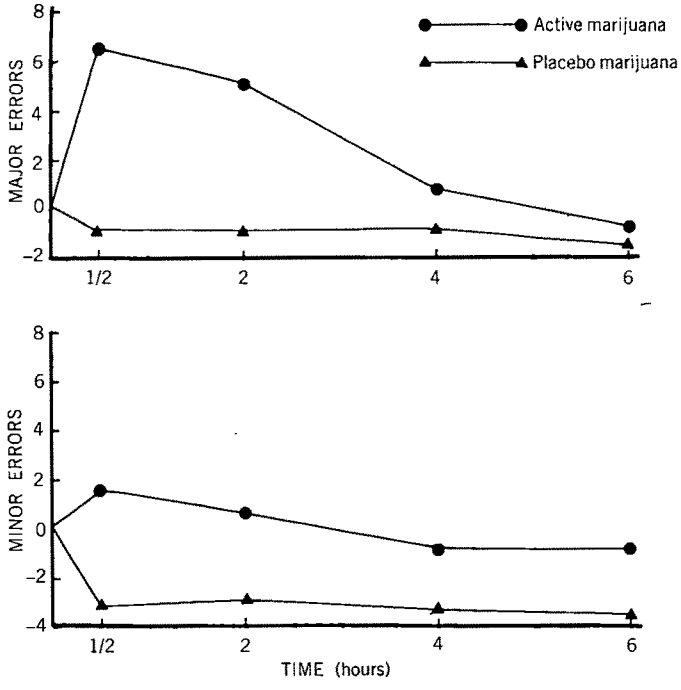
Data were also obtained for 6 of the pilots over a 6-hour period after smoking active drug or placebo to elucidate the time course of the marijuana-induced deterioration in flying performance observed initially at 30 minutes. The flying performances of these 6 pilots were relatively consistent over the 6-hour period after smoking placebo, suggesting that neither learning nor fatigue significantly affected flying performance (by sign test). A decrease in flying ability, not statistically significant, was apparent for most of the 6 pilots 2 hours after smoking active marijuana, but this diminished in magnitude when compared with the statistically significant decrease noted at 30 minutes. The performances of all 6 of these pilots returned essentially to baseline levels by 4 hours (see figures 1 and 2).

DISCUSSION

These results have a number of implications, both for aviation medicine and for the understanding of marijuana's pharmacologic properties. The level of marijuana intoxication in these 10 pilots was generally self-rated as equal to the level they reached when they were socially intoxicated. The time course of the effects of marijuana also followed that encountered when marijuana is smoked socially (1, 4). The results indicate that smoking marijuana in doses used socially (5) causes significant deterioration in simulated instrument flying ability for at least 30 minutes in experienced pilots. The effect probably lasts for 2 hours and disappears in 4 hours.

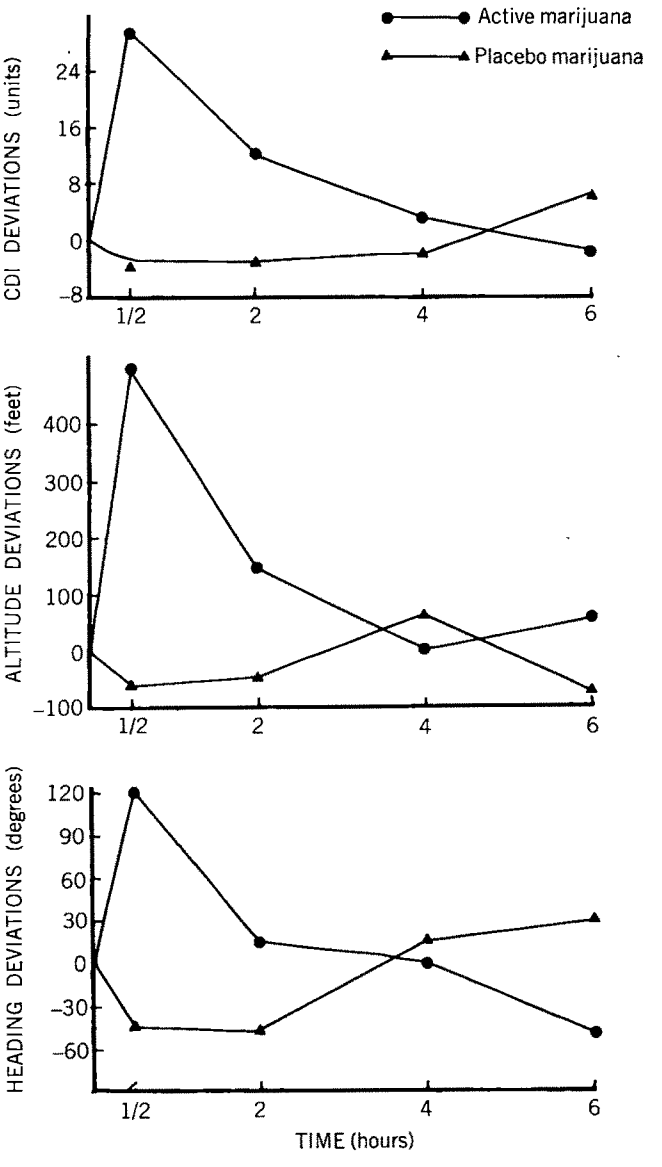
Although the gross detrimental effects of marijuana appear to last for less than 4 hours, more subtle effects, detectable with more sophisticated equipment, may conceivably persist for longer periods of time in actual flight situations or more complex simulated con-

FIGURE 1
Average Number of Major and Minor Errors* of 6 Pilots After Smoking Active or Placebo Marijuana



*The number of errors represents the average number made by the 6 pilots after smoking compared with the average number they made before smoking (baseline levels) during an entire 16-minute flight sequence.

FIGURE 2
Average Deviations from Baseline Levels in Flight Performances of 6 Pilots After Smoking Active or Placebo Marijuana



ditions. Furthermore, the effect of altitude and pressure changes upon the performance of pilots intoxicated with marijuana represents a variable not measured in our experiment. Our data do not support safe instrument flight for at least 4 hours after smoking marijuana.

It is important to note that our flight task was relatively simple and that only a limited number of variables were evaluated. We did not record certain measurements, such as airspeed control, angle of bank, and rate of control movement, in our simulated flight situation, although these measurements were part of the simulated flying task. Although the results noted are quite dramatic, one must note that simulated flying differs from actual flying. Our pilots performed a memorized flight sequence; they had the instructions for the pattern in front of them at all times. In actual flight situations, instructions come sequentially from an Air Traffic Control (ATC) controller and must be accurately noted and repeated (i.e., read back) by the pilot. We believe that the performance of a pilot under these circumstances would be even more adversely affected by marijuana intoxication than in our experimental setting. The level of motivation to perform optimally and the ability to use some visual and kinesthetic cues and radar vectoring by ATC differ in actual flight situations from the simulated flight situation (6).

The deficiencies noted in the performances of our 10 pilots probably reflect marijuana's ability to affect a complex, learned psychomotor test involving memory, skill, concentration, sense of time, and orientation in three-dimensional space and in the performance of multiple complex tasks (5–10). Operationally, the simulated flying task is probably best described as a four-dimensional, complex cognitive tracking test.

It appears to us and to the pilots we tested that the major problems incurred in flying the simulator while intoxicated with marijuana involved certain specific factors. The most important appeared to involve marijuana's ability to affect short-term memory and sense of time. After smoking the active marijuana, the pilots often forgot where they were in a given flight sequence or had difficulty recounting how long they had been performing a given maneuver, in spite of the presence of written instructions and a stopwatch.

Marijuana also appeared to cause alterations in concentration and attending behavior: the intoxicated pilots would concentrate on one variable to the exclusion of other variables or perhaps attend to intrusive thoughts. As an example, several pilots noted that following a momentary lapse in attending to the flying task, they could not tell how long they had been flying or where they were located in the flight sequence. The realization that such inappropriate focusing had occurred seemed to lead to overcontrolling (oversteering) in an attempt to compensate for variables previously ignored and suddenly noted. Thus subjects exhibited a complete loss of orientation at times with respect to the navigational fix, resulting in grossly unpredictable flight performances. This loss of orientation occurred when the pilots were either daydreaming, "lapsing," or focusing on a certain part of their specified routine to the exclusion of other required activities.

We feel that the effects of marijuana on flying performance may represent a rather sensitive indicator of marijuana's psychomotor effects. Because of its multiple requirements, the task of flying may accentuate marijuana's cognitive disruptive effects (5–10) and thus prove useful in evaluating marijuana's interactions with other psychoactive drugs, such as alcohol and barbiturates, which also affect piloting performance (11–15). Thus the use of trained pilots to perform a complex psychomotor task may be a sensitive "bioassay" of marijuana's effects on cognitive functioning that has implications beyond those obvious to the safety of the airplane pilot.

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APPENDIX 1
Standard Holding Pattern

Assuming a few simplifications, a standard holding pattern is oval in shape and is composed of four 1-minute segments, starting at a total elapsed time of 0 minutes, 0 seconds.

The first segment is a right-hand turn, performed at a standard turn rate of 3° per second. This turn is executed on reaching a navigational "fix," a locus defined variously by radio beacon, triangulation, and radar. Thus a standard turn lasting 1 minute results in a heading change of 180°, causing a course reversal (total elapsed time=1 minute, 0 seconds).

The second segment, called the "outbound leg," is a straight and level flight lasting 1 minute (total elapsed time=2 minutes, 0 seconds).

The third segment, also lasting 1 minute, is a right turn, ideally executed at the standard rate of 3° per second. However, the last portion of this turn, encompassing approximately the last 20 seconds, is normally used to align the aircraft on the proper course with respect to the navigational fix. The aircraft has now assumed the original heading (total elapsed time=3 minutes, 0 seconds).

The fourth segment of a standard holding pattern, called the "inbound leg," employs radio navigation to enable the pilot to fly the aircraft accurately to the initial navigational fix. This also takes 1 minute and concludes the standard 4-minute pattern (total elapsed time=4 minutes, 0 seconds).

In actual flight conditions, the pattern may be repeated many times and may incorporate climbs and descents to effect traffic separation, etc. In the experimental situation, the following criteria are employed: climbs—100% power, indicated airspeed (IAS)=120 miles per hour (mph), and climb rate=1,000 feet per minute; descents—50% power, IAS=160 mph, and descent rate=1,000 feet per minute; and standard turn rate—3° per second or 180° per minute.

APPENDIX 2
Specific Experimental Patterns

PATTERN 1

In the task demanded of the pilots, the first pattern flown is a standard holding pattern lasting 4 minutes, as defined in appendix 1. The pattern is flown at a target altitude of 2,000 feet.

PATTERN 2

On crossing the navigational fix at the conclusion of the first pattern (total elapsed time=4 minutes, 0 seconds), the flight plan calls for a climb of 1,000 feet to the 3,000-foot altitude at a rate of 1,000 feet per minute, while simultaneously executing a standard right-hand turn of 180°. Since the maneuvers in both geometric planes take 1 minute, the pilot is required to simultaneously level the aircraft at 3,000 feet

while rolling out of the turn on the appropriate reciprocal heading (total elapsed time=5 minutes, 0 seconds).

The aircraft is then flown on the reciprocal heading at a level altitude of 3,000 feet for 30 seconds, at which time (total elapsed time=5 minutes, 30 seconds) a descent of 1,000 feet to an altitude of 2,000 feet is begun. The standard holding pattern requires that a standard right turn begin 30 seconds later (total elapsed time=6 minutes, 0 seconds). The aircraft is to be leveled at 2,000 feet in the middle of this turn (total elapsed time=6 minutes, 30 seconds). The turn is completed after a total elapsed time of 7 minutes, 0 seconds, with the aircraft aligned with the inbound course to the navigational fix at an altitude of 2,000 feet. The aircraft then flies inbound toward the fix for 30 seconds at 2,000 feet (total elapsed time=7 minutes, 30 seconds), and a climb of 500 feet is instituted with the aircraft simultaneously reaching the navigational fix, leveling at 2,500 feet, and rolling into the standard right turn of the next pattern after a total elapsed time of 8 minutes, 0 seconds.

PATTERN 3

The first 30 seconds of this pattern is flown at an altitude of 2,500 feet while the standard-rate right turn is executed. At a total elapsed time of 8 minutes, 30 seconds, a climb of 1,500 feet to an altitude of 4,000 feet is begun. The aircraft is rolled out of the turn (total elapsed time=9 minutes, 0 seconds) and flown straight on the outbound leg while climbing to 4,000 feet. This altitude is reached at a total elapsed time of 10 minutes, 0 seconds, at which time a turn of 180° is started. Thirty seconds later (total elapsed time=10 minutes, 30 seconds) a descent of 1,000 feet to 3,000 feet is initiated. At a total elapsed time of 11 minutes, 0 seconds, the aircraft is once again positioned on the inbound course to the navigational fix. The pilot continues inbound toward the fix while continuing the descent to 3,000 feet. The descent is terminated at 3,000 feet (total elapsed time=11 minutes, 30 seconds), and the aircraft is assumed to reach the navigational fix at a total elapsed time of 12 minutes, 0 seconds.

PATTERN 4

The last pattern is a standard holding pattern flown at a level altitude of 3,000 feet. The experiment is terminated with the completion of this pattern after a total elapsed time of 16 minutes, 0 seconds.

ized by unremitting anorgasmia in the context of ceaseless extramarital relationships with older men is probably fixated at the oedipal level. Her disorder would not be informational or interpersonal. Psychological ontogenesis clarifies the fact that while this patient's difficulties may present as dissatisfaction with her spouse and appear to be interpersonal, they have the dynamic underpinning of a neurosis. To treat a neurotic disorder as an interpersonal one is to deny effective treatment.

TREATMENT TECHNIQUES

Considering the variety of sexual disorders, the spectrum of etiologies, and sexual ontogeny, no one treatment modality is sufficiently broad to cover all circumstances. Each treatment modality has its specific applications and limitations. In the following section I will discuss general categories of available treatment techniques; I have tried to be complete without being judgmental about efficacy, appropriateness, or compatibility with ethical standards.

1. *Dynamic psychotherapies*. As a general class, these techniques are based on psychoanalytic theory and observation. Core concepts include an ontogenetic psychological-developmental sequence, psychic structure, psychic conflict, defense mechanisms, the active unconscious, and transference development. Psychoanalysis, psychotherapy, and many group and family techniques are based in this framework.

2. *Behavioral modification techniques*. These techniques have their theoretical foundation in learning theory. Disturbed function is viewed as dependent upon disordered learning, conditioning, or habit. Behavioral modification through systematic desensitization and reciprocal inhibition is viewed as the therapeutic goal. The techniques developed by Masters and Johnson are classified here since they establish a hierarchy of sexual behaviors and use systematic desensitization as well as reciprocal inhibition. Related techniques include aversive conditioning, biofeedback, etc. Some forms of "masturbation therapy" (23) would be included here.

3. *Educational techniques*. This includes efforts at education as the primary focus. Lectures, audiovisual materials, the conjoint physical examination, and other methods are used in demonstrating sexual anatomy, physiology, or practice.

4. *Physical manipulation techniques*. The use of surrogates, the "sexological" examination (involving stimulation of the patient by the therapist), and techniques in which sexual manipulation is carried out or closely supervised by the therapist are included.

5. *Medical or surgical procedures*. The diagnostic physical examination and specialized surgical, pharmacological, hormonal, and neurological techniques are included in this category.

While not every specialist will be equally competent in each major category and may wish to avoid using some for ethical, medicolegal, or transference reasons, at a minimum the complete specialist will be familiar with the indications and contraindications for each technique and the indication for referral.

Educational techniques may be used for those patients whose malfunction or sexual hesitancy is related primarily to lack of information or experience. Behavioral modification techniques are useful for those patients whose disorders are in the interpersonal or situational realm or whose conflictual dysfunction is limited rather narrowly to the sexual disorder and is relatively free of continuing dynamic determinants from childhood conflict.

The dynamic psychotherapies are indicated for the treatment of neurotic, characterological, borderline, and psychotic disorders having sexual dysfunction as one component. This is not to say that behavioral or educational techniques may not be useful in a supplementary way, so long as the transference is protected by having them implemented by another therapist (7).

Medical and surgical techniques, of course, are clearly indicated in those disorders with an organic basis. Other treatment techniques may be necessary in a complementary or supplementary role. Directly manipulative techniques may conceivably be useful in some situations. However, transference and ethical considerations make their utility questionable.

ETHICAL STANDARDS

Ethics are extraordinarily important in the treatment of sexual disorders. In essence, ethical standards boil down to the rule that the patient is not exploited either vicariously or directly. This puts limitations on observation, recording, filming, and direct physical manipulation. Some practices, such as therapist participation in the sexual act, therapist nudity, and the use of surrogates are, to my mind, unethical. Perr (24) has outlined the potential legal hazards of the direct involvement of therapists in the treatment of sexual disorders. Aside from the potential medicolegal complications, direct therapist participation in sexual activity is unwise because it completely ignores the transference meaning of such involvement and the consequent deleterious effect on treatment (25).

DIDACTIC COMPONENTS OF TRAINING

Didactically, there are two major areas to be covered. The first deals with treatment techniques, and the second with developmental sexuality.

A firm grounding in the conceptual and theoretical bases of treatment techniques is essential. When the theoretical grounding is absent, treatment techniques are applied pro forma rather than with discrimination. For example, the Masters and Johnson techniques may be applied by rote, without the therapist's realization that they establish an anxiety hierarchy and trade

must be taken into consideration. As Stoller (13) observed, the syndromic classification of psychological disease states must be complemented by the dynamic and etiological.

Even though a complete outline of etiological factors in sexual disorder is beyond the scope of this paper, it is clear that not all cases of impotence are comparable. The same manifest disorder may have different etiological roots, which will determine the treatment most profitably employed. To return to the pneumonia analogy, specific treatment requirements are based upon the patient's inherent susceptibilities (which would vary if he is alcoholic, has obstructive bronchitis, etc.) and the particular pathogen involved (pneumococcus, staphylococcus, Klebsiella, etc.).

Some functional sexual disorders are based on lack of information, experience, and opportunity. There are others based on the interpersonal relationship and communication between partners. Still others are based on childhood experience, with arrest of psychological development. In my experience the most common pattern is some combination of all three. It is a discrimination of considerable finesse, at times, to decide which is the most important factor and the most amenable to intervention.

The etiological categories of sexual dysfunction outlined below are organized at a middle level, dynamically speaking, as a compromise between manifest symptomatology (at the most superficial level) and an individual psychogenetic formulation (at the most molecular). It is recognized that these clinical categories overlap and that there are gray areas between them. Nonetheless, this is a pragmatically useful scheme since it is directly related to the categories of available treatment techniques.

1. *Information-lack of experience-deficit sexual disorders*. These disorders are based in lack of experience, maladaptive education, or misleading information without significant basis in neurotic or characterological disorder or in the interpersonal aspects of the relationship.

2. *Interpersonally based sexual disorders*. These disorders are related to current interpersonal relationships or situations without significant basis in neurotic or characterological disorder.

3. *Neurotically and characterologically based sexual disorders*. These disorders have their basis in an internalized conflict, manifested by either symptoms or behavior, and are relatively independent of current interpersonal and cultural conditions. Excluded from this category are the sexual disorders based on a borderline or narcissistic modification of character.

4. *Borderline or narcissistically based sexual disorders*. These are sexual disorders having their basis in the relatively stable borderline or narcissistic personality disturbances (14–16). Many deviations have their basis in these personality modifications. In particular, the gender dysphoria syndromes are most often seen in severely borderline individuals.

5. *Psychotically based sexual disorders*. These are sexual disorders related primarily to the profound psychological regression and personality fragmentation of psychosis or prepsychotic states. The sexual disorders in these instances may involve any sort of sexual behavior, but the basic issue is the fragmented personality.

6. *Organically based sexual disorders*. These are disorders related to genetic, physiological, and/or anatomical impairment, with or without functional overlay. Impairment may be occasioned in fetal life, by disease states, as a complication of other treatment, or by trauma.

Complementing the mid-level classification of sexual disorders is the concept of developmental lines. In conceptualizing developmental lines, Anna Freud (17) spoke of an unfolding maturational sequence from infantile emotional and physical dependence to adult sexual and work relationships, stressing interplay among predisposition, environment, and already achieved levels of mastery and integration. A simplified developmental line for sexuality, with elements borrowed from a number of authors, is as follows:

1. Fetal development, with the differentiation of internal and external sexual structures, the establishment of cyclic versus acyclic gonadotrophin release, and active versus passive patterns of energy expenditure (18).

2. The establishment of satisfactory symbiosis as a basis for comfortable and trusting intimate human relationships (19).

3. "Separation-individuation," establishing the capacity to relate to another individual as a separate and real person (19).

4. Gender identity formation (20, 21).

5. The elaboration and working through of the oedipal, triangular, competitive relationship.

6. Disavowal of sexual drives during latency.

7. The resurgence of biological sexual drives with reproductive capacity at adolescence.

8. Later in adolescence, the consolidation of genital primacy as a basis for intimacy.

9. Marriage and the establishment of committed intimacy.

10. Reproduction and child rearing in which all the previous issues may be reworked.

(For a further elaboration of these developmental issues, particularly 5–10, see reference 22.)

The mid-level etiological classification system provides a general orientation to the broad spectrum of sexual disorders, whereas a firm grounding in developmental sexuality provides the best framework vis-à-vis the individual patient.

Sufficiently careful history taking to identify the developmental areas in which the individual patient had difficulty, became fixated, or was arrested will allow placement in the appropriate category of sexual disorder. For example, a female patient whose parents were divorced when she was age 5, whose mother subsequently had multiple lovers, whose adolescence was a model of propriety, but whose adult life is character-

shibboleths that are assumed to describe: 1) a problem area in some way intrinsically different from other emotional or physical disorders; 2) treatment techniques that may be applied across the board with little concern for differential diagnosis, etiology, or prognosis; and 3) a group of practitioners for whom traditional values of training and experience have only limited application.

As commonly used, the term "sex therapy" implies that the patient's "sex" can be treated with little concern for the complexities of constitutional endowment, physical health, developmental history, intrapsychic conflicts, or capacity for sublimation.

THE MEDICAL/PSYCHIATRIC APPROACH TO TREATMENT

A number of authors have addressed the issues of constitution, physical health, and psychodynamics in the treatment of sexual disorders.

Masters and Johnson have emphasized the physiological underpinning of sexuality (1) and the importance of this knowledge in the application of their treatment techniques (2). They have also stressed the importance of a screening or diagnostic physical examination.

Among the psychoanalytic authors, Kestenberg (7), Fink (8), and Stoller (9) have emphasized the continuing relevance of dynamic concerns in sexual disorders and have attempted to amalgamate physiological information and short-term techniques within the psychodynamic framework. On a more pragmatic level, Kaplan and Kohl (10) have emphasized the possibility of untoward reactions in patients receiving short-term treatment unless there is adequate consideration of the role of the sexual dysfunction in intrapersonal and interpersonal dynamics. Meyer and associates (11), in their application of the Masters and Johnson techniques, noted a tendency for transference to develop, intrapsychic conflict to be mobilized, and the more pervasive characterological and neurotic roots of sexual disorder to become apparent. These observations indicate that judicious treatment of sexual disorders requires due consideration of physical health, personal history, intrapsychic dynamics, and the current context of the individual's life.

THE MEDICAL CONTEXT

In a medical context there is emphasis on the distinction between treatment (with its medicolegal safeguards and long tradition of responsibility) and counseling, giving advice, or lending a sympathetic ear. A medical context implies familiarity with all categories of sexual disability, not merely one subclass of syndromes. A medical context implies that appropriate and skillful attention will be paid to the issues of evaluation, disposition, and prognosis. This implies the capacity to discriminate among organic conditions, functional conditions, organic conditions with a functional overlay, and organic conditions in which the functional aspect is not an overlay but an intrinsic part of the disease process. A medical context implies skill in a broad range of treatment techniques from the most elemental to the most sophisticated. A medical context also implies that based on evaluation, etiology, anticipated management problems, and prognosis, a treatment modality is selected that promises to have the best results in the individual situation. In other words, there is no a priori commitment to one treatment modality.

To emphasize the point, an analogy seems apt. The pneumonias usually require bed rest and hospitalization as common basic factors in treatment. On the other hand, the selection of particular antibiotic and supportive measures depends on an appreciation of distinct etiologies and prognoses. In general, gram-negative pneumonia does not respond well to simple penicillin.

THE SCOPE OF MANIFEST CLINICAL SYNDROMES

The disorders and problems that should be familiar to a medical subspecialist in the field of sexual disorders are as follows:

1. *The common sexual disorders*, e.g., impotence, premature ejaculation, anorgasmia, dyspareunia, vaginismus, and withdrawal from sexual activity.
2. *The uncommon disorders*, e.g., ejaculatory incompetence and sexual anesthesia.
3. *The paraphilias*, for example, exhibitionism, pedophilia, and voyeurism.
4. *The homosexual syndromes*, both male and female.
5. *The gender dysphoria syndromes*, e.g., so-called "transsexualism" and its variants (12).
6. *The physical developmental disorders*, for example, penile agenesis, hypospadias, and intersex conditions.
7. *Disease-related disorders*, including impotence secondary to diabetes or a temporal lobe disorder as well as some dyspareunias.
8. *The iatrogenic disorders*, i.e., those secondary to psychotropic or diuretic agents or surgery.
9. *The traumatic disorders*, e.g., penile or vulvar injuries.

ETIOLOGICAL CLASSIFICATION OF SEXUAL DISORDERS

The foregoing review of manifest disorders is useful for appreciating the spectrum of sexual disabilities. As medicine learned long ago, however, treatment cannot be based solely on the manifest presentation of disease. Etiological factors and the structural modifications occasioned by the pathological process also

Training and Accreditation for the Treatment of Sexual Disorders

BY JON K. MEYER, M.D.

The field of "sex therapy" is not defined by specific, limited techniques. Rather, it encompasses a subspecialty based on an understanding of psychodynamics and psychological development but requiring additional skills from other branches of medicine. Despite its breadth and complexity, sex therapy training is unregulated, with the public lacking the protection afforded by standardized training programs or certification. The author outlines the scope of sex therapy as a psychiatric and medical subspecialty and delineates the necessary clinical skills for the physician-practitioner.

THE PHYSIOLOGICAL research (1) and new treatment techniques (2) of Masters and Johnson created a healthy ferment in the conceptualization and treatment of sexual disorders. These researchers view sexual disorders as the result of poor education, unfortunate social circumstances, and unhappy interpersonal relationships. They view treatment in a didactic, educational, systematic desensitization framework, with the exclusion of the issues of psychodynamics and transference, the traditional preserves of psychiatry. Their enthusiasm, published results, deemphasis of psychodynamic factors, and nonpsychiatric background provided a model and a rationale for the treatment of psychologically based sexual disorders by a variety of different physicians and allied health professionals.

This process is illustrated by the recent formation of the Eastern Association for Sex Therapy, with membership open to the medical specialties, psychology, and the allied health professions. One purpose of this organization was to formalize standards for sex therapists through its admission requirements (3). The extent of the process is illustrated by the recent decision of the American Association of Sex Educators and Counselors to certify "sex therapists" rather than to accredit "sex counselors" (4). On the positive side, both organizations are concerned with the issues of credentials and accreditation in the field of "sex therapy," a field in which there are few guidelines and many abuses.

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Dr. Meyer is Director, Sexual Behaviors Consultation Unit, Johns Hopkins Medical Institutions, Baltimore, Md. 21205, and Assistant Professor of Psychiatry and Surgery, Johns Hopkins University School of Medicine.

There is no quarrel with the efforts of these organizations to establish credentials and standards of practice for their members and to certify that these standards are met. However, it is important to avoid what Romano called "false egalitarianism" that obscures "the differences in education, knowledge, skill, and responsibility between professionals and paraprofessionals in the interdisciplinary care of patients" (5, p. 433). General standards compatible with training and experience in the allied health professions are not sufficient standards for physicians.

The extent of poorly qualified or frankly unqualified therapist participation in the treatment of sexual disorders is largely known from anecdotal reports or from interviews with surrogates in the popular literature. In every locale, however, there are practitioners whose training and experience, not to mention ethical standards, are open to question. In a recent article Masters was quoted as saying, "of all the clinics in operation in the United States today, at best 50 might be considered legitimate. The rest are frauds" (6). Masters made a strong case for the necessity of medical coverage and responsibility as an irreducible minimum for legitimate clinics.

Medical coverage and responsibility are the minimal requirements for bona fide practice. The characteristics and standards of that medical practice, however, must be defined.

A problem in defining proper training and accreditation for physicians specializing in the treatment of sexual disorders is that the scope of the field is undefined. It is essential to reach a general definition of the field in order to know what one is training and is accredited *for*. After that, a training program and accreditation standards almost automatically fall into place. In certain centers the practice of sex therapy is limited to the treatment of couples using the techniques developed by Masters and Johnson. Other centers focus on physical disorders and use medical and surgical approaches. Still others view the sexual disorder as a secondary symptom and focus on neurotic or characterological features, using psychodynamic approaches. Some integration of these various approaches is clearly required.

"SEX THERAPY"

In attempting to define the field, the terms "sex problems," "sex therapy," and "sex therapist" immediately cause problems. These terms have become

heavily on systematic desensitization. Without this realization, the practitioner cannot select out those components of the method which are essential and those which are not, and modifications are made on personal whim rather than on considered judgment. Similar issues may be raised regarding dynamic techniques. Transference development, elucidation, and interpretation are designed to resolve pathological formations that are concretized in intrasystemic conflict. When intrasystemic conflict is minimal in comparison to the habitual or interpersonal aspects of sexual disorder, dynamic techniques are less useful and less cost effective than education or behavioral modification.

It is also essential to have a thorough grounding in the developmental aspects of sexuality, including psychosexual (intrapersonal and interpersonal), cultural, endocrinological, physiological, anatomical, and neurological factors. Didactic review would include fetal development, the intersex conditions, and related anomalies affecting sexual functioning (26); gender development (27, 28); the role of hormones (29, 30); anatomical factors in sexual relationships (31); physiological factors (1); and cultural aspects of sexual activity (32, 33). (These references are intended merely as examples of the sources that might be read and assimilated.)

CLINICAL COMPONENTS OF TRAINING

Clinical experience may be incorporated into residency training. Subspecialty training, however, is viewed here as requiring extended postresidency affiliation with an active program, allowing a focus on evaluation, diagnosis, prognosis, treatment selection, management, and treatment of a broad variety of sexual disorders, with extensive supervision. This may be done on a part-time basis, making it feasible for the physician responsible for his own livelihood.

ACCREDITATION

Membership in any of the organizations for sex therapists does not adequately fulfill the requirements for standards and experience for psychiatrists and other physicians. These standards cannot be sufficiently exacting by virtue of the broad membership. The organizational issues of accreditation for psychiatrists would need to be considered within the structure of the American Psychiatric Association or under the aegis of the American Board of Psychiatry and Neurology.

CONCLUSIONS

Medical sex therapy is not defined by specific techniques, such as those developed by Masters and Johnson. Rather, the field is defined by the required competence and orientation of the practitioner. As in other areas of medicine, the orientation of the practitioner is toward evaluation, differential diagnosis, prognosis, and treatment selection from among the available spectrum of therapeutic modalities. Suitable competence of the subspecialist in sexual disorders necessarily involves a broad basis in theoretical and developmental issues, as well as experience in treatment application.

Sexuality in its manifold aspects is at the very core of psychiatric practice. As Freud (34) observed in 1905, anyone who is in any way abnormal in mental life will also show an abnormality in sexual life. Clinical experience since then has confirmed that notion. Because of long clinical experience with such phenomena and a theoretical framework within which to conceptualize these findings, psychiatry is in a primary position to contribute to the treatment of sexual disorders. Psychiatrists, however, are also physicians. And within the framework of medical practice is the concept of having a therapeutic armamentarium to be used judiciously according to assessment of the patient.

Medicine, by virtue of its long concern with evaluation, prognosis, disposition, management, treatment, and follow-up, is the best arena for the treatment of sexual disorders. Within medicine the experience, theory, and broad perspective of psychiatry will allow leadership, if psychiatry will take it.

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Change in 1976 Annual Meeting Site

The APA Executive Committee decided to move the May 10-14, 1976 APA Annual Meeting from Atlantic City to Miami, Florida, after hearing the APA Meetings Management Department report that it had ascertained that a major Atlantic City hotel had inadvertently committed several hundred rooms to another organization during the week the APA meeting was scheduled. This would have left APA with the unacceptable alternative of using a myriad of scattered small hotels in the Atlantic City area. It has been confirmed that Miami Beach can accommodate the meeting.

Further arrangements and developments will be reported in future issues of *Psychiatric News*.

The Family in Exile: Cuban Expatriates in the United States

BY RUBÉN D. RUMBAUT, M.D., AND RUBÉN G. RUMBAUT, M.A.

For the nearly one million Cuban exiles in this country, the ordeal of expatriation has meant anguish and uprootedness, challenge and accomplishment. Several factors account for the comparatively successful aspects of their struggle: relatively high occupational and educational levels, formation of vigorous communities that permit the maintenance of a positive ethnic consciousness and the creation of strong social ties, and effectively organized reception by the United States. Expatriation is always traumatic and produces "casualties," but mastery of the struggles it involves can lead to personal growth and expanded horizons. The Cuban expatriate experience shows that the influx of large numbers of refugees can be a creative and enriching process for both the host country and the individual refugee.

JANUARY 1, 1959, WAS the beginning of an accelerated, conflictual, and explosive political process on the island of Cuba. As a result of this process, an increasingly large number of Cubans who rejected the legitimacy of the new regime are now in exile. Seventeen years have passed, and the exodus has not ceased. Cuban expatriates (who now number more than a million) have taken refuge in all parts of the world, but the vast majority have come to the United States.

The magnitude of this radical dislocation of individuals and families should be considered in perspective. When the exodus began in earnest in 1960, the population of Cuba had just reached 6 million. Today there are more than 10 million Cubans, 9 million living on the island and the rest, mostly exiles and their offspring, living abroad. During this period, roughly 10% of the Cuban people have been severed from their homeland as political refugees.

According to James L. Canlin, Deputy Coordinator for Humanitarian Affairs, Office of Refugee and Migration Affairs, U.S. Department of State, 654,314 Cubans arrived in the United States from January 1959 to December 1974. For a variety of reasons (a discussion of which is beyond the scope of this paper), this official statistic probably underestimates the actual extent of the expatriation. Moreover, this figure obviously does not include the many thousands of Cubans who had come to the United States from the turn of the century through 1958, nor the children born to the exiles from 1959 to date, nor the refugees who have come during 1975 and early 1976. The population of individuals of Cuban origin in the United States consists of between 100,000 and 200,000 families dispersed throughout the 50 states and Puerto Rico. Areas with the largest Cuban population are southern Florida, New York-New Jersey, southern California, and Illinois.

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Dr. Rumbaut is Associate Professor of Psychiatry, Baylor College of Medicine, Houston, Tex., and Director, Day Hospital, Veterans Administration Hospital, 2002 Holcombe Blvd., Houston, Tex. 77031. Mr. Rumbaut is Lecturer, Department of Sociology, San Diego State University, San Diego, Calif.

THE EXILE PROCESS

The experiences that led large sectors of Cuban society into expatriation were complex. The best available research, conducted by a group of American sociologists (1), identified seven major types of motives: 1) temporary imprisonment, 2) fear or threat of imprisonment, 3) harassment and persecution, 4) forcible attempts at integration into the revolution, 5) loss of jobs, possessions, or sources of income, 6) disagreement with governmental action deemed arbitrary, and 7) refusal to accept a Communist regime. Thus a wide range of pragmatic and ideological considerations characterized the decision to leave Cuba, including a perceived threat to basic religious values, family life, and liberal democracy. In pointing to the political character of the Cuban exodus, Fagen and associates stressed that,

> It would be a mistake to think of the exiles as alienated from their homeland in the sense that we think of certain youth, minorities and intellectuals as alienated in other countries. . . . The typical refugee affirms a "higher loyalty" to Cuba, to *la patria*. . . . He is in exile from the government in power; he is in exile from Cuba only insofar as the Cuba that he knew and loved has, in his view, been betrayed and despoiled. (1, p. 103)

Chronologically, Cuban expatriates have come to the United States in four distinct phases:

1. January 1959 to October 1962: daily commercial flights between Cuba and the United States were terminated after the latter date.
2. October 1962 to December 1965: refugees arrived in small boats or through third countries.

3. December 1965 to April 1973: an airlift provided two daily flights from Varadero to Miami.

4. April 1973 to the present: refugees continue to arrive in the United States through third countries, in small boats, and from a backlog accumulated in some foreign nations.

The exiles who came during the first phase were disproportionately drawn from the urban middle and upper classes of Cuban society, including those in the higher educational and occupational brackets (2), whereas those who have arrived during the last decade have been increasingly representative of all sectors of Cuban society. However, the available data throughout have precluded any easy explanations of the exile process solely in terms of economic interests or any other unidimensional concept of a multidimensional problem.

From a psychosocial perspective, a recent study (3) has distinguished three types of expatriate groups: 1) those who hope for a quick return, typifying the early exiles (from whom the participants in the 1961 Bay of Pigs invasion were recruited), 2) those who escape from dangerous or uncertain circumstances, more numerous after the invasion and during the second phase, and 3) those who seek improved economic opportunities, more typical of later emigrants who evidence the frustration of their personal aspirations for the future. In general, according to this study, the first two types were "pushed out" by political conditions, whereas the third type was "pulled out" by economic concerns and the hope for self-advancement and a better life.

There are other alternatives to the classification of the refugees' experience that may help to underscore the diversity of these individuals and their circumstances. One study indicated, for example, that the adaptation process has been more difficult and demanding for Cuban women than men, particularly in the case of unaccompanied refugee women (4). Age is another important factor: children are more flexible, young and middle-aged adults have to assume an inordinate amount of responsibility, and older people generally do not have to work but are less flexible, sense the loss of their homes more acutely, and evidence a reduced capacity to learn a new language and achieve a satisfactory degree of autonomy.

One of the most dramatic groups of refugees consists of an estimated 15,000 children who came to this country without their parents (5). There were many reasons why this happened, but the primary explanation is bureaucratic: when many parents panicked and tried to take their families out of Cuba, children under 16 were permitted by the U.S. government to come on waivers of visas, while their parents were required, as is customary, to have visas. The Cuban government also made it easier for the very young and the very old to leave, while working adults or men of military age faced many more obstacles. As a result, hundreds of children remained separated from their parents, sometimes for several years. These children were placed in institutions, foster homes, relatives' homes, and orphanages in 110 cities in 40 states.

Despite their diversity, Cuban refugees (like all refugee groups) share in a common process of expatriation and subsequent adaptation. In this sense we can describe the refugee as a social type and discuss the prototypical experience of exile for analytical and heuristic purposes.

PSYCHOLOGICAL ELEMENTS OF THE EXILE EXPERIENCE

The refugee coming to a new country goes through an extremely complex and intense experience that, when analyzed, breaks down into numerous components. These components can, however, be described using as a metaphor Janus, the Roman god of beginnings. Janus was always depicted at the gate of the cities and had two faces, which enabled him to see in opposite directions simultaneously. This image illustrates the plight of the refugee. The face that looks back sees displacement, separation, uprooting, loss, nostalgia, and, in a certain sense, even death, because some things die inside us when we are forced to abandon our homeland without the possibility of returning at will. The face that looks forward sees new horizons, unknown environments, strangers with unfamiliar customs and languages, real and imaginary perils, a vigorous challenge to survive, adapt, and grow, and even the opportunity of constructing a new identity in sudden anonymity. Thus, psychologically speaking, the refugee's experience combines elements of premature death and rebirth, a peculiar process in which he is both conscious protagonist and conscious spectator.

A conflict of loyalties ensues from the moment the thought of departing enters the mind of the would-be expatriate. The decision to choose—or reluctantly accept—exile entails an inner agony between those forces that bind and those that expel a person from his land. After the initial decision is acted on, the dilemmas that accompany it persist for years. First and foremost one must survive, which channels the decisions and experiences of the refugee along available structures of opportunity. Then comes the agonizing arrangement of priorities, the careful selection among narrow options. Should one focus on an ideological struggle to the exclusion of everything else? Should one settle in Miami, the point of arrival for most refugees, or resettle elsewhere—an alternative that represents a sort of second exile? Is occupational retraining in one's field feasible, or must one switch occupations? How will one fulfill the obligations to family, work, and to a society that offers generous asylum? The choice seems to be "adaptation or ghetto" (6).

Forced uprootedness from one's community is the salient feature of exile. Azcárate (7) has described the psychiatric symptomatology of Cuban expatriates (when it is manifested) as an expression of an overwhelming sense of anxiety, depression, and inability to

cope with the emotional problems presented by the anomic circumstances of exile. This is primarily experienced as an attitude of helplessness, a feeling that nothing can be done. Separation from the family, length of exile, and return to Cuba are all beyond one's control, at least psychologically. The wish is to return to a Cuba that no longer exists.

In the first weeks of expatriation, mild paranoid attitudes (a carry-over) are relatively common; this is followed by a transient sense of relief, relaxation, and even euphoria. Then, the slow realization of the painful limitations and long struggles ahead begins; anxiety mounts and depression deepens. There are few refugees who do not bear deeply ingrained psychological scars. Still, a new path is slowly opened, much as an explorer cuts his way through a dark jungle toward distant filtered light. A shelter of some sort is found, a small income is secured, children register in school, and some acquaintances are made. From then on, the process is one of gradual rebirth.

Unexpected and paradoxical advantages attend the experience of exile. Culture shock deroutinizes the flow of everyday life. One feels strangely free of ties, attachments, social inanities, trivia, and the rituals of participation in countless formal and informal organizations that characteristically consume much time and effort. One discovers that old routines and conventions can be discarded and new perspectives and values can be incorporated. One suddenly recognizes common origins in the many shared elements of the new and the old culture. Indeed, it is one of the ironies of exile that the very circumstance of uprootedness permits a rediscovery of the familiar—of the family and of the most fundamental meanings and functions of human community. One encounters at once the universality and relativity of being human.

Perhaps a reactive overcompensation to the prolonged feelings of helplessness, sadness, loneliness, estrangement, and handicap—combined with added free time and the need to concentrate rigorously on "making it" in lean and austere circumstances—explains why, beyond the numerous casualties that this process inevitably produces, many of the "survivors" excel in their endeavors. All of their inner resources have been fully tested, their dormant qualities and potentials have been evoked, and their purposes and goals have been challenged. For those able to cope successfully with the exceptional demands, a more fulfilling and productive life may lie ahead. After all, a victory is rewarding in direct proportion to the amount of personal effort it requires. A refugee—devoid of citizen status, not belonging to any group, and lacking influence and connections, property and savings, and language and cultural skills—feels somehow alone and bare in an unconquered environment. He has to reconstruct his life, even to the point of learning to communicate again. If he eventually masters the environment, his self-reliance grows, and the strengthened motivation and sense of purpose will predictably exceed the average level.

The experience of a shared crisis may itself facilitate the development of a stronger family bond, affording a common cause around which a more lasting cohesiveness can be built within this interpersonal supportive family network. The children, who are still flexible, malleable, curious, and thirsty for discovery, adapt amazingly well. They learn the new language with no accent and the new customs without hesitation. We feel that it is a myth that bilingualism should be avoided during the period of transition. In fact, the Cuban student not infrequently rises to the top of his classes in spelling, grammar, reading, and writing in English, while keeping an adequate Spanish vocabulary. Penfield (8) has proposed the concept of the "uncommitted cortex" during childhood and encouraged people to learn different languages at an early age. This is in complete accord with our experience.

SOCIAL DETERMINANTS OF THE EXILE EXPERIENCE

For the Cuban exiles, the process of creating new beginnings has occurred not amidst stable conditions in the receiving society but amidst the social and political upheavals that have characterized U.S. history during the last 15 years. We can therefore identify diverse sources of stress. The exile process during the sixties and into the seventies in the United States constituted a double source of marginality and role strain—culture shock was combined with future shock, entailing not only the problem of adaptation to the new society but of coping with rapidly changing conditions that even its own native population found difficult to cope with. Further, these structural sources of stress have been reflected within the family in both the well-known generation gap and in parent-child conflict arising from differential rates of adaptation to the new culture. This "adaptation lag" may be manifested, for example, in a child's embarrassment over his parents' occasional cultural faux pas or in the parents' insistent expressions of concern over the child's increasing "Americanization." In some important respects, the clash of the exiled youth is not so much with the American culture as with the Cuban, particularly in situations where friendship networks consist almost exclusively of primary relationships with English-speaking peers. Such generational problems are accelerated by the coincidence of expatriation with a period of profound social conflict marked by the emergence of a broad spectrum of movements for social change. Psychosocially, the experience of exile has represented to the expatriate an extraordinarily stressful process of dislocation and relocation, loss and rediscovery, and uprootedness and new growth.

Structurally, however, the Cuban emigration to the United States has been called a "golden exile." Portes noted in his study of Cuban refugee families in Milwaukee (9) that "seldom has a foreign group come to the United States so well prepared educationally and occu-

pationally and seldom has this country received one so well." Portes suggests that the ideological congruence between the prevailing values of American political culture and those for which the exiles left their homeland is a factor that helps to account for this Cuban "success story," as the American press has termed it. More significantly, the organized reception accorded the refugees—including concerted community action by diverse national and local groups in Miami and government assistance made available since the 1961 founding of the Cuban Refugee Center—has provided an important source of initial support, resettlement aid, and other necessary help. Other mitigating factors include the exiles' prior proximity to and familiarity with U.S. society and their historical affinity with the large Spanish-speaking minority already present in this country. We view these as necessary but not sufficient conditions in accounting for the structural integration of this massive influx of Cuban exiles.

The development and maintenance of a strong sense of ethnic consciousness and ethnic community have been key social determinants of the adaptation process of Cuban exiles. Despite the patent situational differences between Cuban families in the teeming colony of "Little Havana" in Miami and those resettled elsewhere, Cuban communities throughout the United States have functioned as comparison reference groups for their members and, as one study of refugee families in New Jersey indicated (10), cushioned the loss of occupational status. They permit adaptation within a supportive network of ethnic relationships and affiliations and constitute sources of psychosocial gratification to the expatriate that are unavailable outside Cuban groups. The existence of Cuban communities sustains a singularly Cuban self-image. The proliferation and interplay of discrete aspects of traditional lifestyle—such as Cuban music, dress, literature, mass media, meeting places, celebrations, and even basic items of Cuban cuisine—contribute substantially to the maintenance of the group's symbolic identity under conditions that might otherwise have caused it to atrophy.

In the manner of a self-fulfilling prophecy, the exiles typically have brought a feeling of equality with their American counterparts and a conviction of their ability to adapt in this country after having burned their bridges behind them. Furthermore, they have defined their expatriation as a provisional and temporary state, resulting not from pursuit of the "American dream" but from escape from the "Cuban nightmare." They still think of a return to their homeland, and this conception has strengthened their sense of solidarity in exile. Indeed, coming into exile to the United States was a firmly established precedent in Cuba's political history. Numerous precursors—from José Martí, the founder of the nation, to five chiefs of state, including Batista and Castro themselves—were exiles in this country prior to their return to Cuba and ascent to historical significance or political power. In any case these dimensions form an ethnic consciousness for the expatriates, clustered around a "Cuban is beautiful" attitude and characterized by an ongoing reaffirmation of their cultural values and ethnic roots.

CONCLUSIONS

Despite their diversity, the almost one million Cuban expatriates in the United States share in a common experience of uprootedness, adaptation, and new growth. Accordingly, we have conceived of the prototypical experience of exile as a dialectic of loss and reconstruction, marked by a persistent conflict of loyalties and manifesting identifiable psychological patterns in the individual's response to the severe stress. Furthermore, Cuban expatriation has coincided with a period of rapid sociocultural change in the United States, thus exacerbating for the refugees the problem of adaptation to a radically new situation.

A complex of situational determinants has shaped the course of the exile process, and the resilience of Cuban families can be accounted for by a variety of factors that have mitigated the severe circumstances of expatriation. Among the most significant social determinants we have identified are the following: 1) characteristics of the exiles as a group, including a comparatively high level of education and occupational experience, familiarity with U.S. society, and ideological congruence with basic American values, 2) the creation of a network of relationships and activities within ethnic communities in the United States, the best example of which is the extraordinarily large colony in Miami, which in turn permits 3) the development and maintenance of a vigorous sense of collective identity and ethnic consciousness. Finally, we noted the significance of the reception accorded the refugees by the host country, which did not consign their fate to a fierce and arbitrary individual struggle, but rather organized it effectively through coordination of federal, state, county, municipal, and private agencies around the Cuban Refugee Center in Miami.

The arduous experience of exile has been traumatic for the Cuban expatriates, but it has nevertheless intensified life, tested and evoked their personal resources, and widened their horizons. For the United States as well as for the other nations in the world that face a swelling current of refugees, perhaps a moral can be drawn: the mass reception of refugees need not be a chaotic, counterproductive, or alienating experience—it can be an enriching, creative, and engaging opportunity to grow while learning a poignant lesson of universal history.

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Pseudohomosexuality in Feminist Students

BY ZIRA DEFRIES, M.D.

The author describes the influence of the feminist movement on the intellectual, social, and sexual behavior of feminist college students. The complex interrelationship between sexual political ideology and actual sexual behavior creates confusion and anxiety for some vulnerable students in their attempts to evolve a satisfactory sexual identity. A series of vignettes illustrates the struggle of such students, who initially expressed concern over actual or potential lesbianism and its connection to the movement, to resolve their conflicts concerning feminism and sexual preference.

WITHIN THE PAST decade there has been a rapid development of what has come to be known variously as the women's rights movement, feminism, and women's liberation. None of these terms is specific enough for a proper definition, but implicit in each are aspects connoting the need for political, social, interpersonal, sexual, and gender role change. Despite the amorphousness of the movement, certain pragmatic sociopolitical and ideological tenets are subsumed in its scope and its variegated forms.

A particularly suitable field in which to study various aspects of the feminist movement is a woman's college. Despite the fact that a college population is hardly a representative segment of women, it does permit a view of a group of women who are likely to be sensitive to social change, intellectually oriented, and introspective about themselves in relation to their immediate world. In addition, since college students are at a crucial stage in the development of a definitive sexual identity, they are particularly susceptible to a movement in which sexual values and mores are nuclear concepts and in which ideas implicit in the dogma readily lend themselves into translation into overt sexual behavior. For a limited number of vulnerable feminist students, the complex interrelationship between sexual political ideology and actual sexual behavior results in a syndrome here referred to as pseudohomosexuality. In this syndrome sexual behavior is strongly influenced by political ideology, and the resolution and consolidation of the adolescent phase of sexual identity development, with definitive sexual preference, are difficult and prolonged.

To provide historical perspective I will briefly review the development of the current-day feminist movement. I will also describe its evolution in a women's college of an urban university from groups that were loosely knit to units that became increasingly cohesive and characterized by varying degrees of political and sexual militancy. The different sexual behaviors manifested, i.e., homosexual, heterosexual, and bisexual, will be described within the context of the feminist students' struggle to evolve a satisfying sexual identity and preference.

ROOTS OF THE FEMINIST MOVEMENT

The feminist movement evolved from two main roots: 1) the National Organization for Women (NOW) formed in 1966, consisting initially of women who had been associated with the President's Commission on the Status of Women, and 2) young women who were involved in the civil rights and youth movements of the 1960s. The former, a more conservative group, has worked through the traditional channels of established power groups. The younger and more radical branch has produced more theoreticians and has shaped much of the ideological perspective that emphasizes personal change as a prelude to sociopolitical change. It has developed and spread techniques of consciousness raising as a means of demolishing or redefining stereotypic female concerns, including sex and interpersonal relationships, and has attempted to refashion not only women's ideas but their very identities. Many of the younger women see themselves as radical feminists in search of a "new woman identity" (1, 2). Lesbian feminism is part of the radical feminist ideology; its concern is with an alternative feminist lifestyle in which lesbianism is liberated from its conventional sexual context to a total emotional commitment toward women and a denial of the traditional reliance on men.

In this college the change in women's self-perception began to emerge openly during the 1968 student strikes when politically activist women complained about their oppressive roles in the student-occupied administration buildings. Demands for sexual equality became a unifying theme for the newly developing feminist movement. The first organized group to

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Dr. DeFries is Assistant Clinical Professor of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, N.Y. Address reprint requests to her at 40 East 83rd St., New York, N.Y. 10028.

appear on campus worked on women's issues exclusively, disassociating itself from the larger university group of political activists.

In 1972 the Women's Collective was formed, with the main purpose of raising female consciousness. In 1972 a few zealous feminists formed a group called Lesbian Activists; there was no opposition from the administration, although in 1963 two women had been expelled from the college for reported lesbianism. In 1973 a day-care project and a self-help clinic were opened, and regional conferences and workshops on women's roles (including lesbianism) were held. At the present time a wide range of ideologies exists on campus. The radical lesbian contingent constitutes a relatively small fraction of the overall feminist group, which includes nonradical heterosexual and bisexual students, many of whom consider a categorization of sexual preference irrelevant.

Feminist-oriented college students span an age period in which intense involvement in a cause can be considered an essential step in identity formation and a prelude to normal personality development (3). However, unmoderated or extreme ideological pronouncements that concentrate on selective aspects of interpersonal transactions may well be pathological (4).

For some students in the movement, the various expressions of sexual behavior advocated either implicitly or explicitly—in particular the encouragement of open expression of homosexuality—pose difficult problems. The educational and clinical approach to sexual functioning, including dissemination of information about such matters as masturbation and genital examination techniques, gives high visibility to female anatomy and physiology and helps to demystify sexual functioning. New approaches aimed at creating egalitarian concepts of male-female role relationships, both sexual and nonsexual, downgrade sex-typed behaviors and make unacceptable some ideals of masculinity and femininity that were formerly considered central to the development of a clear-cut sexual identification. Few standards remain for evaluating emotions and behavior in conventional terms. Considerable controversy over which behaviors should be totally discarded and which should be modified—and in what directions—still exists. An ideological stalemate is the inevitable result of seeking a more clear-cut definition of sexual modes of behavior. For some students this indeterminate state of knowledge provokes a great deal of anxiety and legitimizes further inquiry and exploration within a clinical framework.

CASE VIGNETTES

During the past 2½ years, nine feminist students with sexual identity and sex preference confusion were seen in the college psychiatric service. I have selected case material concerning three students as representative of the larger group. These students, in treatment for 2 or more years, were essentially obsessional personalities (and two exhibited hysterical traits). In keeping with the view of obsessionalism as a cognitive disorder, they demonstrate the way in which acceptance of ideology and dogma can be reassuring to the obsessional's doubt and ambivalence and how rejection of this same ideology produces depression and even suicidal thoughts (5).

In the initial interview, a concern over actual or potential lesbianism and its connection to the movement was invariably raised. Thereafter the themes included 1) vacillation between wanting total involvement in the movement to political but not personal involvement to no involvement, 2) alternate excitement and depression about lesbianism, and 3) castigation of men or infatuation with them. These students were preoccupied with the need to establish a unidirectional sexual preference as a means of achieving a satisfactory sexual identity. The problem, laconically conceptualized as "to be or not to be a lesbian," carries with it the expectation that when it is resolved, the feelings of dislocation created by this confusion and by fluctuating reactions to the movement would end.

Material abstracted from the initial interviews illustrates these students' concerns. For example, student 1 thought she was probably lesbian but was confused about whether or not to stand up for her lesbianism since she had a hard time pushing away men who were attracted to her. She wanted to be politically (but not personally) involved with gay people despite the fact that she was engaged in a sexual relationship with an "unliberated sexist female" student who, she said, exploited her feminism and rejected the "political me." She felt torn between politics and sex and longed to be free of the "distraction of politics."

Student 2 stated that as a result of her activity in the women's movement she had made too many major decisions, including the conclusion that she was definitely a lesbian and not even bisexual. She thought this lifestyle was too narrow and made no sense; she wondered why she had so defined herself. Before coming to college, she said, "It was all ideology and no acting out." At college the opportunity for being homosexual made her wish that men were around.

Student 3 claimed she had "gone overboard" with her ideas on feminism: "They are getting out of proportion." She had anticipated a sexual relationship with her closest woman friend and felt estranged when this young woman became attached to a man. Her activity in the women's collective affected her thinking and her friendships. "Movements are strange things—they get one going into ways that are incompatible with what actually goes on." She had been self-righteous about the movement but now wished she could be less defensive and more moderate. She stated: "Movement ideas have been given more of a priority than they deserve."

Shifts between vigorous espousal and rejection of the movement, accompanied by acceptance and rejection of lesbianism, occurred in rapid succession early in treatment and continued, although with decreasing

vehemence, as sexual relationships and ideological concerns were explored in greater detail. Sex was often perceived as the source of evil, and a moratorium was intermittently declared. A retreat to a purely political, asexual position was experienced as a momentary relief from the pressure of having to choose a one-way direction in the homo/heterosexual see-saw. Shifts of acted-out sexual behavior were commonly accompanied by depression and less often by euphoria.

Verbatim examples illustrate these dilemmas and the attempts to resolve them. Student 1 said, "When I became interested in the feminist movement I thought I had to be a lesbian and I was frightened. The more I read, the easier it became to equate lesbianism and feminism. Unless you take a belligerent stand of hating men you fear the old conditioning pressure to be heterosexual will win out." However, after declaring her lesbianism she became involved in a relationship with a man. "I realized how rigidly ideological I had been by saying I couldn't sleep with a man—that was bullshit. I feel trapped and discouraged by politics but I've been programmed to be political; the first day I came here I joined the women's collective and I haven't had a break since."

After a brief period of heterosexuality her feminism reasserted itself, and she literally cried for her lost lesbianism:

> All the strong feminist dreams and strength I felt from my lesbian identity are being crushed. Being a lesbian feminist has been my way of avoiding facing the fact that I can be heterosexual when I am with a man and that my identity is defined by the outside person. It is so confusing to relate to men and women at the same time. I don't have a fixed sense of myself. In the absence of the feminist movement, with its support in being gay, I could not have been a lesbian. On the other hand it is possible that if I had not had the crusade, I might have been more actively lesbian. The most involved feminists are lesbians. Total commitment is necessary. If you become active in feminism and believe in changing society, you question all the values of that society. If you are an intelligent female and can acknowledge sex feelings toward women it becomes impossible to be exclusively heterosexual.

The supreme irony occurred when a man asked her during intercourse if she was a lesbian.

Student 2 had had only heterosexual relationships until she came to college:

> Friendships with women do not require sex, yet relationships with females hover around sex. The things I think about feminism are related to the way I feel about my father; feminism gives me a right to villainize him. For me it is a personal dialectic, but I wonder how much social pressure makes people act in a certain way. I never used to feel secure unless I had a guy around. Now I refuse to be defined by a man's perceptions and I am paranoid about my life being guided by a man.

These feelings led to a dissolution of the sexual relationship she was having with a man:

> I feel alienated by men and am more satisfied emotionally by women, but I wonder how much my feminism is determined by personal feelings. The ideology of feminism is very personal; in most cases it is more than just a political thing. I wonder which way I will go sexually. My friends and I believe a sexual relationship comes out of sharing a close friendship and a similar ideology, but I am too interested in men to be a lesbian forever. Historically there has never been a better opportunity to experiment.

After this seeming complacency, however, she began to feel torn apart: "learning to trust men is not mutually exclusive with having close women friends. I don't know where my identity is at. I don't know what is going to happen to my feminism. I don't know where to put women." She became progressively more anxious about her sexual identity: "I must resolve my personal and ideological feelings so I can gain a sense of direction in life."

Student 3 initially felt that the only place she could find her identity was in the movement: "My identity is threatened by men and heterosexuality." However, she soon felt confused and frightened by men and fearful of women. Although she had been a prime mover of the feminist organization, she could no longer stand it: "The lesbian bar scene is a meat market—self-degrading and masochistic. I just want to be a political lesbian." After a brief affair with a man, she said, "I can no longer find consolation in the movement—it turns me off." This rejection of the movement led to a loss of her sense of self. "Politics used to be my whole self—now I can't relate to them."

She felt she was changing too fast and too drastically:

> It used to be everything was a woman—it was such a strong identity—now I have to learn to see things more realistically. I am adopting a new ideology. Sexuality is not the only thing we live for. I don't know what my sexuality is. I call myself a lesbian; I am a lesbian. I have a beautiful relationship with men—it is a definite conflict—in no time I'll be depressed again.

Instead, she threw herself into an intensive lesbian relationship. "It is great to be a lesbian—with women I am the real me." Even though she was not sure she could swing back to being a political lesbian feminist, she said, "I have the vibes back of my self-identity—feminist ideology has become my life." Not much later she began to feel suicidal as the pressure for a single direction of her sexual orientation became overwhelming: "I want to liberate lesbians from sex—to be a lesbian is to be a feminist—lesbianism is feminism, not just sex." Finally, she felt she could no longer compromise between the personal and political: "It splits my life into separate camps. I have become more certain of my own self and personality and sex preference; I am securely lesbian and have come to grips with this rare species, man." Having thus opted for lesbianism she rejected the movement: "I always believed the personal and the political cannot go together."

DISCUSSION

Undoubtedly family background and early developmental factors predisposed these conflicted students to (at the very least) "tilt" toward homosexuality. In adolescence they displayed strong intellectual and academic interests. Their perception of personal problems in a political context made them appear to have a broader intellectual scope than the more typical apolitical student. Although they often seemed emotionally childlike, they were impressively mature in their dedication to work in and out of the movement as well as in therapy.

Any attempt to predict the eventual outcome of the direction of sex preference of the nine students must remain tentative at this time. For the present, two of the three students still in therapy are tending toward a heterosexual adjustment, and the other appears to be predominantly homosexual. Of the remaining six, three developed a distinctly heterosexual preference, two a less clear but predominantly heterosexual adjustment, and one made what appeared to be a comfortable homosexual resolution.

The term "pseudohomosexuality" as applied to this group is reserved for that period during which ideology and manifest sexual behavior are inextricably mixed and sexual identity so confused that a unidirectional or at least a primary preference is not possible. At the same time a definitive sex preference is considered the means to providing a consistent, well-ordered world view through which feelings of confusion in regard to sexual identity can be resolved. During the period of most acute conflict, anxiety and depression are prominent symptoms. The problem is resolved when interlocking ideological and sexual identity and preference components are separated from one another so that more genuine sexual proclivities can be determined.

Feminist students who use the movement as an attempt to consolidate sexual identity do so for a variety of reasons, including coming to grips with sexual fears, postponing separation from parental ties (in these cases peer group ideology is substituted for family ideology), acting out of hostility toward males, and as an antidote to isolation and loneliness. In the course of therapy, movement concerns abate, militance decreases, and sexual behavior is no longer counterposed uncritically against ideology; thus the need for alternate homosexual and heterosexual acting out is curtailed. The exploration and reexamination of motives continue until either the sexual dilemma is resolved completely or there is an easier acceptance of a still wavering preference. When the young woman becomes an adolescent with a sexual problem rather than an ideologue, the anxiety associated with the movement markedly decreases. No longer does the movement serve as a cognitive and affective organizing focus around which sexual behavior pivots. By this process feminist ideology, formerly considered the essence of a self-identity around which a world view can be structured, loses its value as a means for determining sex preference.

The complexity of personal, social, and sexual factors in feminist doctrine injects a new component into adolescent female sexual identity development and preference that is not readily explainable by existing theories. The inconclusiveness concerning the evidence on the etiology of the mechanisms involved requires study along what Freud considered the circuitous unresolved path of female sexual development (6). Significant modifications of Freud's views have been made by Horney, Jones, Rubenfine, Kestenberg, Barnet, and Sherfey (7). The concepts of gender differentiation and identity described by Money and Hampson (8) and Stoller (9) are crucial additions toward understanding these mechanisms. However, in spite of agreement by developmental theorists that "adolescence is a period of greatly increased and more specific sexual interest, the psychosexual mechanisms remain largely untested and the question of the acquisition of the direction of sexual preference remains an open question" (10).

In an extensive review of the literature on female homosexuality Kenyon (11) confirmed what appears to be true for the students described here—that homosexual behavior in the adolescent may be part of a crisis of identity and should not be looked on as a fixed sexual orientation. Saghir and Robins (12), in their comprehensive study of nonpatient female homosexuals, pointed to a distinctive feature of childhood and adolescent cross gender behavior, starting at a very early age, with developing psychological responses that seem to have been determined early in life. They also cite the important fact of adolescent turmoil and frequent homosexual involvement of basically heterosexual adolescents as part of a specific "situation." Money and Ehrhardt (13) pointed out that in our present state of knowledge, no hypothesis can predict with certainty when a normal individual will become erotically homosexual, bisexual, or heterosexual. In discussing the etiology of homosexuality, Cooper (14) expressed a similar inconclusiveness; he believes that despite a literature of encyclopedia proportions, myriad investigations, and ingenious theories, the importance of various psychological and other factors in the etiology remains undetermined, although the consensus "leaves no doubt that the basic homosexual drive is fashioned in childhood, or at the latest in adolescence." He believes that Bancroft's model (15), which attempts an integration of gender identity and sexual preference based on the principle of cognitive consistency theory is "an eminently sensible way of looking at etiological factors." Cooper (14) finally and cautiously predicted that the complexity of society may defeat investigative efforts at finding a cause.

Developmental theorists with biological, psychoanalytic, and psychosocial orientations will continue to make revisions and emendations of their theories about sexual identity, prodded in part by the feminists' efforts to consolidate existing assumptions of sexual behavior that are at variance with their ideology. The collision, at many points, of psychological theory and

feminist doctrine brings both into the mainstream of current research on many fronts. Both camps have profited from charge and countercharge, and each has had to critique its own doctrines and affirm, extend, or discard particular facets. What better way to plug theoretical lacunae than by continuing controversy in the interest of greater understanding of sexual behavior.

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It Is Always the Woman Who Is Raped

BY DEENA METZGER, PH.D.

Rape is described as an act of power that is sanctioned by literature, myth, and culture. Rape is a dire experience for women because it implies total loss of self—the woman is a function, not a person. The victim experiences total emptiness and isolation from self and society. The author believes that an effective "treatment" for the rape victim is community. Women asserting themselves by speaking out about their experience validate themselves as persons.

RAPE IS AN aggressive act against women as woman. The rapist is educated to his behavior by his society, and rape is the extreme manifestation of approved activities in which one segment of society dominates another. Rape is a ritual of power.

There are at least two ways of knowing. One is intellectual and the other is experiential. With which voice shall I speak to you? After rape there is a terrible silence. Then, if one is fortunate, one begins to learn to speak again. Why is the silence so terrible and so profound? What is the meaning of the crime that has been acted out? Why are its effects so dire?

Rape is the most common of the violent crimes. As the incidence of rape increases and we focus our attention on understanding the rapist—learning almost nothing of the victims—we continue the social patterns that perpetuate the crime.

Literature, art, and myth are full of images of rapes that are approved, even canonized. We distinguish the crime on the street from the "Rape of the Sabine Women," yet the painting by Rubens is informed by the same values that produce the street crime. Rape is the assault of a man on a woman, but it is also the symbolic enactment of social and cultural attitudes. On one level, perhaps unconscious, it is a gross and extreme form of social regulation by which woman is brutally stripped of her humanity and confronted with her definition as a nonperson, a function.

These attitudes are not peculiar to the rapist. The rapist is not an eccentric but an extremist acting out the being and nonbeing scenario that is basic to American society. In this scenario, the rapist uses power to confront the egalitarian aspect of sexuality—community. That the act that physically and symbolically indicates communion should be so often distorted into combat is not ironic but tragic, undermining essential relationships. Rape asserts only combat, brutalizing the communal aspect of sexuality, destroying meaning, relationship, and person, creating a universe of emptiness and ontological terror. There is no distinction between the male social intent and the private female response. A woman who is raped understands her condition with metaphysical clarity.

Rape is mythically asserted, artistically glorified, historically condoned, and symbolically urged in the media and in advertising. Persephone is a classic example of the rape victim. Persephone has been promised by Zeus to Hades. Her primary definition is property. The earth opens, Hades carries her within, imprisoning her in hell; she is isolated. Her mother, Demeter, mourns her, leaves Olympia, and in her grief withholds spring. Neither Zeus nor Hades is condemned. Male power is glorified, while Demeter, condemned for withholding, is reluctantly placated. Persephone is returned; spring comes. However, Persephone, having eaten pomegranate seeds while in hell, must return to Hades every year. Thus Persephone is responsible for winter. The victim

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Dr. Metzger is Assistant Professor of Literature, Los Angeles Valley College, and Director of the Writing Program, Feminist Studio Workshop, the Woman's Building, Los Angeles, Calif. Address reprint requests to her at 4230 Bellingham Ave., Studio City, Calif. 91604.

The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

bears the burden and guilt of the crime that has been perpetrated against her.

We can translate this myth into contemporary experience. A man commits a crime against a woman. The authorities ask her, "Why were you walking alone? What were you wearing? What did you say? Are you a virgin? Did you pull down the shades?" Three basic attitudes of power are involved here: 1) to be weak is a crime deserving punishment, 2) the weak are accomplices in the crimes against them, and 3) the victim has committed the crime.

The stories of Persephone, Leda, and Europa are early mythic tales that teach rape as a learned cultural activity. The rights of men over women, the attitude underlying all cases of rape, were translated later into the droit du seigneur, the "giving" of the bride in marriage, the rites and rights of the marriage bed. These social customs reinforced the idea of woman as property, to be entered and used for man's purposes: woman as function.

A function is not a person. Rape is one manifestation of society's intent to depersonalize woman. It separates the woman from her humanity. Reduced instantly from person to object, property, flesh, vessel, the woman is immediately separated from anyone or anything that can comfort her. The basic experience of rape is isolation. Humanity depends on community, and the effect of rape is to destroy simultaneously the sense of community and the sense of person. Demeter and Persephone are separated because there is no comfort Demeter can give. Persephone is alone and empty. After I was raped, I knew that women were vessels, shells that could be emptied, flesh and bone frames without centers or substances. Women were moons shining only with reflected light, dead planets.

Because women's assertion of self conflicts with social and cultural patterns and with role and biology, the loss, after achievement, is grave. Rape is the instant proof of unalterable conditions, the erosion of years of work. Contemporary feminism has only begun to change this condition. The very unique interest of women in autobiography is a sign of the difficulty women have with identity. Where men always have asked, "What is the meaning of my life?" women are beginning to simply ask, "Do I have a life?" The effect of rape is the same whether the victim is a young girl, virgin, mother, or old woman. Rape is a crime against the person, not against the hymen.

My experience and that of the women I know tells me there is no treatment for rape other than community. Therapy or consciousness raising can be helpful as long as no "cure" for a "condition" or "disease" is implied. Rape is loss. Like death, it is best treated with a period of mourning and grief. We should develop social ceremonies for rape, rituals, that, like funerals and wakes, would allow the mourners to recover the spirits that the rapist, like death, steals. The social community is the appropriate center for the restoration of spirit, but the rape victim is usually shamed into silence and or self-imposed isolation.

In some ways, rape is never erased. Years later, even the word "rape" or the shadow of a familiar face can cause unexplained pain. The raped woman often cannot bear to be touched. Isolation is her condition. Touched, she knows she cannot feel; touched, she remains untouched. She is incarcerated in Hades. Her mother is outside and cannot hear her.

After I was raped, I had intercourse with my husband as a ritual gesture. (I had learned as a child to get back on my bike after falling, lest I never mount again.) Intercourse was easy. It didn't matter. I was an abandoned house. Vacated. Anyone or anything could enter.

Later I went through the ritual of talking to people. It always seemed as if I were talking through glass or underwater. I could never tell my mother; she couldn't bear the pain. Others, it seemed to me, drew away. I could not bear to be alone, but in company I felt abandoned, estranged. For months I looked to my husband for comfort he could or would not give. A year later, we began a divorce.

I felt endangered everywhere. Every noise startled me. Every leaf was camouflage for an assassin. For months a friend of mine described searching the faces on the street as if to ask, "Are you the one?"

Revenge became an obsession. My husband was a party to this; vengeance would relieve his feeling of impotence. We spent days searching the streets in a mythic ritual. He was looking for power. I was looking for self. If a crime had been committed, then perhaps there had been a victim and a victim is a person: perhaps I would find myself.

Rape is usually the assault by the stranger, the unknown asserting his perceived socially, culturally, politically, and God-given rights. In earlier times rape was more frequently enacted by familiars. The woman who was shared, given, offered, dedicated was in effect raped; to be entered without permission, invitation, or desire is to be raped. Today most rapes are forced entrance by a stranger. The stranger is anyone. Simply a hand emerging from the shadows. Being anyone, the rapist is everyone. He asserts that the woman does not belong to herself. She is there for his use, his pleasure. She is nothing in herself.

The rapist acts for the society, concretizing certain ideas through his behavior. He is often marginally integrated into the society, and rape is the way he affiliates himself, if not through the act, then through the attitudes. Through rape he asserts power and possession in a common violent and spontaneous action. By choosing to assert himself against woman and her body, he—like church, state, schools, advertisers, and the media—is simply asserting certain rights and prerogatives over woman, using her for his own purposes. He is translating cultural thoughts into action.

The rapist is any man against any woman. Some form of anonymity is essential. He acts against the woman who happens to walk down the street, happens to be stranded, happens to be alone. It is an act not against someone but against anyone. Chance is an es-

sential element. It reinforces the nonperson status of the victim, who is attacked at the moment of her nonbeing, in the shadows, alone, in silence, nameless; the rapist forces the identity of nonperson indelibly upon her. Whoever she had previously assumed herself to be, after the rape she is nothing: a body, a thing.

Yet rape is an assault on one's most private being. At her core, woman has a deep biological sense of herself, created through the cycles of blood and ovulation, through the pattern of puberty, menstruation, pregnancy, lactation, and menopause. This identity is enhanced by the cultural and symbolic values that the woman's body represents. It is this essence that is also violated: what she is and what she means. Finally, rape is a crime that gives the criminal pleasure. Not only is the victim possessed as by a demon, but she hears the demon laughing. She is used against herself. The groan of pleasure makes her feel despicable. Even before she is accused by police, parents, physicians, interrogators, she experiences herself as complicitor. Self-hate reinforces her personlessness.

Perhaps for these reasons, there are almost no stories of rape written by women. Leda is assaulted by a "feathered glory." A magnificent bull kidnaps Europa. Daphne, pursued by Apollo, becomes a laurel, but Apollo is not deformed. Woman is one of the expected spoils of war. Cassandra is not the only one who speaks and cannot be heard; there have been millions.

Because rape is an act of power that male society will not undermine, the woman is defiled. Perhaps less consistently than at other times, the victim is isolated or isolates herself; she enters a psychic quarantine as if she were contaminated, diseased, scarred. Although today women do not automatically lose their social and economic value through rape, they are devalued through accusations of seduction. The woman is suspect while the man is protected. Society asserts he is ill, marginal, or falsely accused. Men protect his rights and worry that the accusation may be false or the evidence contrived. The rapist is protected because he is themselves. The issue is always power. Rape, like the immaculate conception, becomes an act without an actor—the immaculate defilement.

The nonperson status of women is maintained in various ways, all of which seem to focus on sexuality. Symbolically, woman is the door, the opening, the entrance that must be sealed, protected. In one sense, according to Octavio Paz, woman is the essential violated one; she is the wound, the entered, *La Malinche*, *La Chingada*.[1] Her condition is one of violation, and it is against identification with such openness that man arms and armors himself. When man himself is entered and violated he is, according to Jean Genet, used as a woman, womanized, degraded.

Yet society simultaneously asserts that woman is the not-yet-entered, the innocent. The virgin is the unknown. To have intercourse is to know and be known. Sexuality is therefore knowledge, and woman is often insulated from this knowledge. That is part of the pattern of power and powerlessness, of being and nonbeing. To be without knowledge is to be nothing. For we are what we know. Woman at her best (as virgin) is thus nothing. Traditional heroines or women of power are grandmothers beyond sexuality, like Golda Meier, or virgins, like Mary, Joan of Arc, Elizabeth the Virgin Queen. The combined image of power and knowledge (sexuality) is terrifying: ogress, witch, Medusa, Clytemnestra, Medea—something to be destroyed.

The rapist acts against the societal injunction to protect the innocence of woman. He acts against the protection of woman, he denies the fortress of the private house. In so doing, he acts on the myth that for woman one error is fatal. Men, like the prodigal son, are capable of change, growth, learning. Women, for whom definition is finite, are confined within a single fall. Therefore the rapist has absolute power; he can destroy identity with a single act.

The raped young woman goes from nonbeing to Nonbeing—to not being at all. Rape is the act by which woman is known without knowing anything herself, the means by which knowledge and therefore presence is bypassed and nonbeing (innocence), which was tentative or temporary, becomes permanent. Raped, the woman becomes known, manifest, without gaining the identity that comes from knowledge. The only knowledge she gains is the knowledge of her lack of self.

With the exception of a few remarkable and brave women writers, including Doris Lessing, Simon de Beauvoir, Anaïs Nin, Kate Millet, and Colette, contemporary and historic literature (which reflects and creates social values and attitudes) asserts the nonbeing of women. From D. H. Lawrence's *Plumed Serpent* and *The Man Who Died Twice*, to Henry Miller's *Tropic of Cancer*, Norman Mailer's *American Dream*, and Erica Jong's *Fear of Flying*, nonbeing is the definitive condition of woman. And rape, which the film critic Arthur Knight recognizes as a "film cliché," is the act that confirms this nonbeing.

The literary, mythic, and historic attitudes toward rape are still with us. The charge against Joan Little for the murder of the rapist jailer who assaulted her is the classic case of the victim accused of the crime. It is as if simple defenselessness, isolation of woman in itself, invites attack. The circle is complete. Men isolate women and then accuse that isolation. Rape is socially seen as response rather than provocation—it is the desperate male response to a powerlessness that is confirmed for him by the spectre of female identity. Similarly, when Inez Garcia acted as a person against her attacker, she was the one charged. Worse than the image of an attacked woman is an image of a woman

[1] *La Malinche* refers to the mistress of Cortez, the Indian woman through whom the invasion occurred: "The Mexican people have not forgiven *La Malinche* for her betrayal. She embodies the open, the *chingado*, to our closed, stoic, impassive Indians. . . ." "The *Chingada* is the Mother forcibly opened, violated, or deceived. . . . This passivity, open to the outside world, causes her to lose her identity . . . she is no one; she disappears into nothingness; she is Nothingness. And yet she is the cruel incarnation of the feminine condition" (1).

counterattacking. Here, the contradiction of nonbeing creates panic.

In a society such as ours, where the fantasy of power is a constant stimulus and Superman, John Wayne, James Bond, and Kissinger are cultural heroes, while powerlessness and alienation are the reality, it is inevitable that rape occurs frequently. Males denied power in their daily lives find means to act as if they had it. Women are the obvious victims because male society still defines them as powerless, functional nonpersons. Providing men with the feeling of power has been considered to be one of woman's duties.

In 1968 I was raped at gunpoint. Later, lying naked on the floor, I could only whisper over and over again, "One doesn't treat people like this." I had had only a momentary glimpse of the rapist, a stranger. From my brief description, a police artist drew a face identical to a photo I later identified in police records. This man, who was on parole, had been incarcerated for rape. His parole officer called him in for questioning but concluded on the basis of an alibi that he had been too far from the scene of the crime.

The first fictional account I wrote of the rape (in an unpublished novel titled "Flying With a Rock") was couched in fantasy. I described the rape of a madwoman. I was still maddened by the assault and could only develop a character who had lost all sense of herself. Four years later I wrote a more accurate version, which describes a woman both overpowered and divested of her power (2). Her last words are "I cannot." She is emptied out. I decided to make this public in order to break some of the silence and isolation which reinforces the personlessness of women. The private voice in the public sphere confirms our common experience through which we begin to assert ourselves. Unlike my character, now, I can.

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The Rape Victim: Psychodynamic Considerations

BY MALKAH T. NOTMAN, M.D., AND CAROL C. NADELSON, M.D.

Rape challenges a woman's ability to maintain her defenses and thus arouses feelings of guilt, anxiety, and inadequacy. Women's individual responses are determined by life stage considerations as well as their defensive structures: concerns about separation-individuation may be aroused in the young woman; a divorced or separated woman may find her credibility questioned; older women's fears of sexual inadequacy may be compounded. In counseling, the victim's previous adjustment should be assessed, she should be given support and reassurance, and specific considerations related to her life circumstances should be acknowledged and dealt with.

The authors are with Beth Israel Hospital, 330 Brookline Ave., Boston, Mass. 02215, where Dr. Notman is Psychiatrist and Dr. Nadelson is Associate Psychiatrist and Director, Medical Student Education. They are also with Harvard Medical School, Boston, Mass., where Dr. Notman is Assistant Clinical Professor of Psychiatry and Dr. Nadelson is Assistant Professor of Psychiatry.

THE EXPERIENCES that we call rape range from surprise attacks with threats of death or multilation to insistence on sexual intercourse in a social encounter where sexual contact is unexpected or not agreed upon. Consent is crucial to the definition of rape. The importance of mutual consent is often overlooked and misinterpreted; many people assume that certain social communications imply willingness for a sexual relationship. Although men, women, and children are raped, the majority of rape victims are women; this paper will focus on understanding rape as a psychological stress for the woman victim.

Burgess and Holmstrom (1) divided the rape victims they studied into three groups: 1) victims of forcible completed or attempted rape, 2) victims who were "accessories" because of inability to consent, and 3) victims of sexually stressful situations where the encounter went beyond the woman's expectations and ability to exercise control. Despite the different circumstances, the intrapsychic experiences of rape victims in all categories have much in common.

The rape victim usually has had an overwhelmingly frightening experience in which she fears for her life and pays for her freedom in the sexual act. Generally, this experience heightens a woman's sense of helplessness, intensifies conflicts about dependence and independence, and generates self-criticism and guilt that devalue her as a person and interfere with trusting relationships, particularly with men. Other important consequences of the situation are difficulty handling anger and aggression and persistent feelings of vulnerability. Each rape victim responds to and integrates the experience differently depending on her age, life situation, the circumstances of the rape, her specific personality style, and the responses of those from whom she seeks support.

RAPE AS A STRESS

Rape can be viewed as a crisis situation in which a traumatic external event breaks the balance between internal ego adaptation and the environment. Since it is an interaction between an extreme environmental stimulus and the adaptive capacity of the victim, rape is similar to other situations described in the literature on stress, including community disasters (2, 3), war (4–7), surgical procedures (8, 9), etc. The unexpectedness of the catastrophe and the variability of victims' resources for coping with an experience that may be viewed as life-threatening are critical factors in rape, as in other crisis situations.

Although there are cultural and personality style differences, descriptions of stress reactions generally define four stages, which vary in intensity and duration (10). These responses, listed below, are also found in rape victims.

1. Anticipatory or threat phase. In this stage, anxiety facilitates perception of potentially dangerous situations so that they can be avoided. Most people protect themselves with a combination of defenses that maintain an illusion of invulnerability, with enough reality perception to allow them to protect themselves from real danger. When a potential stress is planned (i.e., elective surgery), an individual can protect his/her ego integrity by strengthening those defenses which will ward off feelings of helplessness.

2. Impact phase. Varying degrees of disintegration may occur in a previously well-adapted person during this second phase, depending on the degree of trauma and the adaptive capacity of the individual. There may be major physiological reactions, including vasomotor and sensorial shifts. Tyhurst (2) reported on the extremes of fire and flood victims' reactions, which ranged from "cool and collected" to "inappropriate" responses, with "states of confusion, paralyzing anxiety, inability to move out of bed, hysterical crying, or screaming." The majority of these victims showed variable but less extreme responses—they were "stunned and bewildered" and demonstrated restricted attention and other fear responses, such as automatic or stereotypical behavior. This picture is also seen in rape victims.

3. Posttraumatic or "recoil" phase. Emotional expression, self-awareness, memory, and behavioral control are gradually regained in the recoil phase. However, perspective may continue to be limited and dependency feelings are increased. The individual perceives adaptive and maladaptive responses in him/herself and may question his/her reactions. A positive or negative view of one's ability to cope may affect the course of resolution of the trauma and future capacity to respond to stress, and self-esteem may be enhanced or damaged.

Group support during this phase enables the victim to feel less isolated and helpless. Obviously, the rape victim, who is usually alone during the attack, can only hope for support later. Women are often disappointed by the failure of family, friends, and the community to validate their experience.

Janis (9), in his study of surgical trauma patients, noted that any threat that cannot be influenced by the individual's own behavior may be unconsciously perceived in the same way as were childhood threats of parental punishment for bad behavior. This results in attempts to control anger and aggression in order to avoid provoking "punishment." The absence of overt anger is also a very prominent finding in rape victims.

4. Posttraumatic reconstitution phase. A process occurs during this phase that may alter future life adjustment. The loss of self-reassuring mechanisms that had fostered a sense of invulnerability may result in a decrease in self-esteem. The victim then blames him/herself for lack of perception or attention to danger. Kardiner and Spiegel (11) studied war stress and stated, "As soon as fear is directed inward in the form of questioning the individual resources to cope with external danger, or toward the group in the form of questioning its ability to be a protective extension of the individual, then a new and more serious danger situation is created." Maladaptive responses have been reported in the war neuroses of World War II (12), in which the individual develops mechanisms that are protective against further exposure to trauma but are psychologically costly and may involve loss of pride and self-esteem (13).

The rape trauma syndrome described by Burgess and Holmstrom (1) can be considered a form of stress reaction that can lead to traumatic neurosis. They reported an acute disorganizational phase with behavioral, somatic, and psychological manifestations and a long-term reorganizational phase with variable components depending on the ego strength, social networks, and specific experiences of the victim. They focused on the violent life-threatening aspects of the crime. Two types of response they noted are: "the expressed style," in which the victim is emotional and visibly upset, and the "controlled style," in which denial and reaction-formation seem to be the most prominent defenses. They also described feelings of shock and disbelief in many victims and the prevalence of

guilt and self-blame in the initial phase. The reconstitution phase varies considerably with each individual; however, the patterns of response appear to be similar to those reported in the other types of stress reactions we have discussed.

THE DYNAMICS OF THE RESPONSE TO RAPE

The important considerations in understanding the dynamics of women's responses to rape are 1) affects, 2) unconscious fantasies, and 3) adaptive and defensive ego styles.

Anger

A striking phenomenon in rape victims is the initial display of fear, anxiety, guilt, and shame—but little direct anger. There are several probable reasons for this.

1. Since rape may evoke memories of childhood threats of punishment for misdeeds (9), the victim may feel that she is being punished or is in some way responsible. Her anger may be repressed and experienced as guilt and shame, despite her concomitant feelings of helplessness and vulnerability. Most of the angry feelings appear later in recurrent nightmares, explosive outbursts, and displacement of anger as the woman attempts to master the assault.
2. Expression of aggression in women has been highly conflictual because of cultural restrictions and expectations of passivity and greater compliance for women. Women have often tended toward a masochistic orientation, in which anger is transformed into culturally supported patterns of self-blame. Identification with the aggressor, a mechanism that serves as an attempt to gain mastery, may also make it difficult to acknowledge anger toward the rapist.
3. The socially reinforced suppression of aggression in women has a possible adaptive function, since women are usually smaller and physically weaker than men. Therefore, not responding with a counterattack may prove beneficial. This is an important consideration in understanding the concept of consent. In the past, legal expectations included evidence of force or a struggle in order to establish rape. Current laws accept threat of force as sufficient, recognizing that a woman may submit in fear rather than risk fighting and being overcome.

Guilt and Shame

Despite the varying circumstances of rape and the different degrees of violence, surprise, and degradation involved, guilt and shame are virtually universal. The tendency to blame the victim, thereby assigning responsibility to her, fosters guilt and prevents her from adequately working through the crisis. It is common for a rape victim to feel that she should have handled the situation differently, regardless of the appropriateness of her actual response. Concerns about the amount of activity or passivity that might have prevented the attack or the rape are frequent. The assumption is that the woman should or could have handled the situation better, that her unconscious wishes perhaps prevented more appropriate assessment and more adaptive behavior.

The guilt of the victim is further increased by focusing on the sexual rather than the violent aspect of the experience. Although aggression is most prominent in the victim's perception, society regards rape as sexual. Since long-standing sexual taboos still persist for many people, even an unwilling participant in a sexual act is accused and depreciated. The popular adage that advises the woman who cannot avoid rape to "relax and enjoy it" misconstrues the attack as a sexual experience. In reality, the rape experience is depersonalizing and dehumanizing. The woman is often a faceless object for the rapist's expression of hostility, and the victim feels degraded and used. Furthermore, since women are expected to exert impulse control in sexual encounters, the rape victim's sense of failure in setting limits, impossible though this may have been, contributes to her guilt.

Unconscious Fantasies

The question of unconscious wishes translated into provocation of a rape must be seriously considered. While undoubtedly there are unconscious fantasies in which rape plays a part, and some women do have fantasies in which submission to a stronger man may be linked with forbidden oedipal wishes, on the conscious level the women knows she is submitting because any other behavior would result in real danger to her life. However, this is not so clearly differentiated in the unconscious. The universality of rape fantasies certainly does not make every woman a willing victim—or every man a rapist. The unconscious fantasy does not picture the actual violence of the experience.

An individual's defensive organization usually protects him/her from acting out such fantasies. However, if the defensive barrier breaks down and unconscious destructive, aggressive, or masochistic wishes gain expression, anxiety over the loss of control combines with guilt regarding the impulses. Rape involves an overwhelming confrontation with another individual's sadism and aggression and one's own vulnerability. This challenges the woman's confidence in her ability to maintain her defenses and controls.

Many women feel some ambivalence toward men as a result of past developmental experiences. Women expect men to be their protectors and providers, as well as relating to them sexually. Men may also be seen as potential aggressors and exploiters, and the experience of rape confronts the woman with this violent potential. The betrayal by the supposed protector who turns aggressor has a profound effect. Almost all rape victims say they trust men less after the rape. All men may be suspect, and all are potentially on trial. Uncertainty about one's ability to control the environment reverberates with concerns about the ability to control and care for oneself.

Men's Responses to Rape

It is important to consider the responses of men who participate in discussions about rape. They often feel indignation and sometimes identify with both the victim and aggressor. They may feel their masculinity is violated by both the attack on a woman who is felt to "belong" to them and by their own helplessness deriving in part from early feminine identification as well as from their actual failure to have prevented the attack. This may be particularly threatening to men who need to reject any latent feminine components of their own personalities and may lead to a defensive identification with the rapist in an attempt to escape the anxiety of their own sense of vulnerability. Some men have difficulty coping with the impulse for revenge, which would reestablish their sense of control and the ability to protect "their" women.

A man whose daughter, girlfriend, or wife has been raped may react by becoming overprotective, partly as a result of his sense of guilt for not having been protective enough. However, it may also evolve as a defensive means of handling his anger at the attacker of "his" woman and at the woman for having allowed herself to get in this position. A complex series of feelings about his own sexual impulses may be evoked, and a man may find himself unable to be supportive or helpful to the woman after the rape, despite a previously close relationship. He may have difficulty with his own rape fantasies, his concerns about "used merchandise," and even the breakthrough of homosexual impulses. He may withdraw from the woman as a result of this anxiety. The woman who is deprived of support from a man who is important to her is particularly vulnerable to adverse reactions after a rape. The man may be unaware that he is not supportive, since denial operates to minimize the experience so it can be forgotten.

LIFE STAGE CONSIDERATIONS

It is difficult for anyone to predict how he/she will actually behave in a crisis. In the state of panic evoked during a rape, most women think about how to behave to avoid being physically injured or killed. Some talk, some resist, and others become passive, depending on their assessment of what is going on and their past styles of managing stress (14). There are, however, some specific issues related to age and life stage.

The Young Single Woman

The single woman between the ages of 17 and 24 is the most frequently reported rape victim. She is vulnerable often by virtue of being alone and inexperienced. Her relations with men have frequently been limited to the trusted, caring figures of her childhood or the young men she dated in high school. She enters the adult world with little sophistication in some of the nuances of human interaction, and she may easily become involved in an unwelcome sexual encounter. In this age group, the frequency with which rape victims report prior knowledge of the rapist is striking, and this is often the reason for a victim's refusal to prosecute. A young woman may have been raped by a date, an old friend, or even an ex-husband, and she oftens reproaches herself because she should have "known better" or been more active in preventing the rape.

As was discussed earlier, feelings of shame and guilt are prevalent regardless of the circumstances of the rape; coupled with the victim's sense of vulnerability, these feelings color the victim's future relationships with men. This is especially true for the very young woman who may have had her first sexual experience in the context of violence and degradation.

The experience of rape may revive concerns about separation and independence. A young woman's sense of adequacy is challenged when she asks, "Can I really take care of myself?" Parents, friends, and relatives often respond with an offer to involve themselves in taking care of her again. Although the offers may be supportive and reassuring, they may also foster regression and prevent mastery of the stress and conflict evoked by the experience.

Problems for the younger rape victim also affect her perception of and tolerance for gynecological examination. She may have suffered physical trauma, she is susceptible to venereal disease, and she may become pregnant. An examination is indicated, but it may be perceived, especially by an inexperienced or severely traumatized woman, as another rape. She is concerned about the intactness and integrity of her body and wants reassurance. However, she may have difficulty dealing with the necessary procedures if they stimulate memories of the original rape experience.

The Divorced or Separated Woman

The divorced or separated woman is in a particularly difficult position because she is more likely to be blamed and have her credibility questioned. Her lifestyle, morality, and character are frequently questioned. Her apparent sexual availability makes her seem more approachable sexually. She may experience the rape as a confirmation of her feelings of inadequacy, and she is especially likely to feel enormous guilt that can lead to failure to obtain aid or to report the crime. Her ability to function independently is challenged. If she has children, she may worry about her ability to protect and care for them, and others will probably raise questions about her adequacy as a mother. The woman with children must deal with the problem of what, how, and when to tell them about the rape. If the event is known in the community, its implications for her and her children may be difficult to manage.

The Middle-Aged Woman

For the middle-aged married woman, issues of her ability to have control and her concerns about independence are particularly important. She is often in a peri-

od of critical reassessment of her life role, particularly in the face of changed relationships to her grown-up children. Husbands in their own midlife crises are often less responsive and supportive to their wives' sexual and emotional needs.

There is a common misconception that a woman, married or single, who is past her most sexually active period has less to lose than a younger woman. One cannot quantify the self-devaluation, feelings of worthlessness, and shame in any woman—especially a woman who may already be concerned about her sexual adequacy.

ATTITUDES OF PROFESSIONALS

Until recently, many psychiatrists felt that rape was not a psychiatric issue and that psychiatrists had little to offer the rape victim. They often shared the view that the victim "asked for it," and she was seen as acting out her unconscious fantasies and therefore was not a "true victim." Thus the woman who had been raped did not receive the empathy and understanding usually extended to people in a crisis. There is also the common belief that many accusations of rape are false. We have not found this to be true in the majority of cases seen at the Rape Crisis Intervention Program at Beth Israel Hospital, nor have others in this field with whom we have spoken.

Professionals have shared the image of the rape victim as a young, sexually attractive woman who in some way exposes herself to an avoidable danger or uses the accusation of rape to save herself from criticism. This view of rape implies that it happens only to marginal people, who collude in some way, and this idea fulfills several functions. It protects the individual who accepts the view from anxiety about his/her feelings of vulnerability. It is also another way to deny that rape occurs and that its incidence is increasing. This defensive position is further expressed by the focus on the sexual aspect of rape. If it is sexual, then one can think that the victim and the rapist were both seeking sexual gratification. The professional is thus protected from any sense of guilt or responsibility.

In our own experience in the development of a rape crisis program at Beth Israel, we saw a change of attitudes in participating professionals. An increase in interest results in recognition of the crisis nature of the experience and increased dignity for the individual victim.

CONSEQUENCES OF RAPE

Attention to the long-term effects of rape is important. It is difficult to predict all the long-term needs of the rape victim, since the working out of the trauma proceeds in many different ways. The feelings aroused may lead to behavior that seems out of character and would be puzzling if it were not for the rape. Some of the issues that reemerge in some women at a later time are 1) mistrust of men, with consequent avoidance or hesitation; 2) a variety of sexual disturbances; 3) phobic reactions to situations that are reminiscent of the rape; and 4) anxiety and depression, often precipitated by seemingly unrelated events that in some small details bring back the original trauma.

COUNSELING CONSIDERATIONS

Counseling of rape victims should involve an assessment of previous adjustment, including stress tolerance and adaptive resources. In addition, it is also important to learn whom in her environment the victim views as supportive and to attempt to involve these people if possible.

The woman in this situation needs support and reassurance about the way in which she handled the encounter and her efforts to cope afterward, even if she seems volatile, disorganized, or guilty. Negative countertransference feelings may be evoked if she displaces her anger onto those who are attempting to help, e.g., friends, doctors, or the police. It is important that she have the opportunity for constructive catharsis with a caring and empathic person. The counselor's patience may be tested by the victim's repetitive retelling of the story. The counselor may need to be available frequently for the more overtly upset rape victim. The more subdued victim may need to be encouraged to communicate and should be offered the opportunity for future counseling.

Each woman presents special considerations and requires the acknowledgment and support of the counselor in verbalizing and working through the complex problems she faces. The young woman needs help in confronting her family, her relationships with men, and her feelings about her sexuality; the woman with children must deal with her communication with them; and the older woman may have to face her sexual anxiety more openly.

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Coping Behavior of the Rape Victim

BY ANN WOLBERT BURGESS, D.N.SC., AND LYNDA LYTLE HOLMSTROM, PH.D.

The coping behavior of rape victims can be analyzed in three distinct phases–the threat of attack, the attack itself, and the period immediately thereafter. The authors analyzed the reported coping behavior of 92 women diagnosed as having rape trauma. Most of the women used verbal, physical, or cognitive strategies when threatened, although 34 were physically or psychologically paralyzed. The actual rape prompted coping behaviors in all but 1 victim. Escaping the situation or the assailant is the primary task immediately after the attack. In counseling the rape victim, it is important to understand her individual style of coping, to be supportive of it, and to suggest alternatives for future stressful situations.

THE BEHAVIOR of people faced with critical life situations is receiving increased attention in the literature (1). The immediate efforts people use to deal with highly stressful situations are an important assessment point for clinicians who see patients in acute crisis. These coping behaviors may be viewed as problem-solving attempts an individual makes when facing demands that are highly relevant to his/her safety and that tax adaptive resources (2). Before describing the various coping strategies used by rape victims facing attack, it will be useful to review some coping strategies found in other types of crisis or stressful situations.

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Dr. Burgess is Associate Professor of Nursing and Dr. Holmstrom is Associate Professor of Sociology, Boston College, Chestnut Hill, Mass. 02167.

Researchers have focused on different aspects of the coping process. Extrasensory awareness of disaster situations was studied by Stevenson (3) in 19 people who survived the sinking of the Titanic. Glass (4) considered behavior over time in order to be able to view various phases of behavior separately. Most of the clinical studies have reported on the use of psychological mechanisms as part of the coping process: examples include denial in people with acute myocardial infarction (5), isolation of affect, denial, and regressive behavior in victims of concentration camps (6), denial, isolation of affect, and motor activity in parents of children with malignant disease (7), and emotional constriction, repression, suppression, denial, and delusion-hallucination formation in severely burned patients (8). Lazarus (9) took a behavioral approach and classified four important coping reaction patterns: 1) actions aimed at strengthening the individual's resources against harm, 2) avoidance, 3) attack, and 4) inaction.

There have been several recent studies of victim crisis situations involving major crimes, including forcible rape and situations in which victims have successfully interrupted or prevented attack (10–12). Giacenti and Tjaden (13) reported that out of 915 cases in the Denver area, 319 victims were able to interrupt the rape by active resistance, fleeing, physically fighting, crying aloud, verbal refusals, and outside intervention. In studying rape prevention, Brodsky (14) has focused primarily on verbal or vocal responses in the interpersonal prerape situation.

We saw a variety of ways to look at the coping behavior of rape victims in the literature. We drew on these studies and took a combination of areas to analyze, emphasizing the thoughts, feelings, and actions of rape victims as they related to specific time phases of the attack.

TABLE 1
Coping Behavior of 92 Rape Victims in Response to Threat of Attack

Description of Behavior	Number of Victims
Victims with strategies (N=58)*	
Cognitive assessment	18
Verbal tactics	57
Physical action	21
Victims without strategies (N=34)	
Physically paralyzed	22
Psychologically paralyzed	12

* Many victims had more than one strategy; therefore, the total numbers exceed 58.

METHOD

We interviewed all persons entering the Boston City Hospital Emergency Department over a one-year period with the chief complaint of having been raped. Subjects were interviewed at the hospital, usually within hours of the attack. The interview included a series of open-ended questions in which victims were asked how they felt and reacted to the circumstances prior to the attack, the attack itself, and the chain of events following the attack. Follow-up interviews were possible with 85% of the total sample of 146 victims and included further recounting of the details of the attack. Diagnostic categories were devised from the total sample, and a subsample of 92 women aged 17–73 diagnosed as having rape trauma (15) was used in this analysis of coping behavior before, during, and immediately following the attack.

BEFORE THE ATTACK: COPING BEHAVIOR IN A THREATENING SITUATION

Most of the victims perceived the rape as a life-threatening experience. The minority who did not so perceive it still saw the rape as an acutely stressful, frightening, and degrading experience. For almost all victims, this attack was something far out of the ordinary that seriously taxed their adaptive resources.

Early Awareness of Danger

Appraisal of the degree of danger, threat, or harm is a psychological process that intervenes between a stressful event and coping behavior. This early awareness may be cognitive, perceptual, or affective—often, the victim describes it as a "sixth sense" or a feeling of impending danger. The coping task during this phase is to react quickly to this warning.

Only 15 of the 92 women we interviewed spontaneously reported some cognitive or perceptual awareness of the potential danger they faced, and they were not totally clear about the nature of the danger—they just knew that something was wrong. Reports of this vague, obscurely formulated consciousness of danger varied. Victims said they saw a strange man and either thought he might do harm, wondered why the man had been hanging around all evening, remembered seeing the man before, looked at the car that pulled up, thought it strange the apartment light did not go on, or heard a noise in the kitchen and went to investigate.

Several victims described a subjective awareness alerting them to danger such as being scared when their boyfriend was pushed out of the car or feeling frightened when they realized what was happening.

Threat of Attack

The threat of attack is the point when the person realizes there is definite danger to his or her life—the coping task at this stage is to attempt to avoid or escape the situation. The person is aware something critical is going to happen but may not realize that rape is the imminent danger; for example, the person may instead fear robbery or aggravated assault.

Coping behavior was analyzed in terms of whether or not victims were able to react to the confrontation with danger. This ability to react often depended on the amount of time between the threat of attack and the attack, the type of attack, and the type of force or violence used.

A majority of victims used one or more strategies and a minority of victims were unable to use any strategies (see table 1).

Basic Strategies

Cognitive assessment. Victims may cope by mentally assessing the situation to determine possible alternatives, e.g., they may think about how they can get away from the assailant's grasp or escape from a car or room safely, or they may worry that the man will panic and hurt them and plan how to keep calm.

Verbal tactics. The majority of the coping strategies were verbal; they included talking one's way out of the situation ("I tried to engage them in conversation such as asking where they went to school and why they were doing this"), stalling for time ("I tried to talk to him; tried to get him to come for coffee at a restaurant down the street"), reasoning with the assailant by trying to change his mind ("I'm a married woman"; "I'm a virgin"), trying to gain sympathy from the assailant ("Look at the trouble you're causing me"; "What will I do?") using flattery ("You're an attractive man; surely you don't have to do this for sex"), bargaining with the assailant ("There's my TV, take it and go"), feigning illness ("I'm sick"), threatening the assailant ("My husband is due home"; "My kids are in the next room"; "A policemen lives in this building"), verbal aggression ("Get your hands off me"; "Don't touch me"; "What are you doing?"), changing the assailant's perception of the victim ("I talked to him like a mother."), joking and sarcasm (a woman awakes to see a man coming in her room saying, "I'm escaping from the police"; she says, "OK—I'll let you out the back door").

Physical action. Some victims took direct action aimed at preventing the attack by fleeing from the situ-

ation or fighting the assailant ("I tried to stab him with the broken glass"; "I tried to push him back out of the apartment").

Lack of Strategies

One-third of the victims were unable to use any strategy to avoid the attack. The victim might be physically paralyzed and totally overpowered by the assailant. For example, several victims were in their beds sleeping when the assailant gained access to their apartments and attacked them ("It was around 3 a.m. . . . I woke to feel someone jumping on me"). The victim might be walking down the street and suddenly grabbed by one or two assailants. The use of a weapon often paralyzed a person. The following example from a referral case illustrates early awareness of danger and physical paralysis: "The door buzzer rang . . . I was expecting friends and opened my door . . . saw three men with a paper in one's hand . . . I froze . . . paralyzed for a moment . . . something went through my head . . . shut the door but they pushed it back open . . . with the gun . . .." In some cases, the victim was totally stunned or surprised by the change in behavior of a man whom she knew as a friend, neighbor, or acquaintance, and said "He just grabbed me before I knew it."

Victims may be psychologically paralyzed either through their defensive structure ("When I realized what he was going to do I blanked out . . . tried not to be aware of what was going on") or because of their use of alcohol or drugs prior to the attack. Thoughts of death may paralyze a victim ("I thought he'd be the last person I'd see alive").

Multiple Strategies

Victims may try a number of strategies: 31 women had multiple strategies, 27 had one, and 34 had none. One woman who was successful in avoiding attack said, "First I tried to calm him down; tried to talk softly to him and said, 'OK, we be friends.' Then I said my brother was due home any time . . . I tried all I knew, verbal and physical . . . I screamed and fought . . .." The brother did come home and the assailant left without completing the rape.

Another victim who tried several strategies was not successful with three men who had forced her into a car as she waited for a bus after her evening classes at a local university. She tried verbal tactics ("My husband will be worried and probably call the police if I am not home"), but the assailants told her that such remarks would "get me dead." The victim then became silent. She later tried bargaining ("I offered them my money to let me go") and finally decided to comply ("I decided the only way was to play it their way").

In another case, one can see the coping behaviors of early awareness, an affective reaction of fear, cognitive assessment, and verbal tactics of joking: "I got a warning . . . saw two men at the end of my hall . . . got frightened . . . didn't know how they got there. They said they needed to use my phone. I tried to joke and said, 'Who you trying to call, Red China?' "

TABLE 2
Coping Behaviors of 90 Women During Rape

Description of Behavior	Number of Victims
Cognitive strategies	28
Affective response (N=25)	
Crying	17
Anger	8
Verbal strategies (N=23)	
Screaming	14
Talking	9
Physical action	23
Psychological defense	17
Physiological reaction	10
No strategy	1
No data	8

DURING THE ATTACK: COPING WITH THE RAPE

At the moment of the actual rape attack, it becomes clear to the victim that forced sexual attack is inescapable. The coping task during this phase is to survive the rape despite the many demands forced upon the victim, such as oral, vaginal, and anal penetration. She also may be forced to have conversation with the assailant. Victims coped with the actual rape in a variety of ways, as table 2 indicates.

Cognitive Strategies

Victims often cope by mentally focusing and directing their attention to some specific thought to keep their minds off the reality of the event and focus on their survival. Remaining calm was a strategy in which the victim specifically controlled herself mentally so as not to provoke additional violence. The victim might talk to herself, as in the following case:

> I kept thinking . . . keep cool. He said he'd kill me. He hit me, he choked me, he could kill me. I said to myself, 'You can handle anything; come on, you can do it.' I decided not to fight him . . . he was holding my neck so tight . . . I responded a little to him . . . that blew his mind that I acted as I did. It was very quick, thank God.

Memorizing details was a strategy that paid off later in some cases. One referred victim said, "I focused on their faces and thought to myself, 'I'll see you guys in court if I get out of this alive.' " She did. Another victim said, "I played detective . . . tried to observe everything, like the tatoo on his arm, remarks he made, route of travel of the car, license of the car."

Recalling advice people have given on the subject of rape is another strategy victims report: "I remember a conversation I had with my husband. He said if I was attacked not to resist if he wants sex. My husband said

the guy could kill me or the children . . . but sex wouldn't kill me." Another victim said, "I remember talking with people about rape and they always said not to resist . . . that a female could be killed, beaten, or mutilated. I didn't want that to happen."

Memories of previous violent situations provided some victims with alternatives ("I struggled a little . . . then remembered when I was 12 I fought a neighbor boy and got my nose broken"). Praying for help is a tactic to decrease stress and tension ("I wasn't listening to them but concentrating on praying that my friends who had keys to my apartment would come"). Concentrating on the assailant in terms of who he is and what has led to this attack is also a strategy ("I remember thinking that this person must have a home—must live somewhere . . . why would he do this on Mother's Day? . . . I thought of the irony of it . . .") Compliance is a strategy used to "speed it up . . . get it over with." To many victims, the attack seemed interminable.

Verbal Strategies

Victims combined verbal and affective responses by screaming and yelling. This tactic served both to relieve tension involuntarily and to deter the assailant from his full intent. This tactic brought police to the scene in several cases, which sometimes resulted in the assailant being apprehended during the attack.

Several victims believed that talking with the assailant during the attack helped them avoid additional violence. The assailant may demand to know how the victim is "enjoying" the rape. One victim handled the situation as follows: "He kept wanting to know if it felt good and I had to say yes to keep him happy . . . He said, 'I'm on drugs lady and I need money . . . fuck me good or I'll kill you.' He needed to be reassured."

If the attack continues over a period of time, the victim may try verbal tactics to calm the assailant and thus avoid further demands ("I talked to calm him down . . . I asked questions and he kept talking"). Sarcasm may be used as a coping strategy, especially if that is the victim's usual verbal style.

> As he was molesting me he asked if I enjoyed it and I said, "Oh, sure, it is great." I decided to go along with him. He seemed to need reassurance . . . I wasn't scared then. First thought he'd get his kicks and then it'd be all over—I'd be dead. I got faith that he wasn't going to harm me.

Some victims tried to gain control in the situation by scaring the assailant(s) ("You'll be in real trouble if you kill me"; "You'll be sorry . . . I'll get someone to kill you"). This strategy was partially successful in some cases.

Physical Action

Victims reported struggling and fighting with their assailants to avoid penetration ("I struggled and tightened my muscles"). Sometimes the victim struggled to a certain point and then stopped ("I fought and struggled until I realized he was going to rape me. He wanted to rape me more than I could manage to resist"). Some victims quickly discovered that struggling and fighting were just what the assailant wanted ("The more I screamed and fought, the more excited he would get").

Psychological Defenses

Defense mechanisms are another way rape victims cope with the overwhelming fear of attack. Defenses close the cognitive field and thus block out the unbearable feeling. Some women denied the attack ("I never thought it could happen"), experienced dissociative feelings ("I pinched myself to see if I was real"), suppressed the rape ("I am missing 10 minutes of my life"), or rationalized ("I felt sorry for him if this was the only way he knew how to get sex"). One victim described her reaction as follows: "I did not struggle because of the knife. All those things you read about or plan to do don't help . . . I felt I was not going to get out alive . . . I was resigned; I felt nothing, empty; felt this can't happen to me."

Physiological Response

Not all coping behavior is voluntary and conscious. Certainly, some screaming and yelling is involuntary, and victims also reported physiological responses of choking, gagging, nausea, vomiting, pain, urinating, hyperventilating, and losing consciousness. One victim described an epileptic seizure:

> Only thing I remember is getting the key into the lock to get into the building. Then I got warning signs to my seizure attacks . . . getting overheated and the ringing in my ears and that's all. When I regained consciousness I was in the hall by the door to the basement. I dragged myself out as I heard someone saying, "Who left their keys in the door?"

Another victim described how her involuntary reaction scared the assailant off after he raped her: "I felt faint, trembling and cold . . . I went limp. I think he got scared and thought I was out."

AFTER THE ATTACK: ESCAPING FROM THE ASSAILANT

The stressful situation is not over when the actual rape ends. The victim must alert others to her distress, escape from the assailant, or free herself from where she has been left. The coping task immediately following the rape is to be free or escape from the assailant.

Alerting others. Victims are always hopeful that someone will come to their aid, and they may spend time concentrating on how to obtain help.

Bargaining for freedom. Often, the victim must negotiate with the assailant through a bargaining process. The assailant sometimes apologizes and tries to gain sympathy and thus get the victim to promise not to tell, or he may give the victim orders or instructions

("I'll kill you if you tell or go to the police"; "Don't move from that position for 30 minutes"). During the bargaining process the victim may cope by remaining silent or agreeing to instructions. Some victims promised not to tell anyone or invented stories to preserve their lives: "I told him my girl friend had this happen and when she went to the police they didn't believe her. I told him I'd never go to the police."

Freeing oneself. The victim may have to free herself from the situation—if she has been tied and gagged, she has to cope with physically freeing herself. Cognitive assessment of the situation and keeping calm will be most useful strategies to her, as was the situation in the following referral case: "I lay still for a moment . . . then realized that the faster I got myself untied, the faster I could get to the police and my friends . . . ankle ties . . . getting cramps in my legs . . . so I had to tell myself not to panic and I worked the wrist ties and ankle ties next . . .."

Mastery, in terms of survival of the attack, may be verbalized by the victim as well as her family and friends when this stage has been managed successfully: "The worst is over . . . I got through it . . . I am grateful to survive."

Once free of the assailant, the victim must still cope with the stress of the aftermath of the rape. Victims are often immediately faced with trying to cope with the institutions that are set up to deal with rape (16).

COUNSELING IMPLICATIONS

Understanding a person's coping behavior when faced with a life-threatening situation is an essential step in crisis intervention. Rapoport (17) noted that patterns of coping in crisis situations may be adaptive or maladaptive. Parad and Resnik (18) stated that the purpose of actively focused crisis intervention is to steer the person toward adaptive coping and away from maladaptive behavior. Adams and Lindemann (19) formulated coping principles drawn from a study of catastrophic disabling injuries; they emphasized identifying the acute crisis and the psychological means by which the crisis is to be managed if not mastered.

The assessment of coping behavior and strategies provides the clinician with two therapeutic measures. First, such assessment can be used as a supportive measure. By listening to the victim recount the rape, clinicians can identify the coping behavior and acknowledge this information to the victim. This support tells victims that their coping behavior was a positive adaptive mechanism used to survive a life-threatening situation. By verbally conveying this information, the clinician attempts to alleviate some of the guilt suffered by a victim who may think, "I did not do enough—I could have done more." This therapeutic approach sets in motion a positive sense of self-esteem and worth. As was noted by one victim, who was raped by two assailants at knife point, "At least I wasn't so whacked out that I couldn't get a description and license of the car." Reaffirming the strategic use of her cognitive abilities helped this victim to see herself as a person who was able to *do* something in a highly stressful situation, even though she was physically immobilized. Such a response by the clinician strengthens the therapeutic alliance in terms of providing positive expectations regarding the victim's capacity to restore herself to her precrisis level of functioning. Thus we see that Murphy's model of resilience (20), wherein a person sees herself as someone who is resilient and expects to recover from the trauma, has significance in counseling rape victims.

The second use of assessment of coping behavior is to give the clinician a reference point from which to begin the clinical negotiation for crisis service (21). As the victim states her request and it is negotiated, additional requests may be elicited. For example, one request may be related to primary prevention in terms of what other strategies might be used in such situations. The clinician can explore with the victim the crisis behaviors she used and analyze their effectiveness. New strategies might be suggested as possibilities in future stressful situations. It is important to help expand the person's problem-solving capabilities (22).

We believe our analysis of the coping behavior of rape victims opens up other research areas. There are two questions of major importance: 1) what coping strategies seem to result in less psychological trauma after a rape? 2) What additional therapeutic techniques are needed to aid the rape victim?

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Development of a Medical Center Rape Crisis Intervention Program

BY SHARON L. MCCOMBIE, M.S.W., ELLEN BASSUK, M.D., ROBERTA SAVITZ, M.D., AND SUSAN PELL, R.N.

The Rape Crisis Intervention Program at Beth Israel Hospital utilizes volunteer multidisciplinary counseling teams drawn from psychiatry, social work, psychology, and nursing staffs. The premise of the program is that early crisis intervention can prevent later development of psychological disturbances in victims. Counselors accompany victims throughout emergency room procedures; follow-up begins 48 hours after the initial contact and continues at regular intervals for at least a year. The authors discuss the problems of implementation, which include staff resistance, funding questions, and varying levels of counseling sophistication, and describe how these difficulties have been handled in their program. They note that this program is becoming a resource center for the community.

RAPE is a physical, social, and psychological attack upon the person. It is the fastest rising violent crime in the United States: the FBI reported a 68% increase in rape (from 31,000 to 51,000 cases) between 1968 and 1973, and police figures in Boston show a 43.5% increase between 1972 and 1973 (1).

Rape has been viewed primarily as a sexual rather than a violent assault. The traditional assumption is that the woman in some way invited the attack. This attitude obscures recognition of the trauma experienced by the victims and interferes with the development of adequate community and institutional resources to treat them. This is reflected in the absence or fragmentation of medical and psychological care for rape victims.

In response to this problem, a comprehensive support system for the rape victim has been developed at the Beth Israel Hospital, a metropolitan teaching facility oriented toward community medicine. Particularly in urban areas, hospital emergency rooms are the typical health care facility utilized by victims at the time of the rape. Prior to the development of our project, emergency room records showed that an average of one victim a week received medical care during 1972–1973. The psychiatric service was consulted only if the victim presented a management problem for the emergency room staff. Follow-up was generally inconsistent. Clinicians were impressed, however, by the number of women who appeared at the Psychiatry Clinic months or years later for whom the rape was a major component in the presenting symptomatology. At the time of the assault, these women had not sought psychiatric assistance. Indeed, many had never told anyone about the rape.

The Rape Crisis Intervention Program is based on the premise that early intervention can prevent the development of psychological and psychosomatic distur-

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The authors are with Beth Israel Hospital, 330 Brookline Ave., Boston, Mass. 02215, where Ms. McCombie is Director, Rape Crisis Intervention Program, Drs. Bassuk and Savitz are Assistants in Psychiatry, and Ms. Pell is Head Nurse, Inpatient Psychiatric Unit. Drs. Bassuk and Savitz are also Instructors in Psychiatry, Harvard Medical School, Boston, Mass.

bances. The program's aim is to integrate and expand available medical services and provide psychiatric crisis counseling (2). This paper will describe some of the problems encountered in implementing such programs in a medical setting.

RAPE AS A LEGITIMATE HEALTH ISSUE

There is both community and hospital resistance to recognizing rape as a legitimate health issue requiring medical and psychological services. In the past, the psychiatric community has not participated in providing resources for victims. Throughout the country, most services come from grass roots women's organizations, which often view psychiatry as a male-dominated, pathology-oriented profession that stigmatizes the victim by labeling her a mental patient.

Because hospitals have only recently acknowledged the needs of rape victims, feminist organizations have tended to regard these institutions as part of the problem. These women's groups have attempted to minimize the victim's contact with health professionals by establishing alternative facilities. However, there is no way to bypass the necessity for professional gynecological services. The medical, surgical, and psychiatric resources of the emergency room, their 24-hour availability, and the legal requirement for medical evidence bring the victim into the health care system. Because of the multidisciplinary make-up of the general hospital, it is a potential center for comprehensive treatment of both the physical and emotional needs of the rape victim.

Hospital resistance to mobilizing supportive services can be traced to two major sources. First, staff prejudices about rape can jeopardize the respectful, thorough treatment of the victim. Second, staff working in a treatment model geared to respond to medical and surgical emergencies may underestimate the significance of emotional crises. If the woman does not exhibit wounds that testify to her victimization and unambiguously signal her need for medical assistance, her status as a legitimate consumer of health services may be questioned. Regardless of the presence or absence of physical injury, the emotional trauma of forced violation—frequently under threat of death—establishes the victim's right to sensitive professional care.

PROGRAM DESCRIPTION

There have been four major objectives in the implementation of our service for rape victims:

1. Providing both immediate and follow-up counseling that is aimed at resolving the psychological crisis.
2. Encouraging emergency room personnel to respond sensitively to the emotional needs of the victim.
3. Developing an understanding of the special needs of this patient population in order to provide expert consultation to community and professional groups.
4. Conducting research on the acute and long-term impact of rape on life adjustment.

There are still many unanswered questions about the psychodynamics of rape trauma and the adult victim's reactions and long-term adaptation (3–5). To provide quality crisis counseling and assessment and to gather sound clinical observations, we needed mental health clinicians on the staff of our program. Because of this need and the funding problems we encountered, a volunteer counselor roster was organized. The counseling team is a self-selected group of volunteer personnel from psychiatry, social service, psychology, and nursing. This multidisciplinary approach distinguishes our program from the single-discipline model used in other hospitals (6, 7).

Training and supervising a multidisciplinary group of volunteers is complicated by the various levels of counseling sophistication that are represented in the team. Individual learning needs are addressed in weekly supervisory sessions based on the model used for teaching psychotherapy. All members of the counseling team are required to attend a series of weekly didactic seminars on crisis intervention techniques and the special problems of rape victims. Nurses, gynecologists, and other interested hospital personnel are encouraged to attend these meetings.

The volunteer model has proved to be cumbersome, unreliable at times, and difficult to coordinate. However, it has had the advantages of sensitizing a large number of hospital personnel to the emotional needs of the rape victim and of encouraging awareness of psychological issues in the treatment of all general hospital patients.

The 24-hour on-call system ensures that a counselor will be available to accompany the victim throughout all emergency room medical procedures. The initial role of the counselor is to provide emotional support and information to the victim. The counselor also meets with friends or relatives who have accompanied the victim to discuss their concerns and mobilize their support. Follow-up begins within 48 hours after the initial emergency room contact. Subsequent contacts are made at regular intervals for at least 1 year. The frequency and content of the intervention is based on the needs of the victim, as determined by clinical assessment and timing of anticipated periods of exacerbation of symptoms, such as court appearances.

The counseling goal is to increase the individual's adaptive capacity by delineating and working through the crisis-related issues. If the victim has unusual difficulty resuming her precrisis level of functioning, she is referred for psychotherapy.

ORGANIZATIONAL INTERFACES AND POLICY ISSUES

Official sanction from the Department of Psychiatry was crucial to the implementation of our program.

Staff time was requested for the recruitment, training, and supervision of the counselor team. Initially, there was hesitation about supporting the program because of the demands on staff time. There was also a tendency to see rape as a social problem rather than as a crisis warranting psychiatric intervention.

Since crisis intervention techniques could be taught with rape counseling as a model, the program was consistent with the Psychiatry Department's commitment to training and its active hospital liaison program, which integrates psychological understanding with patient care (8). At the same time, the growing public awareness of the needs of women helped to offset objections stemming from the controversial nature of our target population.

The chiefs of social service, nursing, and medicine were approached to enlist their cooperation in personnel training and coordination of service delivery. This initiated a continuing dialogue with these departments carried on through periodic visits to their staff meetings.

In order to establish administrative backing, we had to develop a policy regarding payment for counseling services. Currently, the woman is charged the routine fee for the emergency room visit and is not billed for the counseling services. The cost is absorbed by the hospital as loss of staff hours from the professional volunteers' regular duties.

There is a continuing debate as to whether the hospital, the community, or the victim should be responsible for the costs incurred. One argument is that, as in all preventive health care programs, the treatment of rape victims is cost effective in the long run. However, because of the economic recession and the rapidly rising overhead of hospitals, each program must demonstrate its cost effectiveness.

A variety of complex factors must be considered before an optimal financial policy can be determined. These include the following:

1. The majority of the victims are young, single women with low incomes, inadequate health insurance, and doubtful ability to pay.
2. Some of the medical procedures are for the collection of evidence for the state's case against the alleged criminal rather than for the victim's health care.
3. There is clinical concern about the meaning transmitted to the victims when they are set apart from other consumers of health care who pay for services.

GYNECOLOGY AND EMERGENCY ROOM LIAISON

We have emphasized liaison with the gynecology and emergency room staffs. The mechanics of the gynecological examination are usually handled with skill (9). However, anxiety about how to respond to a woman who has just been raped can lead to staff withdrawal or denial of the event. Female staff members are reminded of their own vulnerability. Male staff members become acutely conscious that they may be the first man the victim sees after the rape. The victim's anger and helplessness are frightening, and there is a tendency to counterattack or to infantilize.

The medical examination of the rape victim occurs in the interface between the health and legal professions, since the physician's findings may become evidence in a court of law (10). Since the doctor can be required to give testimony, he may hesitate to become involved or may respond by trying to judge for himself whether the case is a "real" rape.

The protocol of the American College of Obstetricians and Gynecologists for the examination of rape victims is a detailed procedure for the gathering and preservation of specimens for evidence (11). It has the advantage of providing complete and careful documentation. Nevertheless, in the pressures of the emergency room, it may be regarded as a tedious chore. Other factors affecting the physician's response to the rape victim are his concept of his professional role, his personal feelings about rape, and his attitudes toward the legal and penal systems.

Gynecology and nursing meetings have been used as forums to identify and discuss staff concerns. Treating victims of violent sexual assault is openly acknowledged as stressful. Through the use of videotaped interviews with victims, role playing, and discussion, personnel are sensitized to the stresses the victim experiences in the emergency room.

When a woman who has recently been raped takes on the label of patient, she is assuming a role characteristically associated with being damaged and helpless. The victim-as-patient assumes a status that reinforces the helplessness and fears about being damaged that are natural sequelae of the rape experience. Her physical and emotional pain become tangible reminders of her inability to protect and to defend herself. The confusion and lack of privacy in the emergency room can prolong and compound her discomfort. A pelvic examination done abruptly can be experienced as a repetition of internal invasion. The hospital becomes an institutional transference object upon which both negative and positive feelings are focused. The staff must be equipped to deal with the intense affects aroused and to help the victim work through them. The rape crisis counselor is a consultant to medical and nursing staff in the emergency room, and his/her interpretation of the victim's behavior and empathy with the medical staff's feelings reduce anxiety and enhance sensitive treatment of the patient.

The introduction of a new program staffed by nonemergency-room personnel into the routine emergency room system has presented several unsolved problems. For example, while our counselors relieve the nurses of certain pressures and responsibilities, there is also a mixed message about the nurses' role and competency. Although we have explicitly invited them to be trained as rape counselors, few are able to do so because of the demands of their regular duties. We have approached this difficulty by including an emergency room nurse as a member of the program's ad-

ministrative and planning group; this has resulted in an improved cooperative team effort and, secondarily, has supported nurses' continuing efforts to expand their role.

DISCUSSION

Although our program is still in the experimental phase, progress has been made in establishing rape as a legitimate health issue and in including the care of rape victims among the repertoire of services provided within a medical center. The liaison work has been essential in successfully setting up a multidisciplinary program of this kind. The most important interfaces have been with the Departments of Psychiatry, Nursing, and Gynecology. Our continuing dialogues with these departments have emphasized the generalizability of our crisis approach to other patients.

As organizational issues are settled, the program will increasingly concentrate its efforts on developing research on the long-range resolution of the rape crisis and will attempt to differentiate the implications for women at various stages in the life cycle. This information is critical for optimally effective service and for evaluation of the preventive impact of early intervention on later adjustment. The didactic series used in teaching our counselors is being refined and compiled into a training manual for use by other hospitals. The Rape Crisis Intervention Program is rapidly becoming a resource center for the community through consultation and education programs for police, educators, health professionals, and the public. Our aim is to encourage and assist other community medical facilities in providing a support system for women dealing with this life crisis.

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Assaults on Psychiatrists by Patients

BY DENIS J. MADDEN, PH.D., JOHN R. LION, M.D., AND MANOEL W. PENNA, M.D.

The authors describe the results of a survey of 115 psychiatrists regarding assaults by patients. Forty-eight psychiatrists stated that they had been assaulted; a total of 68 assaults was reported. Psychiatrists were most often assaulted in the early stages of their career or while they were working in high-risk settings such as prisons or emergency rooms. Many reported retrospectively that they might have had a role in provoking the assault and that they could have anticipated it. The authors believe that psychiatrists avoid dealing with the issue of violence by patients for a variety of reasons, and they recommend that clinicians learn techniques for managing potentially assaultive patients.

THE RECENT American Psychiatric Association Task Force Report on Clinical Aspects of the Violent Individual (1) referred to a number of published studies regarding assaults on psychiatric staff within mental hospitals (2–8). These reports dealt for the most part with assaults by psychiatric patients on other patients or on staff personnel.

Ekblom (4) was able to review the literature on acts of violence by psychiatric patients from 1889 to 1970 in 2½ pages, which suggests that the literature is sparse. Ekblom cited only one study, by Kolle (4, p.10), which suggested that the attitudes of physicians and nursing staff might provoke acts of violence. Kalogerakis (5) reported incidents of assault and attempted to make psychodynamic and sociological interpretations in each case.

Our work with violent patients led us to question whether psychiatrists might either overtly or covertly provoke the violent behavior of their patients. This hypothesis, in addition to the lack of substantive data in this area, led us to undertake the present study.

Revised version of a paper presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

The authors are with the Clinical Research Program for Violent Behavior, Institute of Psychiatry and Human Behavior, Department of Psychiatry, University of Maryland School of Medicine, 6465 W. Redwood St., Baltimore, Md. 21201, where Dr. Madden is Clinical Assistant Professor, Dr. Lion is Associate Professor, and Dr. Penna is Assistant Professor.

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METHOD

We surveyed by questionnaire 115 psychiatrists who held full or part-time clinical appointments at the University of Maryland School of Medicine. This group represented a variety of clinical and administrative positions, including private practice, mental health clinics, medical school appointments, forensic and prison settings, and state hospital positions. Each psychiatrist was sent an initial questionnaire asking whether or not he had in the course of his career been assaulted by patients and what the nature, timing, quality, and other characteristics of the assault had been. Respondents who indicated that they had been assaulted were then interviewed personally or by telephone to elicit more data regarding the nature of the assault, the patient's diagnosis, and level of therapeutic involvement with the patient by whom they were assaulted.

In addition to sampling our own faculty, we published letters in the August 21, 1974, issue of *Psychiatric News* and the November 1974 issue of the *American Journal of Psychiatry* requesting information on assaults. As a result of these letters we received 15 responses relating 20 incidents of assault.

RESULTS

Although all of those sent the questionnaire did not respond to our first inquiry, a second questionnaire and phone calls resulted in a 100% response.

Of the 115 psychiatrists sampled, 48 responded that they had been assaulted, and 67 reported that they had never been assaulted. The total number of assaults reported in questionnaire responses was 68.

Psychiatrists who reported being assaulted varied considerably in age and level of experience, but most of the assaults occurred when clinicians were in early phases of training. The majority of psychiatrists were assaulted by patients who were in active treatment, and most of these therapists continued to see their patients after the assault occurred.

Some vignettes can give an indication of the type of incidents reported to us. In the following case there had been a long therapeutic involvement prior to the assault.

Case 1. The patient, a 41-year-old male, had been in therapy for 2 years. He had had several earlier attacks of psycho-

sis, during which he did bizarre things, but there was no record of previous violent assaults. At the end of one session, the therapist was saying good-bye when the patient suddenly lashed out with his right fist, catching the therapist on the jaw. One week later, while the therapist was writing a prescription, the patient stabbed him with a pin in the left thigh. The patient was emotionally upset and embarrassed, as he had been after the first attack. He indicated that the pin-stabbing signified a desire to be given an injection, since this had helped terminate a previous acute psychotic decompensation rapidly. He also saw the jabbing as a confused expression of dissatisfaction with the therapist, who had not made him well fast enough.

Another assault occurred when the therapist had had no contact with the patient.

Case 2. The patient was a 30-year-old male who had been hospitalized with a diagnosis of acute paranoid schizophrenia but had no history of violent behavior. The therapist was just beginning his 3-month rotation on psychiatry as an intern. He and eight other staff members were attempting to restrain a patient who had attempted to rape another patient and had hit several orderlies who tried to stop him. While this was taking place the assaulted patient suddenly grabbed the doctor from behind in a head lock. It was later learned that this patient thought that he was God and believed that the patient who had attempted to rape him was one of his "disciples." He felt the need to protect his disciple and grabbed the first "enemy" he came to.

Characteristics of Patients and Therapists Involved in Assaults

Table 1 summarizes the results of the survey. Psychiatrists who were assaulted usually sustained only minor injuries. However, one senior psychiatrist reported that he had been shot in the chest. A paranoid patient came into the therapist's office and informed him that he had to shoot him in order to stop some other doctors from giving him "bad medicine." The wound did not seriously injure the therapist, and he was able to resume his practice in a few weeks. Another serious injury was reported to us by a therapist who had had his nose fractured by a patient.

Table 1 also gives a breakdown of the types of assaults reported. Most were a slap to the face or a blow on the head. Some psychiatrists reported having objects such as an ash tray or other object from their desk thrown at them. One patient tried to set the therapist's clothes on fire, and another therapist was tied up by a group of patients. There was one report of an amorous "attack," in which the patient tried to kiss the therapist in what was perceived by the therapist as an inappropriate and assaultive manner.

The majority of patients who assaulted psychiatrists were judged to be psychotic, with diagnoses ranging from paranoid to catatonic schizophrenia. A smaller number were diagnosed as having borderline or personality disorders. The remainder of assaultive patients were diagnosed as alcoholic, hyperactive, and adjustment reactions. One patient (included in the "other" category) was diagnosed as depressed. The majority of patients were young; we encountered few older patients who assaulted physicians.

TABLE 1
Characteristics of 68 Assaults by Patients Reported by 48 Psychiatrists*

Item	Percent
Patient characteristics (N = 68)	
Age group	
1–10	6
11–20	25
21–30	37
31–40	12
41–50	13
51–60	4
61 and older	3
Diagnosis	
Schizophrenia	63
Other psychosis	9
Borderline syndrome	6
Personality disorder	6
Other	16
Treatment status	
In active treatment	57
Not in active treatment	43
Status of assaulted psychiatrists (N = 48)	
Resident	65
In private practice	16
Administrative position	19
Type of assault reported	
Head	41
Body	44
Object thrown	9
Set on fire	1.5
Shot	1.5
Tied up	1.5
Kissed	1.5

*Table based on data from questionnaires returned by University of Maryland faculty members.

Attitudes of Therapists toward Assault

We also questioned the therapists regarding their retrospective anticipation of the assault. This question, although somewhat nebulous, indicated our interest in the role of therapists in provoking assaultive responses on the part of patients. Fifty-five percent of the respondents reported that they could have anticipated the assault, and 53% felt they had acted in a provocative manner. A majority of the therapists felt that they could have anticipated the assault because they had made comments or interpretations that were unfavorably received by the patients. One therapist resented the notion that he or other therapists might have acted in a provocative role and compared this to viewing the rape victim as the provoker. He felt that society, the Viet Nam war, and the prevailing culture were more responsible than individual therapists for such assaults.

We asked clinicians what they thought triggered particular assaults. Among the responses received were: refusing to meet a patient's request, forcing a patient to take medication, setting too many limits or not setting enough limits, transference reactions, homosexual

panic, and the material being dealt with in therapy. Many therapists felt, at least in retrospect, that they had been at times too insistent that a patient confront upsetting material. Such therapists felt that the incident might not have occurred if they had backed off. Several therapists reported candidly that they did not particularly like the patient who assaulted them and might have projected this feeling to the patient. One therapist reported that after a patient had asked him five times to write a letter for him he responded in a flippant way that if the letter was so important the patient should write it himself.

In addition to asking what might have precipitated the particular assault reported, we asked what the respondents thought caused assaults in general. Inexperience and fear of patients, which at times are translated into an omnipotent facade, were mentioned by several of the clinicians interviewed. One senior psychiatrist said that it had been his experience that those residents who allowed patients to act out in therapy sessions were the ones who were most frequently assaulted. On a more personal level, some of the clinicians felt that therapists might act in a seductive manner, project an unconscious expectation that an assault will take place, or attempt to veil an underlying hostility with kindness, all of which can lead to assaultive behavior on the part of the patient. The following incident demonstrates how the feelings of the therapist may at times enter into an assault.

Case 3. The patient, a 30-year-old male, had been transferred from another hospital with a diagnosis of paranoid schizophrenia. He was one of the first patients that the therapist had treated as a resident. The patient had been in treatment for about a year, and during that time the therapeutic relationship had been a difficult one for the therapist. The patient exhibited much hostility and directed much sarcasm toward the therapist. Just prior to the attack, the patient had requested a pass to return to an area where he had been involved in inappropriate financial deals. At the same time the therapist was preparing to go on vacation. When the patient was told that he would not be given the pass he became furious and verbally threatening. The therapist was extremely anxious and also experienced difficulty in dealing with his own anger. During this stormy session the patient grabbed the therapist's shirt and tie and had to be restrained by an aide. In retrospect, the therapist felt he had been angered by the patient's persistent attacks on him, which had included critiques of his personality, mannerisms, dress, religion, and other aspects of his life. The therapist viewed the patient as one of the "nastiest" persons that he had ever encountered, clinically or otherwise.

Some clinicians were assaulted on several occasions. Several psychiatrists who sustained multiple assaults worked in rather dangerous settings, such as prisons, forensic units of state hospitals, or emergency rooms, where the potential for incidents of assault was higher. One physician from an indeterminate sentence facility prison hospital was assaulted on numerous occasions and commented that assaults on staff were quite common.

DISCUSSION

The finding that 42% of the clinicians surveyed reported having been assaulted by a patient was interesting and surprising to us. It is surprising because we believe that the majority of psychiatrists are attracted to psychiatry, on at least a theoretical level, because of its contemplative nature and its use of verbal skills rather than physical management as the primary tool for treating patients. This is in contrast to other medical specialties, in which physical handling of patients is more prevalent.

It seems reasonable to suggest that there are high-risk areas where psychiatrists stand in jeopardy of being assaulted by patients about whom they make decisions regarding incarceration, continued incarceration, or commitment. However, as our study shows, assaults were not limited to these individuals but also occurred among physicians who were in private practice and in a variety of other, less dangerous occupational settings.

It is our belief that denial plays a heavy role in both the ways clinicians deal with patients who might be assaultive and their recollection of assaults. One clinician who had been threatened many times by a patient and had tape recorded these threats on his life was still hesitant to take any action to prevent a potential assault. The alarming nature of patient assault on clinicians is brought home by reports of two psychiatrists who were killed by patients within the last two years. One psychiatrist on the staff of a state hospital in the Boston area was killed while attempting to disarm a patient, and another psychiatrist in New Haven was killed by a patient he had seen on only one occasion. According to several questionnaire respondents who mentioned the incident, this patient apparently incorporated the psychiatrist into a well-developed paranoid delusional system and then shot him.

Few clinicians thought assaults had occurred without their own involvement or on a random basis. Those who reacted defensively and blamed the patient or the setting for the assault usually worked in prison or forensic settings, where they were exposed to a large number of antisocial assaultive patients over whom they had little control and with whom they had little therapeutic relationship. One clinician who worked in a prison reported that he carried a gun to protect himself from threats of assault, even though he had only been assaulted twice. Another clinician who was exposed to an equally dangerous group of patients in a criminal court system had never been assaulted and had in the entire course of his ten-year career been threatened on only one occasion. A psychiatrist in private practice was assaulted several times, and a clinician in a state hospital for the criminally insane had sustained several assaults and looked upon them in a more or less philosophic fashion as part of the line of duty.

In our work with violent patients we have focused on countertransference reactions to such patients and

the anxieties, denial, and projection that clinicians experience when working with such individuals. It is difficult to ascertain the degree to which transference phenomena occurred among patients, but the vehement responses of several clinicians to our study indicated that they were very angry with several patients who could be classified as violent and did not wish to deal with them. Some clinicians are obviously more sensitive and skilled in the management of aggressive individuals, but there are also superimposed social and cultural factors that play a role in therapists' dislike and avoidance of violent patients.

It is our belief that the subject of violence among psychiatric patients tends to be minimized for a variety of reasons, both conceptual and psychodynamic. First, psychiatrists are apt to be more in tune with the internalization and introjection of aggressive urges and thus they may reward patients who show depressive symptoms, which they consider more treatable than aggressive ones. This leads to a shunning by clinicians of violent patients and, we believe, a distinct lack of training and expertise in the management of assaultive individuals. Not all assaultive patients are suitable candidates for treatment, and the management of assaultive patients is best carried out in crisis situations by people skilled in physical restraint techniques (3).

We believe that more attention should be paid to assessing on a clinical basis a violent patient's propensity toward acting out in a transference situation. We also feel that clinicians must become more sensitive to their role in dealing with potentially violent patients so that they can institute effective intervention policies.

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Assault on the Therapist

BY ROY M. WHITMAN, M.D., BEATRICE B. ARMAO, M.S.W., AND ORAN B. DENT, PH.D.

The authors conducted a questionnaire survey among psychotherapists in psychiatry, psychology, and social work to determine how many patients posed a threat to others or to the therapist and how many actually assaulted the therapist. They found that 9.2% of all patients seen by 101 therapists in one year presented a threat to others, 1.9% posed a physical threat to the therapist, and .63% actually assaulted the therapist. The authors conclude that attacks on a therapist are infrequent but almost inevitable and suggest the development of techniques for coping with assaultive patients that therapists can use in crisis situations.

WE FIRST BECAME INTERESTED in the subject of assaultive patients when one of us (B.B.A.) reported during a supervisory consulting session that she had been threatened by a potentially violent patient. The literature offered little information concerning therapists' fears of assaultive patients; neither did our associates. It appeared that the subject was often ignored during training and that it did not arise very often in consultation.

Our discussion of the situation raised several questions: How many other therapists are similarly threatened? How do they handle both the patient's and their own feelings? Do level of training and experience make a difference in the clinical approach to these problems?

We were surprised at the variations in our colleagues' responses to questions about assault. A number of them said that they had never been frightened or assaulted by a patient, but more recalled incidents that had happened recently or some years past. The willingness of some therapists to relate in detail dramatic incidents in which they had been involved was impressive.

Therapists then came to us with more experiences not recalled at first inquiry. Recall and subsequent discussion had a cathartic value for them. Our interest was further augmented by the fact that incoming trainees were encountering threatening patients. The pooled clinical experience of therapists and trainees suggested that impulsive, violent patients were being seen more frequently in mental health facilities than ever before. It seemed to us that the Viet Nam experience, for example, had left many of our veteran patients with residual rage that was poorly controlled.

The literature on assaultive patients that we surveyed (1, 2) tended to emphasize countertransference on the part of therapists and to deemphasize realistic fear reactions. Lion and Pasternak (1) commented that therapists have "some anxiety about treating a violent patient," but the main emphasis of their paper was that the physician distorts and exaggerates the patient's dangerousness by means of projection.

Macdonald (2) studied how seriously the therapist or other evaluator should take homicidal threats toward others; we are asking how seriously a therapist should take threats toward himself. Should he be afraid or not? What does the therapist feel when he is threatened with assault? How does he cope with the patient and his own feelings?

METHOD

We sent a questionnaire[1] to 184 mental health workers in the three professions of psychiatry, psychology, and social work in the Cincinnati, Ohio, area. The respondents could remain anonymous if they wished. The questionnaire requested information on the discipline of the therapist, number of years in practice, and number of patients seen during the calendar year 1972. Questions were then directed toward the number of patients seen during the year who the therapist felt posed physical threats to others, the number who the therapist felt posed threats to himself, and the number who actually assaulted him. We then requested a narrative description of the most frightening experience the therapist had had with an assaultive patient in 1972. If he had had none that year, we asked for an account of an experience from any time during his professional career. We asked for the strategies that the therapist used to deal with the situation described, the therapist's reactions, and the final outcome of the event.

Revised version of a paper read at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Whitman is Professor of Psychiatry, Ms. Armao is Instructor in Psychiatric Social Work, and Dr. Dent is Adjunct Associate Professor of Psychology, University of Cincinnati College of Medicine and the Veterans Administration Mental Hygiene Clinic, Cincinnati, Ohio. Address reprint requests to Dr. Whitman at the University of Cincinnati College of Medicine, 231 Bethesda Ave., Cincinnati, Ohio 45267.

[1]Copies of the questionnaire are available on request from the authors.

One month after our initial mailing, we circulated the same questionnaire again to the same group, urging them to fill out the form if they had not already done so.

RESULTS

Seventy-eight of the 184 therapists (42%) responded to the first mailing. The second mailing increased the total number of responses to 101 (55%). Of the 96 psychiatrists contacted, 53 (55%) responded; 27 of 43 psychologists (63%) responded; and 21 of 44 social workers (48%) responded.[2] Thus 52% of the sample of 101 therapists were psychiatrists (the ratio of residents: staff was 17:36), 27% were psychologists, and 21% were social workers. These percentages approximate the relative numbers of active psychotherapists in the Cincinnati area for the three disciplines.

Twelve of the respondents were from the University of Cincinnati Counseling Center, 14 from the Northern Kentucky Comprehensive Care Center, and 75 from various settings (including 17 in private practice) affiliated with the University of Cincinnati Department of Psychiatry. Of the 101 respondents, 80 worked primarily in outpatient settings and 21 in inpatient settings.

The psychiatrists were more experienced as a group and saw more patients than either psychologists or social workers. The range of experience for psychiatrists was 1–40 years, with a median of 5 years' experience; psychologists reported 1–22 years' experience, with a median of 4; and social workers had 1–20 years' experience, also with a median of 4. All three disciplines reported a full range for number of patients seen. Psychiatrists, however, reported a median of 81–100 patients seen, while both psychologists and social workers reported a median of 41–60 patients. Our findings reflect a population in which the psychiatrists were older and saw more patients than psychologists or social workers. A surprising number of therapists reported that they had seen more than 100 patients during the year (24 psychiatrists, 3 psychologists, and 4 social workers). In estimating the total numbers of patients seen, the 1–20 category was counted as 10 patients, 21–40 as 30, and so forth.

As shown in figure 1, of the 3,810 patients seen by the 53 psychiatrists during 1972, 416 (10.9%) were perceived as presenting some assaultive threat to other people in their environment, 91 (2.4%) posed some physical threat to the therapist, and 33 (.87%) actually assaulted the therapist. The 27 psychologists reported that they had seen 1,740 patients and that 85 (4.9%) presented some threat to others, 18 (1.0%) posed some threat to the therapist, and 2 (.11%) assaulted the therapist. Twenty-one of the social workers reported that they had seen 1,170 patients and that 114 (9.7%) presented some threat to others. Twenty of the social workers reported that 17 of 1,000 patients (1.6%) posed some threat to the therapist and that 7 of 1,070 patients (.65%) actually assaulted the therapist.

Of the total of 6,720 patients, 615 (9.2%) presented some assaultive threat to others, 126 (1.9%) posed a physical threat to the therapist, and 42 (.63%) actually engaged in assaultive behavior toward the therapist. A major finding of this study thus emerged as a ratio—patients who presented threats to others: those presenting threats to the therapist: those who assaulted (15:3:1).

As shown in figure 2, 79% of the total group of 101 therapists (77% of 53 psychiatrists, 81% of 27 psychol-

FIGURE 1
Percents of Patients Who Posed Threat to Others or to Therapist or Who Assaulted Therapist

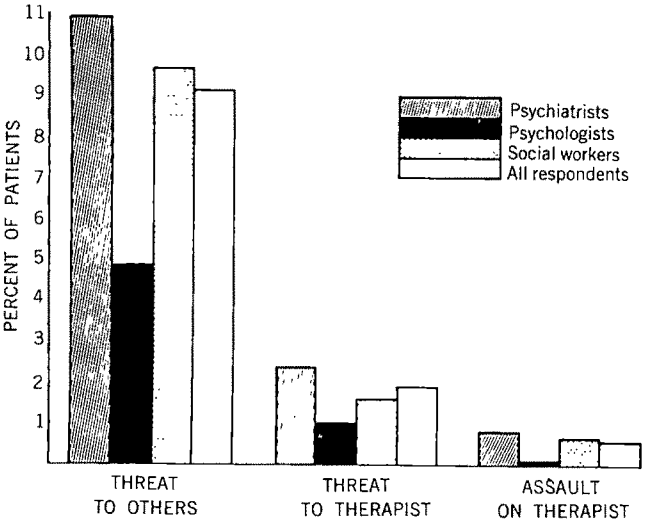
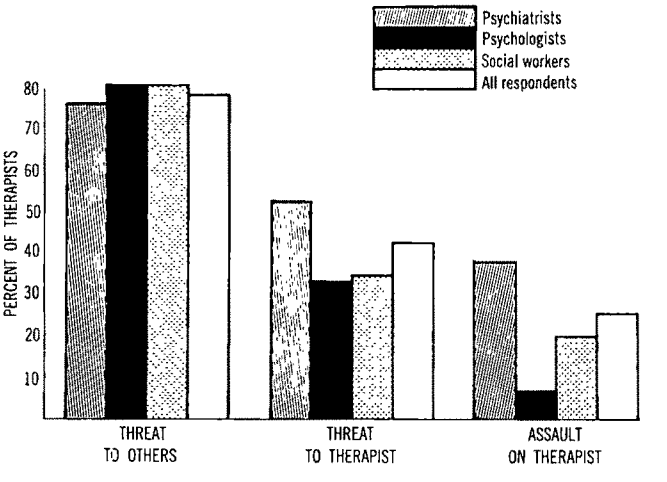


FIGURE 2
Percents of Therapists Who Reported Having Seen One or More Threatening Patients in 1972



[2]One of the responses from a social worker was incomplete: this respondent reported data on 100 patients and answered only the questions on how many of these patients posed some threat to others and whether he had ever been assaulted by a patient in the past.

ogists, and 81% of 21 social workers) reported at least one incident in which a patient was perceived as presenting some threat to others. Forty-three percent of 100 therapists (53% of 53 psychiatrists, 33% of 27 psychologists, and 35% of 20 social workers) reported at least one incident in which the therapist felt personally threatened. Twenty-four percent of 100 therapists (34% of 53 psychiatrists, 7% of 27 psychologists, and 20% of 20 social workers) reported that they were actually assaulted during the year by one or more patients.[3] No psychologist reported more than one assault for the year, but 8 (15%) of 52 psychiatrists and 1 (5%) of 20 social workers reported two or more. Although the percent of patients who actually attacked therapists is low (less than 1%), the likelihood that a therapist who sees a large number of patients will be assaulted is strong.

A series of chi-square tests were performed to investigate relationships within the data.[4] No differences were found between staff and students for personal threats and for actual assaults. Staff psychologists and psychiatrists reported a significantly smaller percent of patients who presented threats to others than student psychologists and psychiatrists ($p<.05$), but a comparison of staff and student social workers showed no difference.

Divided at a median of 9 years, the effect of number of years of experience of staff psychotherapists (those with more than 3 years' experience) was investigated for each of the three threat categories; no differences were found.

Differences for the setting in which the therapists worked were also investigated. The only finding was a disproportionately low number of personal threats felt by the therapists at the University of Cincinnati Counseling Center, who were working with a population of college students.

No significant differences were found among psychiatrists, psychologists, and social workers for the percent of cases reported in the three threat categories. Likewise, no significant differences were found in a comparison of male and female therapists.

As might be expected, the more patients seen by a therapist, the more threats were reported in each of the three categories. Furthermore, the more patients a therapist reported as posing threats to others, the more patients he felt posed a threat to himself; and the more patients he felt posed a threat to himself, the more patients actually assaulted him.

Seven major categories of coping techniques emerged from the therapists' descriptions of assaultive incidents. The research team added an eighth technique. These techniques are: 1) interruption of the interaction, 2) physical restraint, 3) nonverbal minimization of self as threat, 4) verbal responses, 5) protection of self, 6) encouraging motoric discharge of anger, 7) assistance from supervisors and/or colleagues, and 8) countertransference maneuvers. These coping techniques, as well as the reactions of therapists to assault and the final outcome of the event, will be discussed in more detail in subsequent papers.

DISCUSSION

The overall data indicate that in one calendar year 1 of every 11 patients (615 of 6,720) seen in a variety of psychotherapy settings presented an assaultive threat to others in their environment, 126 were physically threatening to the psychotherapist, and 42 actually engaged in assaultive behavior toward the therapist. The 42 patients who assaulted the therapist were distributed among a variety of mental health professionals to the extent that 24% of 101 therapists were attacked during one year by at least 1 patient. Thus attack on the therapist is an infrequent but also inevitable phenomenon that may occur at some time to every therapist. Since one onslaught can end a therapist's career, every therapist should have a working knowledge of a wide variety of techniques to manage assaultive patients that he can put into effect with little delay or deliberation. Although only 24% of the 100 therapists who responded to the question on attack in 1972 were attacked during that year, 74% of the 101 therapists who responded to the question on attack at any time described at least one incident in their professional careers that involved an assaultive patient.

The research team pooled the knowledge that the responding therapists had gained over the years in dealing with violent patients. The eight coping techniques listed above represent this collective experience.

Another way of organizing the eight coping categories is the following: 1) biological—physical and chemical methods of control by others or the self, 2) psychological—verbal methods of control, such as firmness, honesty, and humor, and 3) social—use of family influence, pressure from social institutions, and peer influence.

We have observed that therapists tend to use only one type of technique in coping with assaultive patients. Verbally oriented therapists use verbal techniques, even when physical techniques are clearly indicated, and therapists familiar with physical techniques inappropriately put these into effect when an empathic response would serve better.

Our data indicate that responses to the threat of patient attack are stereotyped and instantaneous; only occasionally are they highly creative. It therefore seems useful for therapists to engage in extensive discussion, rehearsal, and other forms of training so that useful techniques can be employed without delay or

[3]Even if we assume that the 83 nonrespondents and the 1 respondent whose questionnaire was incomplete were not assaulted during 1972, the percent of therapists who reported that they were actually attacked in that year is 12% of the total number of therapists contacted (N=184).

[4]A detailed report of the statistical analysis is available on request from the authors.

deliberation in a crisis situation. If one is aware of other therapists' innovations as possible alternatives, and if the innovations have been practiced, one may have useful additions to a repertoire of therapeutic responses to the assaultive patient. Role-playing techniques, in which the therapist takes the role of an assaultive patient, are one useful way to gain insights into the feelings and impulses of such patients and into the patients' responses to various therapeutic maneuvers.

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Nursing Aides and Patient Violence

BY PAULINE LEVY, R.N., AND PETER HARTOCOLLIS, M.D., PH.D.

The authors studied the incidence of violence in a psychiatric unit organized as a therapeutic community whose nursing staff was composed entirely of women in comparison with another unit staffed according to the traditional male and female nursing pattern. The unit staffed by women had no incidents of other-directed violence in the one-year period of the study, but the traditionally staffed unit had 13 violent incidents initiated by 5 patients. The authors state that the expectation of violence among psychiatric patients and the consequent use of male nursing aides to control violence may represent a self-fulfilling prophecy.

CONCERN THAT PSYCHIATRIC PATIENTS may suddenly become assaultive and generally destructive is always present among hospital staff members. This concern and the need to handle violent patients has traditionally and more or less explicitly been the reason for using male nursing aides on the staffs of psychiatric hospitals. Our research suggests that the use of male aides is more likely to lead to violence than prevent it.

BACKGROUND

Two years ago we were asked to reorganize a predischarge transitional ward of a private psychiatric hospital into an autonomous treatment unit. The unit had been operating for several years with a reduced nursing staff, all of whom were women—an unusual arrangement in psychiatric hospitals. We were impressed with the fact that the unit had a very low incidence of violence.

The fact that all of the nursing staff were women was thought to be an outgrowth of the virtual absence of behavior problems on the unit, but we chose to interpret it as the cause rather than the result of the unit's history of nonviolence and to make it part of the new unit's program. Specifically, we hypothesized that violence-prone patients may find female nurses and aides less provocative than male staff and that, conversely, when confronted with threatening patients in the absence of male aides, female staff may be more apt to rely on nonaggressive manners and feminine intuition rather than resort to reactive, policelike methods, which are typical of masculine behavior and values in our culture.

THE PROJECT

The new unit, which had 20 beds and an open-door policy, was located in a private psychiatric hospital that included two other long-term inpatient units of approximately equal size. Our unit was staffed with the following personnel: 3 part-time psychiatrists, 1 resident psychiatrist, 1 full-time and 2 part-time social workers, 1 psychologist, 3 activities therapists, and 15 nurses and nursing aides; all of the nurses and aides were women. The patient population included slightly more women than men, was predominantly young (ranging from 17 to 55 years of age), and represented all diagnostic categories (the predominant diagnosis,

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Ms. Levy is Senior Staff Nurse and Dr. Hartocollis is Director, C.F. Menninger Memorial Hospital, Topeka, Kans. 66601.

however, was borderline personality organization). The average length of patient stay during the one-year study period was six months.

The unit's treatment program was based on the principles of a therapeutic community. Sharing of problems and confrontation by fellow patients and staff members took place in group meetings of patients with staff every day of the week. The meetings could be formal or informal and small or large. Patients elected their own government representatives, who had the right to negotiate issues affecting the patient community in regular meetings with authorities. Responsibility for treatment and the welfare of the community was to be shared by patients as well as staff. This expectation, along with the explicit renunciation of violence, constituted the cardinal value of the unit's philosophy.

The program was designed for direct but voluntary admissions. Preselection was expected to be minimal; the only patients excluded from the unit were those who were currently assaultive. Variables known to contribute to episodes of violence—crowded quarters, shortage of staff, social disorganization, and problems in communication—were carefully monitored and controlled to the best of our ability.

We defined "violence" as behavior that involved aggressive action between 2 or more persons on the unit and produced at least minor physical injury (including scratches and bruises) or destruction of property. Data about violence were obtained from written daily nursing reports. Data from a unit staffed according to the traditional female and male nursing pattern but similar in all other respects were used for control purposes.

FINDINGS

During the one-year period of the study, the incidence of violence in our unit was zero. There were several crises and episodes of disorganization that involved threatening behavior and abusive language, but there were no acts of physical violence on this unit. One young male patient verbally insulted the staff several times but repeatedly assured them and his fellow patients that he would not strike a woman. An older male patient made aggressive gestures toward staff members during a hypomanic episode. He shook his fists, pointed his finger at the staff, and once touched a nurse's nose angrily, but he never hit anyone.

Staff members were impressed with the high degree of self-control patients were able to exercise. Noteworthy, too, is the fact that male patients helped the nursing staff contain the aggression of their fellow patients, providing a source of reassurance for both staff and disturbed patients during stressful times. For example, when a young male patient threatened to strike anyone who tried to prevent him from leaving the unit, other male patients chose to remain in the area until the crisis was resolved. Thus the nursing staff were able to maintain their self-awareness, contain their own fear and anger, and deal with the threatening patient firmly but kindly, managing to prevent him from regressing further into violent action.

In spite of the facts that there was no violence among the patients and no external threat developed, the nurses on the night shift felt vaguely anxious after hours, when no male staff were around and most of the patients were in bed. For example, when a female patient had a male visitor who persisted in prolonging his visit on the unit after midnight, the nursing staff called the night supervisor, who came accompanied by a male aide. Male patients later expressed the wish to be awakened when this sort of incident occurred. On several occasions when this was done, the patients proved very effective in helping nurses deal with problems during late hours, even though their methods did not always satisfy the nurses.

Although there were no episodes of violence against others, self-directed violence did occur a number of times. However, there were no fatal injuries or injuries serious enough to require treatment outside of the unit during the study period. Suicidal threats were fairly frequent.

In contrast, the nursing notes for the control unit showed 13 instances of assaultive behavior initiated by 5 different patients.

No systematic count of episodes of violence was carried out in the other long-term treatment units of the hospital, which continued to use the traditional female and male nursing staff pattern, but a sampling of the records of these units and anecdotal data indicate that over the one-year study period they experienced a proportionally greater amount of violence. Whether the incidence of self-directed violence or threats of such violence was different in other sections was not investigated.

DISCUSSION

Even though the incidence of violence may not be as great in psychiatric hospitals as it is in other total-care institutions, the idea that patients in a psychiatric hospital may suddenly become assaultive is prevalent, not only among laymen but also among psychiatric personnel—especially nurses and nursing aides (1). The expectation of violence and generally destructive behavior may constitute a self-fulfilling prophecy that needs to be recognized and treated as a transference or countertransference problem. When nursing staff fear that patients under their care may become violent, the probability is that they are concerned about their own propensity for violence and the fear that they may not be able to contain it if sufficiently provoked. Feelings of inadequacy concerning the staff's ability to handle a difficult situation, when accompanied by frustration and anger, may scare a disturbed patient to the point of driving him to violence (2).

Spiegel (3) pointed out that confrontation is an effective means of resolving group conflict and promoting

social change but that it carries with it "the strong possibility of physical violence." Confrontation is the technique most widely used to resolve conflict and to effect change in a therapeutic community. However, for confrontation to become dangerous, there must be an intent and a reciprocal expectation of violence, based on the belief that for confrontation to be effective there should be violence or the option to do violence. When a male aide is used as an instrument of confrontation, the probability of violence becomes greatly enhanced because the male aide's formal or informal role as masculine authority is based on the intent to do violence. This intent is potentiated by female nursing personnel who expect the male aide to confront troublesome patients for them.

Storr (4) found that male aggression tends to occur spontaneously in a wide array of conditions, such as rivalry, territoriality, and display, but that aggression in females is fully aroused only or mainly in response to a threat that involves their young. Bigelow (5) noted that one of the causes of severe aggression in animals is "the crowding of strangers, especially near such valued resources as food or mates."

Crowded conditions in a hospital, which are known to cause violent episodes and regression of all sorts, mean not only deprivation of space and privacy but also shortage of personnel in the sense that relatively few patients can get the attention they need from very busy staff members (6). Patients who cannot or are not allowed to use their own potential for mutual assistance and sharing of responsibility tend to compete for the attention of nurses and aides, whom they perceive as "valued resources" to obtain aggressively or, if denied, to destroy in frustration.

In our experience, the possibility of violence threatened the nursing staff most when the structure of the patient-staff community was weak. When the ability of the community members to support each other or, more generally, when its ability to adhere by the common rules and values (beginning with the renunciation of violence) were not trusted, fear and unrest prevailed. External controls were then desired, and, as a crisis developed, they were sought in the form of phone calls to an authority outside of the unit and in the importation of male aides. The night personnel were more apt to mistrust the community's controls and often failed to make full use of the community because they had the least exposure to its working sessions. All of the regular community meetings took place during the day. Nurses and some patients longed for the presence of male aides, especially after dark.

In nonhuman primate social groups as well as in primitive human societies, order is maintained through the power of males (5). Likewise, in mental hospitals male aides have been traditionally employed to contain aggressive patients or to enforce unpopular rules by a show of sheer muscular force. In the case of night personnel, there is also the real or imaginary fear of intruders. Perhaps a metaphor, or dynamic, can be applied to nurses—as wives who wish for their husbands to come home at night. The patients' wish for a male aide in the evening may express a parallel dynamic—the wish for a father to come back home when a day of work is over. The wish for the presence of male nursing staff, especially at night when other male staff are not around, may also indicate concern about the sexual feelings of the female nursing staff or of the patients toward these women and the need to safeguard the nurses' professional roles as substitute mothers.

CONCLUSIONS

We believe that the exclusive use of female aides may keep the incidence of other-directed violence in a psychiatric hospital to a minimum. We also feel that the absence of male aides should not necessarily hamper the treatment process. The wish of some female nurses and aides to have the benefit of the presence of male aides seems to us less related to good treatment and more related to professional, cultural, or dynamic factors that could interfere with treatment.

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A Violence Clinic: Three Years' Experience

BY JOHN R. LION, M.D., DENIS J. MADDEN, PH.D., AND RUSSELL L. CHRISTOPHER, M.D.

The authors describe a clinical facility established for the neuropsychiatric treatment of individuals with a variety of psychopathological disorders where the common management problem is one of aggressive behavior. The authors present case vignettes to illustrate the clinic's consultation with mental health workers about the management of violent patients, countertransference problems, ward problems, the role of organic factors, medication, and prediction of violence. They urge that the subject of violence be included in the psychiatrist's training.

IN THIS PAPER we will discuss the consultation, teaching, and research functions of a specialized clinical service within the psychiatric unit of a general hospital. The clinic, begun as an extension of our interest in clinical problems of aggression (1–4), sees a range of patients with psychopathology including severe characterological disturbances, adjustment reactions, toxic and functional psychoses, and organic brain syndromes. In all of these disorders, aggressive behavior is the common management problem. Our experience to date with more than 400 patients roughly parallels statistical data and profiles previously described (5, 6).

A combined psychological and neurological approach to the problem of aggression has involved a study of psychodynamic factors as well as brain dysfunction, since we believe that aggression is a phenomenon closely linked to impulsivity (7) and, as such, is a behavior in which organic factors may contribute to the poor control of aggressive urges and the paroxysmal expression of hostile outbursts. Our patients are treated with psychotherapy and the adjunctive use of medications.

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At the time this work was done the authors were with the Department of Psychiatry, Institute of Psychiatry and Human Behavior, University of Maryland School of Medicine, Baltimore, Md. 21201, where Dr. Lion is Associate Professor and Director, Clinical Research Program for Violent Behavior; Dr. Madden is Clinical Assistant Professor and Associate Director of the Program; and Dr. Christopher was Chief Resident. Dr. Christopher is currently Staff Psychiatrist, Neuropsychiatric Medical Group, Huntington Beach, Calif.

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As the clinic has become a recognized entity, we have found ourselves involved in the following tasks.

CONSULTATION

Social and medical agencies frequently request consultation regarding the management of a violent patient. In such instances personnel have grappled with the problem of treatment or have questions regarding the diagnosis and the proper use of medications. In other instances staff difficulties or therapist countertransference reactions become apparent.

Vignette 1. A borderline psychotic woman was seen at a local hospital because of repeated assaultive and self-mutilative behavior. She had attacked members of the staff and had swallowed objects, necessitating five laparotomies. She was isolated and identified as a notorious patient among the several thousand patients of the hospital. In discussion, two elements of therapeutic importance emerged. The staff's anxiety in dealing with the patient had magnified her dangerousness to the point where avoidance was seen as preferable to confrontation, which might evoke some aggression. The avoidance minimized therapeutic interaction. In addition, the patient received only moderate amounts of medication and a pharmacological repertoire had not been tried in any systematic fashion. Discussion of both issues with staff let to improved patient management with a significantly reduced number of aggressive outbursts.

This case illustrates several issues. First, staff reactions to a patient can be a strong force in alienating the patient and eventually worsening his psychosis or behavior by virtue of the induced sensory isolation. Anger that is displaced by staff onto the patient can intensify such punitive seclusion as personnel find it difficult to ventilate their helplessness about a difficult patient (4). A second issue was the inadequate use of medication. The aim of treating a violent patient with medication is not to render him helpless and inert but to allow him to effectively modulate his own aggressiveness. To this extent, however, medication must be used in a forthright and systematic manner. We have been active in formulating guidelines for the use of specific drugs in the control of violent patients (8).

The next case is a more social one, involving not a particular patient but an entire hospital.

Vignette 2. Distant consultation was requested in a northeastern state hospital due to what was labeled an "out-

break of violence" among the patients. Upon their arrival, clinic personnel discovered that several members of the staff had been indicted for physical abuse of patients. Inspection of the situation revealed a paucity of psychiatrists and the recent arrival of a patients' legal rights advocate. Thus there was not enough staff to properly manage difficult patients, and because of the excessive concern about the rights of patients, any necessary physical restraint of an aggressive patient was perceived as a possible legal transgression.

This case illustrates the complexity of the problem of violence "epidemics," a phenomenon with which we have now gained some experience. Generally, the issue is one of morale that is passed down to patients, or it may be one of staff conflicts similarly handed down. Epidemics reflect staff splitting and dissension, and such phenomena are seen on a smaller scale on ward settings as well. Consultation in such matters is a delicate operation since the issue of staff mismanagement may well arise and feelings of defensiveness are thus heightened. At one recent consultation to a school for delinquent adolescents, the staff stated that "a problem existed with regard to the acting out of the patients." Such euphemistic phrases obscure the true level of violence and yet at the same time reveal the staff's feelings about violence and their own inability to express violent fantasies and urges.

From time to time we have been asked to see rather esoteric cases of violence. Some of these cases are forensic, and others involve social and medical policy issues.

Vignette 3. A mentally retarded man was hospitalized with a request for psychosurgery because of intractable violent behavior and self-mutilation. The patient was kept physically restrained or secluded almost continuously. He had enucleated one eye and disjointed a nasal septum. The possibility of stereotactic amygdalectomy was discussed, but it was apparent that a full medical regimen had not been tried and no justification for any surgical procedure existed since the patient was not epileptic. However, it was recognized that he was probably doomed to remain incarcerated unless an effective medical regimen was used.

Recent social policy regarding psychosurgery for aggressive patients has justifiably raised doubts about the ethics and efficacy of such procedures, and the entire matter has been reviewed by government agencies (9, 10). While it appears that there may be a moratorium on psychosurgical research, it must also be recognized that there does exist a rare class of patients so hampered by violence as to make their existence quite intolerable for themselves and others unless drastic action is taken. In the case just described, we were not convinced that a full medical armamentarium had been tried and made academic suggestions to the referring hospital. Their reply, understandably, was that it was simple for us to suggest higher dosages of chlorpromazine since we did not have to care for the patient. Other patients we have seen in prison settings have presented similar dilemmas. The following case illustrates a social policy issue of a different nature.

Vignette 4. A 25-year-old surgical resident was referred because he had been assaultive to a patient on the geriatric ward. The physician had experienced much helplessness as a child and reacted inappropriately to an infirm and elderly patient who tended to harass him and make unreasonable medical demands. The dynamics of the case were clear to the interviewer, who felt that psychotherapy was imperative. The resident refused such treatment, and his superiors then insisted that he undertake such therapy or leave the program.

This difficult consultation raised the issue of confidentiality and coercive treatment. The phenomenon of medical staff reacting inappropriately toward older, helpless patients or toward children is not uncommon and can be seen in child abusers as well. We have seen several physicians and policemen with tendencies toward temper outbursts of sufficient severity to bring them to the attention of superiors. The common dynamics in these cases were helplessness and strong reaction formations against passivity and weakness. The appearance of disorders of aggression in professionals is in contrast to the usual finding that aggressive patients come from lower socioeconomic strata. We believe that such a skew reflects in part society's handling of aggressive individuals, whereby those middle- and upper-class patients who become violent are treated differently within the legal and medical systems.

TEACHING

Within the medical school, the clinic serves to instruct students and residents in the evaluation and management of violent patients. For some time it has been our feeling that patients with propensities toward violence are shunned by a profession that is more in harmony with the introjection of aggressive urges (6). This is particularly true of patients who suffer from personality disorders and who may have criminal histories. Such individuals are perceived as material for courts and prisons, rather than psychiatry. Clinical decisions are of course necessary to distinguish the treatable from the nontreatable and purely manipulative individuals who wish psychiatric help to avoid court action, for example.

Yet in the history of our discipline, knowledge of aggression has often been derived indirectly through the study of normal aggressive drives in nonaggressive patients, such as depressed individuals or those with psychosomatic diseases (7). We submit that to learn about aggression and the dynamics of that drive, one must study patients whose primary problems are with aggression. Too often, the aggressive patient is still handled dispositionally and shunted to end-stage facilities such as state hospitals.

Vignette 5. A patient was hospitalized because of temper problems at work and home. He presented a hostile facade with a belligerent, provocative stance. The staff came to fear

him and expected him to live up to their expectations, that is, to explode with anger in group therapy or on the ward. In time, with supervision, the staff was able to see that the patient was actually an individual with a fragile sense of self-esteem, which he could acknowledge. Further, his outbursts were not random or instantly paroxysmal but came as the result of a perceived insult, and the patient could be taught to recognize premonitory signs and symptoms indicative of an incipient outburst. Thus he was able to verbalize his anger rather than handle it behaviorally.

Examples such as this illustrate the dynamics in patients with severe characterological disorders, who are prone to handle affective issues behaviorally and create disturbances within the milieu (11).

Management problems exist in individual therapy also.

Vignette 6. A psychiatrist phoned to seek advice regarding the handling of a paranoid schizophrenic patient. The patient had made homicidal threats on the telephone, and the therapist had dealt with these threats by not responding and hanging up. His anxiety mounted as the threats continued. In discussion, it became evident that the patient had made similar joking threats in therapy as intimacy progressed, and the therapist had casually dismissed such threats and gestures. The therapist was encouraged to confront the patient and discuss what appeared to be unresolved transference and countertransference issues.

This is a frightening example that dealt in large measure with a problem of denial, a defense we have often noted in the therapy of such patients. Homicidal threats often bespeak underlying nurturant needs that are frustrated on some level, and such threats demand confrontation and clarification. Residents often require guidance in the therapy of patients whom they come to perceive as dangerous; in such instances reality issues must again be separated from countertransference problems (12).

Of particular interest is the need to teach students to accurately assess violent propensities. This is done by inquiry into the patient's past use of violence, criminal history, driving history, ownership of weapons, and by other questions that can evoke anxiety. Interviews with and treatment of provocative victims are mandatory in the therapy of violent patients once a pathological relationship has been established (6). Awareness of organic factors is necessary in the management of certain violence-prone individuals who manifest some degree of brain dysfunction. The dysfunction can range from rare psychomotor epilepsy to more subtle states of brain dysfunction detectable by careful cortical function examination, including psychological, neurological, and EEG testing (11).

RESEARCH

We have been active in the psychopharmacological control of aggression (13). To summarize briefly, there is no one drug for the treatment of aggression since aggression is a manifestation of a variety of psychopathological conditions. However, once an etiology is established, one class of drugs may be more useful than another. For example, anticonvulsants or benzodiazepines with anticonvulsant properties may aid in the treatment of explosive personalities of patients prone to ictal forms of aggression; lithium may be useful in treating aggressive patients with underlying affective disorders when the manifestation of the affective disorder is psychomotor excitation rather than true mood swings.

In addition to pharmacological work, we have been interested in psychotherapeutic techniques that involve an elaboration of fantasy in individuals who appear to have a poor ability to anticipate the consequences of their acts. We are also interested in teaching patients, particularly those with personality disorders of paranoid, explosive, passive-aggressive, or antisocial types, to recognize their affective states and describe them verbally, thus making their feelings amenable to communication (14). This is done in individual and group therapy (15).

COMMENT

Our clinic is no more than a group of individuals interested in problems of aggression. We do not advocate the establishment of violence clinics but do urge that the training of psychiatrists include education in the topic of violence. If we are to understand aggression to the same degree as we hope to conceptually understand and therapeutically deal with depression, we must work with and learn from patients who are aggressive.

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Journal Policy on Multiple Authorship

It has come to our attention that there has been some misunderstanding regarding the *Journal*'s policy on polyauthorship. The number of authors listed on a paper is not a factor in the review and acceptance/rejection procedure. However, once a decision has been made to publish a paper, we do apply *Journal* policy regarding the number of authors in the by-line: only principal researchers and writers of articles are listed in the by-line; collaborators who have provided technical and other assistance are acknowledged in a footnote. In papers that list more than 4 people as principal authors, we query the first author to verify that those individuals were substantial contributors to the research or ideas reported. Since certain types of psychiatric research may require multiple workers, we will of course include all of the names in these cases.

EDITORIAL

Rape: "The Ultimate Violation of the Self"

THE PUBLICATION IN this issue of the *Journal* of four articles with an empathic focus on rape victims marks a sharp departure from much of the previous medical literature on the subject. Most striking has been the absence, until recently, of any significant literature about the victim other than that limited to medicolegal concerns, although a variety of articles have focused on the need to understand and rehabilitate the rapist. The reader was provided with a mass of instruction designed for the medicolegal protection of the examining physician rather than the victim. Further, the assumption permeating many of the articles was that the victim may have provoked the crime: had she been drinking, was she seductive, sexually active, did she know the assailant? Similar attitudes prevail in the criminal justice system, with its stringent corroboration requirements in rape cases; such corroboration has been supported because "We know from the lessons of the past that all too frequently such complainants have an urge to fantasize or even a motive to fabricate . . ." (1).

The theme of woman as liar pervades our constructs about rape victims and underlies the assumption that women often make false charges of rape against men, even men they do not know. While law enforcement personnel are aware that false charges of crime occur, it is only in rape cases that it is assumed that the usual safeguards in the system cannot protect the innocent from a lying witness (1, 2). In robbery, for example, it is understood that property was taken from the victim without his/her consent, and there is no need to prove that fear of death or grave bodily harm was at issue. The law, then, grants more protection to property than to the person, especially if the person is female.

Both medical and criminal justice institutions reflect the same mythology that society perpetuates about rape. The abundant mythology suggests that prior sexual experience on the part of an unmarried victim is reasonable evidence of "provocation"; women ask for rape by seductive behavior and dress; "nice girls don't get raped and bad girls shouldn't complain" (1); and women can't be raped unless they want to to be, corollary being that women actually enjoy rape. The victim is thus perceived as a responsible and not an innocent party to the crime.

The act of reporting a rape initiates a complex process. The victim is confronted with the usual institutional patterns of the hospital and criminal justice system, which at best are experienced as confusing and alien. She presents herself to these authorities at a time of crisis that differs from other crises in that her usual support system is more likely to be disrupted. Families, friends, and lovers, because of their own perceptions about rape, may desert and further isolate her. Moreover, the crisis is never limited to one's person since the victim, by the act of reporting, becomes public property and is at the mercy of the hospital, police, courts, media, and community opinion.

Rape is "the ultimate violation of the self" (3) short of homicide and is best understood in the context of a crime against the person and not against the hymen. Rape is an act of violence and humiliation in which the victim experiences overwhelming fear for her very existence as well as a profound sense of powerlessness and helplessness which few other events in one's life can parallel. The victim

needs empathy and safety and a sense of control over what has happened to her and what will happen in her encounters with hospital and law enforcement. In the absence of sensitivity to these needs, the experience of reporting becomes another assault.

It is not surprising that the four papers on rape in this issue were written by women. Recent attitudinal shifts have been largely due to the initiative taken by the countless numbers of women who have begun to sensitize our medical, social, and legal institutions about the extent to which cultural biases have determined the maltreatment of the victim. Deena Metzger's paper (4) stands as a deeply moving account of the meaning of the personal violation which defines the rape experience. Her concept of rape as a "ritual of power" is supported by a growing body of information (5, 6) about the social context in which rape occurs; the same concept is presented with eloquence by Susan Brownmiller (6), who suggests that rape has the same meaning for women as did lynching for blacks: "That *some* men rape provides a sufficient threat to keep all women in a constant state of intimidation, forever conscious of the knowledge that the biological tool must be held in awe for it may turn to weapon with sudden swiftness born of harmful intent."

Innovative and empathic services to victims will serve as a deterrent to the crime by facilitating reporting of the crime and thereby the apprehension and prosecution of the assailants. Ultimately, however, the elimination of rape demands a massive restructuring of social values to include a reconsideration of the relations between the sexes. Only when the sex roles of both men and women are defined by individual needs and talents rather than by stereotypic expectations based on sex and power motives will there be an end to rape.

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ELAINE HILBERMAN, M.D.

Dr. Hilberman is Assistant Professor of Psychiatry, School of Medicine, University of North Carolina at Chapel Hill.

BRIEF COMMUNICATIONS

Reduced Platelet Monoamine Oxidase Activity in a Subgroup of Schizophrenic Patients

BY JOSEPH J. SCHILDKRAUT, M.D., JAMES M. HERZOG, M.D., PAUL J. ORSULAK, PH.D., STUART E. EDELMAN, M.D., HARVEY M. SHEIN, M.D., AND SHERVERTH H. FRAZIER, M.D.

Platelet monoamine oxidase (MAO) activity was significantly lower than control values in a subgroup of 16 schizophrenic patients (most of whom were paranoid) characterized by the presence of auditory hallucinations and delusions. Platelet MAO activity was not reduced in 16 other schizophrenic patients without auditory hallucinations. This finding suggests that reduced platelet MAO activity is not found in all schizophrenic patients but tends to occur in a clinically identifiable subgroup.

SEVERAL STUDIES have reported that some patients with schizophrenic disorders have lower platelet monoamine oxidase (MAO) activity than controls (1–4), but this has not been observed in other studies (5–7). It has been noted that reduced platelet MAO activity occurs more commonly in chronic than in acute schizophrenic disorders (8). However, other clinical characteristics that may be associated with reduced platelet MAO activity in schizophrenic patients have not yet been demonstrated.

In a pilot study of platelet MAO activity in schizophrenic patients hospitalized at the Massachusetts Mental Health Center or the McLean Hospital, we noted that reduced platelet MAO activity regularly occurred in a subgroup of schizophrenic patients (most of whom were paranoid) with auditory hallucinations and delusions. In order to confirm this initial observation, we examined platelet MAO activity in a subsequent series of patients hospitalized at the Massachusetts Mental Health Center with the diagnosis of schizophrenia. In both of these studies the clinical diagnosis was made in accordance with the *Diagnostic and Statistical Manual of Mental Disorders* of the American Psychiatric Association (9).

METHOD

The medical records of the schizophrenic patients were reviewed in detail by a psychiatrist blind to the biochemical data. The patients were then categorized according to whether they had a record of both auditory hallucinations and delusions. The subgroup of patients with both documented auditory hallucinations and delusions (N=16) was designated S-2, whereas the remaining subgroup of schizophrenic patients (N=16) was designated S-1. Each subgroup contained 8 men and 8 women. Most of the male patients and half of the female patients in the S-2 subgroup had delusions or auditory hallucinations that were of a persecutory or accusatory nature and thus were designated paranoid.

Platelets were isolated from blood routinely obtained at the time of hospital admission by a modifi-

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Drs. Schildkraut and Frazier are Professors of Psychiatry, Drs. Herzog and Edelman are Clinical Instructors in Psychiatry, and Dr. Orsulak is Associate in Psychiatry, Harvard Medical School, Boston, Mass. Dr. Schildkraut is also Director and Dr. Orsulak is Associate Director, Neuropsychopharmacology Laboratory, Massachusetts Mental Health Center, 74 Fenwood Rd., Boston, Mass. 02115. Dr. Frazier is also Psychiatrist-in-Chief, McLean Hospital. Dr. Shein, until his death on July 18, 1974, was Associate Professor of Psychiatry, Harvard Medical School, and Psychiatrist, McLean Hospital.

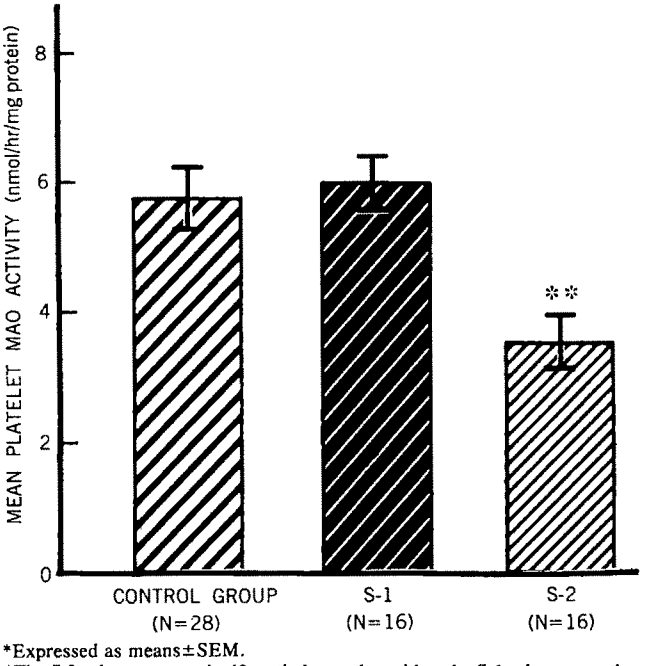
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cation of the procedure of Murphy and associates (10). MAO activity was determined by measuring the deamination of ^{14}C-tryptamine bissuccinate and was expressed as nanomoles of tryptamine deaminated per hour per milligram of platelet protein (11). When this method was used in a series of 26 subjects on whom 3 or more consecutive daily samples were examined, the intrasubject coefficient of variation was 10%±1.5% (mean±SEM). Similarly, the average coefficient of variation for samples of pooled platelets analyzed repeatedly over a period of 4 months was approximately 10%.

Patients with histories of alcoholism were excluded from this study since we and others have observed reduced platelet MAO activity in some patients with alcoholism (12, 13). Patients were also excluded if there was a prominent affective disturbance (mania or depression) or evidence of any major neurological or medical illness. In order to control for the effects of advancing age on platelet MAO activity (14), patients over 45 years old were not studied. We also studied platelet MAO activity in an age- and sex-matched control group consisting of 28 staff members of the Massachusetts Mental Health Center.

FIGURE 1
Platelet MAO Activity* in Two Subgroups of Schizophrenic Patients



*Expressed as means±SEM.
**The S-2 subgroup was significantly lower than either the S-1 subgroup or the control group ($p<.001$, t test).

RESULTS

As shown in figure 1, patients with a diagnosis of schizophrenia but no documentation of auditory hallucinations and delusions (the S-1 subgroup) had platelet MAO activity that was very similar to that of the control group. In contrast, schizophrenic patients with both auditory hallucinations and delusions (the S-2 subgroup) had significantly lower platelet MAO activity ($p<.001$) There was very little overlap in the individual values of MAO activity in patients in the two schizophrenic subgroups.

The S-1 and S-2 subgroups did not differ in age or sex distribution, and MAO activity was not correlated with antipsychotic drug dosage. Moreover, MAO activity was not related to the number of previous hospitalizations or to the length of time since the first documented hospital admission, thus suggesting that the reduced platelet MAO activity in the S-2 subgroup was not merely related to chronicity of illness. Within the S-2 subgroup, the lowest individual values of platelet MAO activity were observed in patients who were paranoid, as suggested by observations made in our earlier pilot study.

DISCUSSION

Our findings indicate that reduced platelet MAO activity is not found in all schizophrenic patients; rather, it tends to occur in a clinically identifiable subgroup. The original blind selection of the S-2 subgroup was made on the basis of a history of both auditory hallucinations and delusions. However, further examination of the data suggested that the primary discriminating symptom may have been the presence of auditory hallucinations since none of the patients in the S-1 subgroup were found to have auditory hallucinations, although many had delusions. Thus it is possible that our S-2 subgroup may correspond to schizophrenic subtypes identified in other studies on the basis of auditory hallucinations (15).

A prospective study is now under way to determine whether the presence of auditory hallucinations (particularly in conjunction with paranoid features) may be a sufficient criterion for identifying a subgroup of schizophrenic patients with reduced platelet MAO activity. We also hope to clarify the relationship of the subgroup of schizophrenic disorders with reduced platelet MAO activity to paranoid schizophrenia.

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The 24-Hour Lithium Level as a Prognosticator of Dosage Requirements: A 2-Year Follow-Up Study

BY THOMAS B. COOPER, M.A., AND GEORGE M. SIMPSON, M.D.

The authors have previously described a technique whereby individual lithium dosage requirements can be predicted from 24-hour blood samples. Further experience over a 2-year period has shown the predictions to be reproducible over time. A micromethod for lithium determination is described, as are several cases in which aberrant results were found to indicate inadequate laboratory techniques or patients' failure to take medication. Because the technique reveals immediately those patients at the extremes of dosage ranges, toxicity and the need for frequent sampling can be avoided.

APPROXIMATELY two years ago, we wrote two papers on the kinetic analysis of data collected from patients given a single 600-mg dose of lithium carbonate. In the first paper (1), we were able to show that we could predict, with acceptable clinical accuracy, the individual dosage requirements of each patient. In the second paper (2), we demonstrated that each patient's dosage could be ascertained from a single blood sample collected 24 hours after a 600-mg dose of lithium carbonate. A correlation of .97 (N=19) was obtained when the 24-hour blood level was compared with the steady state level after a fixed dosage regimen. The predicted individual dosage requirements, derived from the empirical data described in the previously mentioned papers (see table 1), have not required alteration after experience with more than 100 patients' predictions. Thus, from a single blood sample taken 24 hours after a 600-mg dose of lithium, it is possible to predict an individual dosage regimen to ensure that a patient will be within the therapeutic range.[1]

During the intervening years, our experience has shown that in patients who ingested the medication and from whom blood samples were collected 24 hours later, the prediction always resulted in a steady state lithium level of 0.6–1.2 mEq/liter. The steady state level is that found in blood collected immediately before the next dose of medication is due; this sample approximates the lowest level to be found during the continuous ingestion of the medication on a fixed schedule.

The convenience of the technique is best seen in an outpatient situation, wherein the patient is given 600

The authors are with the Rockland Research Institute, Orangeburg, N.Y. 10962, where Mr. Cooper is Chief Investigator and Dr. Simpson is Principal Research Psychiatrist.

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[1]Since this paper was accepted for publication, Seifert and associates (3) have confirmed this prediction technique using a long-acting form of lithium.

mg of lithium carbonate by the clinician or to take at a later time so that attendance at the laboratory or physician's office will be within normal working hours. The results of the blood analysis should be available on the same day and the patient should then be started on the predicted dosage schedule. When laboratory results are not quickly obtainable, the clinician can start the patient on a schedule of medication immediately after the 24-hour blood sample has been collected and the dosage can be adjusted if necessary when the laboratory data are available.

During the years the technique has been in use, there have been several patients who have had 24-hour blood levels determined and who received lithium but stopped taking it for a variety of reasons. On retesting, their 24-hour blood levels were found to indicate the same dosage requirements as were determined previously. In a study of the effects of lithium on tardive dyskinesia (4), 600-mg doses of lithium carbonate were given and 24-hour blood samples were collected on 3 separate occasions, each dose being separated by a drug-free interval of at least 1 week. As table 2 indicates, the level is highly reproducible and the predictions are therefore equally reproducible. In all cases, the patient achieved satisfactory blood levels on the predicted dosage.

Schou (5) reported that renal clearance of lithium was decreased in older patients. Examination of the predictions obtained for the aforementioned study of lithium and tardive dyskinesia in older patients (4) (age range 57–82 years, mean age=72) indicated that all but 2 patients required 300 mg three times a day or less to achieve a level of 0.6–1.2 mEq/liter. This confirms Schou's original observation.

TABLE 1
Dosages Required to Achieve a Serum Lithium Level of 0.6–1.2 mEq/liter*

24-Hour Serum Level After Single Loading Dose (mEq/liter)	Dosage**
Less than .05	1200 mg three times a day
.05–.09	900 mg three times a day
.10–.14	600 mg three times a day
.15–.19	300 mg four times a day
.20–.23	300 mg three times a day
.24–.30	300 mg twice a day
More than .30	300 mg twice a day***

*Reprinted from Cooper and associates (2).
**The regimen selected minimizes fluctuation in the plasma level while maintaining a schedule the patient can adhere to. Variation in this regimen can be made at the choice of the clinician, but the total daily dose must remain the same. All steady state values are collected just before the next medication is to be taken.
***Use extreme caution.

TABLE 2
Lithium Levels (mEq/liter) 24 Hours After Administration of 600 mg of Lithium Carbonate*

Patient	Date of Administration		
	8/22/74	9/6/74	9/12/74
1	.18	.19	.16
2	.23	.25	.21
3	.24	.24	.21
4	.29	.30	.30
5	.23	.25	.24
6	.21	.20	.19
7	.25	.27	—
8	.31	.31	.27
9	.20	.17	.18
10	.22	.22	.21
11	.21	.23	.21
12	.19	.19	.16
13	.26	.27	.28
14	.18	.17	.18

* Figures are for three separate loading doses for each patient.

MICROMETHOD FOR LITHIUM DETERMINATION

With the greater likelihood of "difficult veins" and the general frailty of a geriatric population, as well as the current interest in the use of lithium in children, a true micromethod for the determination of lithium seemed esssential. We therefore developed a suitable method, requiring 50 μl of plasma, wherein blood can be collected by finger prick into a heparinized microhematocrit tube (technical details have been published elsewhere [6]). For convenience, the technique is given in abbreviated form in appendix 1. The precision and accuracy of the micromethod were not significantly different from our routine macromethod, which has a coefficient of variation of 1.5%. Therefore, because of the high correlation between the methods, they can be used interchangeably to predict dosage requirements or to ascertain the steady state plasma level. In our own hospitals, nursing staff collect blood via finger prick and then place the hematocrit tubes in a clean, stoppered, labeled tube for transportation to the laboratory. An additional benefit is that the red cell lithium: plasma lithium ratio can be determined. This measure has been claimed to be increased in manic-depressive psychosis and to have importance in patients who have toxic symptoms without abnormal plasma levels (7–9). (We have never observed a change in the ratio in patients demonstrating this phenomenon.) The ratio has also been claimed to be of value in selecting a subgroup of depressed patients who may respond to lithium as an antidepressant (10).

PREREQUISITES FOR PREDICTION

Clearly, the prediction technique depends upon the following: 1) the patient ingesting all of the medication, 2) the blood sample being collected at the correct time (less than 60 minutes before the next scheduled dose), 3) the laboratory being capable of accurate, reproduc-

ible analysis, and 4) the clinician understanding the limitations of any predictive technique.

Blackwell (11) has written an excellent mini-review and described a study determining the failure rate in patients taking any kind of medication. In our own experience with lithium and other psychotropic drugs, there have been patients whom we trusted and who we felt confident were taking their medication—but hard laboratory data revealed that they were not. Thus, we have seen unmeasurable serum lithium levels 2 hours after a routine 600-mg dose of lithium, but a level of 0.6 when the medication was dissolved in orange juice and the patient was observed for the full 2-hour period. These patients have admitted hiding the medication and taking it later, discarding it, or giving it to other patients; some even have the ability to hold medication in the nasopharyngeal area to avoid detection. One outpatient who consistently had steady state levels of lithium within the therapeutic range admitted that she took two capsules (600 mg total) 2 hours before visiting the laboratory each month, but that was the only time she took the medication. These examples are cited to draw attention to the fact that if an aberrant result is found, the patient as well as the laboratory technique should be suspected; one may thus identify patients who will require careful monitoring to ascertain that they are taking their medication.

The determination of serum lithium levels is not a difficult laboratory procedure, and it is not unreasonable to require accuracy to the second decimal place. Our general experience with this technique when used in other units by other workers has been good (i.e., they found the prediction technique useful); however, the exceptions have, upon investigation, yielded somewhat startling findings.

REASONS FOR ABERRANT RESULTS

Several investigators informed us that if they split samples and send them to the same laboratory as different samples, the results can vary by as much as 0.5 mEq/liter. Recently, a resident in our psychiatric training program who had used this technique transferred to another hospital and found a laboratory result of 0.86 mEq/liter at 24 hours for the first patient given 600 mg of lithium. After waiting for 3 days, she repeated the procedure and a level of 0.39 mEq/liter was reported. Table 2 shows that this kind of variation is impossible unless the patient took extra lithium the first time or the laboratory was incapable of measuring lithium levels. This experience makes it clear that laboratory accuracy is a prerequisite for this technique. It may be necessary for the physician to make the pathologist and/or clinical chemist aware of the importance of this particular sample so that additional care will be exercised. This, however, is a poor substitute for a well-controlled laboratory procedure.

Early in our experience, one clinician said that he found our predictions consistently high and that he always used lower dosages; upon further inquiry it turned out that this physician did not want his patients to have serum lithium levels above 0.9 mEq/liter; he preferred a range of 0.6–0.8 mEq/liter. The technique as it currently stands cannot predict such a narrow range, but we find that even the physician who demands the narrowest range can still obtain valuable information, in that he can at least know that a patient will require very little (300 mg twice a day or less) or a great deal of medication (900 mg three times a day or more) to achieve the generally accepted range (0.6–1.2 mEq/liter) and can tailor his own prescribing with this in mind.

In another case a clinician collected the blood 2 hours after the patient ingested the medication rather than immediately before the next dose. Clearly, this will produce higher levels and the erroneous conclusion that the technique is invalid.

DISCUSSION

We should emphasize that this technique predicts individual dosage requirements with acceptable clinical accuracy; it does not, however, predict subsequent changes in renal clearance or other physiological changes. We have been asked about this on occasion, and there is no way that we can predict this situation—nor, as far as we know, can anyone else. It is clear that a patient who was found to require 600 mg three times a day to achieve steady state levels might well become toxic on this dosage if renal clearance diminishes during the therapy. The data on the needs of the elderly population studied (see table 2) clearly suggest that the amount of lithium required decreases over many years.

Since the prediction technique has become routine, we have found that less frequent blood sampling has been required during the initial medication period and that medication changes are rarely needed. Subsequent monthly and, later, quarterly or semiannual blood determinations are still required with or without the prediction technique.

Blood levels can vary widely among patients. We have found patients who have required less than 300 mg twice a day or more than 3 g a day to achieve steady state levels within the therapeutic range. One of our patients requires 150 mg (1/2 tablet) a day and on this regimen has a steady state level of approximately 0.7 mEq/liter. The majority of the clinicians who are using the technique have found it to be of value. We feel that the exceptions are idiosyncratic, e.g., a clinician who feels there is no need for such a method because the manic episode is controlled by neuroleptics, while prophylactic lithium does not require rapid achievement of a therapeutic level. Our own opinion is that because the technique is simple and indicates immediately those patients at the extremes of the dosage range, toxicity and the need for frequent blood sampling can be avoided.

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APPENDIX I
Micromethod for Serum Lithium Determination

The finger is cleansed and the standard finger prick method is used. The blood is collected by means of heparinized capillary microhematocrit tubes, which are centrifuged so that a clear plasma layer is obtained. The plasma is withdrawn (50 μl) by means of a Hamilton Syringer number 70001-N (sufficient plasma can be obtained from two capillaries) and placed in a small vial containing 1 ml of deionized water.

Aqueous standards containing 0.5, 1.0, 1.5, and 2.0 mEq/liter of lithium are prepared fresh from a concentrated stock solution each day. These standards are further diluted with deionized water (1:20), and to 1 ml of each of these diluted standards is added 50 μl of lithium-free plasma or 50 μl of 6% dextran solution. These solutions are then aspirated directly into a Perkin-Elmer Model 303 automatic absorption spectrophotometer with a DCR-1 attachment (or comparable instrumentation) and four readings are obtained from the equipment (operating under continuous mode). The last three readings are recorded and the mean is determined. The equipment is calibrated before the tests to read out directly in milliequivalents by aspiring a larger volume of the 2.0 mEq/liter standard, similarly diluted. Readings are obtained until the equipment stability and calibration are ascertained.

Our standard macromethod uses 0.5 ml of serum plus 2 ml of water and standards similarly diluted with either serum or 6% dextran. The coefficient of variation for this method is 1.5%.

Extrapyramidal Side Effects in Lithium Maintenance Therapy

BY MARC H. BRANCHEY, M.D., JESS CHARLES, M.D., AND GEORGE M. SIMPSON, M.D.

The authors neurologically examined 36 patients who had been maintained on lithium therapy for periods ranging from 6 months to 7 years to determine the presence of parkinson-like side effects. Only a few patients demonstrated rigidity, including cogwheel rigidity, and this was at a low level of severity. These results do not support the previously reported frequent occurrence of cogwheel rigidity in patients on lithium maintenance.

A PARKINSON-LIKE syndrome that includes tremor, rigidity (including occasional cogwheel rigidity), and salivation was recognized as one of the side effects of such neuroleptic medications as phenothiazines and butyrophenones shortly after their introduction on the drug market (1). Simpson and associates developed (2) and extensively tested (3) a scale, based on the clinical evaluation of various symptoms, that can quantify the severity of this syndrome. Neuroleptic-induced extrapyramidal symptoms are thought to result from the catecholamine blocking action of these drugs, which induces an imbalance in the parts of the central nervous system that control motility; they are known to respond to anticholinergic agents.

The motor disturbance most frequently induced by lithium is evidenced by a fine, rapid tremor. Its frequency is about 10 cps in contrast to the 5–6 cps noted in Parkinson's disease; it is usually not accompanied by rigidity and it does not respond to anticholinergic drugs (4). These differences suggest that the mechanism of action of lithium is different from that of neuroleptic drugs.

However, a recent paper by Shopsin and Gershon (5) described the occurrence of cogwheel rigidity in more than half of a group of patients receiving lithium therapy for over a year. We therefore decided to examine the frequency of this symptom in our outpatient lithium maintenance clinic, employing the rating scale that we have used extensively for a large number of studies over many years.

Dr. Branchey is Research Psychiatrist and Dr. Simpson is Principal Research Psychiatrist, Rockland Research Institute, Orangeburg, N.Y. 10962. Dr. Charles is Director, Outpatient Psychiatric Department, Bergen Pines County Hospital, Paramus, N.J.

METHOD

Thirty-six outpatients (11 male and 25 female), with a mean age of 46.1 years, who had received lithium for a period ranging from 6 months to 7 years comprised the study population. There were 33 manic-depressive and 3 schizo-affective patients. None of the patients had received neuroleptics for at least 6 months; 8 of them were taking antidepressant medication in addition to lithium. In all patients, lithium serum levels were within the therapeutic range (0.6–1.2 mEq/liter).

Patients underwent a neurologic examination at the time of their regular outpatient visit. The Neurological Rating Scale for extrapyramidal side effects, developed by Simpson and Angus (3), was used to record the severity of symptoms. The scale consists of 10 items, with a severity rating range from 0 to 4. Seven of these items (gait, arm dropping, shoulder shaking, elbow rigidity, wrist rigidity, leg pendulousness, and head dropping) are related mostly to the level of rigidity; the other items are tremor, glabella tap (which measures the severity of induced blepharospasm), and salivation. An additional item, cogwheel rigidity (also rated from 0 to 4), was added to the original scale for the purpose of this study and measured the amount of rapidly fluctuating resistance to stretching of the patient's forearm by the examiner while the patient remained passive.

In addition 15 patients were reexamined by the investigator who participated in the development of the scale (G.M.S.), and several patients were examined more than twice. In view of the fluctuating nature of extrapyramidal symptoms (2), the highest score was recorded for those subjects examined two or more times.

RESULTS

Only 3 of the 36 patients had some amount of cogwheel rigidity (score of 1); this occurred together with other limb rigidity and tremor of the extremities. These 3 patients had been taking lithium for 2 years or more. Six other patients had rigidity (not of the cogwheel type) and tremor; 15 showed tremor but no rigidity, and 2 had rigidity but no tremor. Four patients who showed a positive response to glabella tap were among the ones showing evidence of tremor. The 10 remaining patients appeared to be free of neurologic symptoms. It should be pointed out that the degree of rigidi-

ty observed in the 11 patients with positive ratings was very low. None of the individual items related to rigidity was scored higher than 1, a rating that could occasionally be observed in a patient who does not relax completely. The low level of rigidity is also indicated by the fact that usually only 1 or 2 out of the 7 indicators of rigidity were positive.[1] A fairly large number of patients (24 out of 36) showed tremor, which was fine and rapid and mildly or moderately intense. Very few patients had subjective complaints, and some were surprised to note tremor during the examination. There did not appear to be any systematic relationship between the duration of lithium treatment and the occurrence of tremor or rigidity.

DISCUSSION

These findings do not support the previously reported high prevalence of cogwheel rigidity among patients undergoing lithium maintenance therapy. Even the 3 patients who had been taking this medication for 6 years or more were without cogwheel rigidity on two separate measurements.

It is worth noting that all the patients showing cogwheel rigidity also demonstrated evidence of rigidity and tremor. Whether cogwheel rigidity is simply a combination of tremor and rigidity or a separate entity is debatable (6). A possible explanation for the high level of cogwheel rigidity in the sample examined by Shopsin and Gershon (5) may be that many of their patients had been previously treated with neuroleptics and that a certain amount of rigidity was present as a residual effect. One of us (G.M.S.) had previously reported the occurrence of extrapyramidal symptoms in patients whose neuroleptic treatment had been discontinued for more than 90 days (2).

The glabellar reflex has long been considered as pathognomonic of a lesion of basal ganglia, as seen in Parkinson's disease (7). Pearce and associates (8), however, also reported its occurrence in diffuse cerebral disease. Klawans and Goodwin (9) showed that the administration of L-dopa to patients suffering from parkinsonism decreased the severity of the glabellar reflex. Our observation of this symptom in 4 patients on lithium maintenance therapy is therefore an argument in favor of an extrapyramidal effect of lithium; the possibility that it indicates a nonspecific effect on the central nervous system cannot, however, be ruled out.

We noted tremor, in a mild or moderate degree, in 66% of the patients. This frequency of occurrence is comparable with that reported by others; according to Vacaflor and associates (10), tremor can be found in up to 80% of patients taking lithium. The tremor observed in this study was fine and rapid, essentially different from that of the neuroleptic-induced extrapyramidal syndrome.

It is possible that the long-term use of a drug such as lithium may produce side effects that are not clearly observed following shorter periods of administration. For instance, it is now well known that the long-term use of neuroleptic drugs can induce a complex dyskinetic syndrome; it took many years of extensive use of these drugs before the dyskinetic syndrome was properly recognized and identified (11). Our findings in patients maintained on lithium for up to a few years are essentially negative; however, a rather high occurrence of tremor, an occasional glabellar reflex, and a low level of rigidity were noted. In view of the increasing use of lithium maintenance therapy and our few positive findings, a careful monitoring of the neurologic conditions of patients treated with lithium seems warranted.

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[1]None of the patients had a total rigidity score higher than 3, which is considered to be within normal limits.

Catatonia, Gastric Hyperacidity, and Fatal Aspiration: A Preventable Syndrome

BY ROBERT F. BORT, M.D.

Sudden death from aspiration pneumonitis during acute catatonia is a generally recognized but poorly understood occurrence. The author describes a case in which a schizophrenic patient died following aspiration of gastric contents. In view of findings that suggest that the acidity of gastric contents is related to morbidity following aspiration, he recommends that liquid antacid medication be given routinely to catatonic patients.

SUDDEN DEATH in patients with schizophrenia is not uncommon, especially among older and chronically ill patients (1, 2). The cause of death has been attributed to cardiac arrhythmia, hypotension, and aspiration of gastric contents (1–5). Fatal aspiration appears to be related to a primary dysfunction of esophageal motility in schizophrenic patients (5, 6) and unrelated to the side effects of phenothiazines on esophageal function (3, 4, 7). Some recommendations for the prevention of fatal acid aspiration can be derived from the following case.

CASE REPORT

A 19-year-old man was referred to a maximum security state forensic hospital for evaluation of competency to stand trial on a charge of armed robbery. He had a history of two prior hospitalizations for severe schizophrenic illness, which had responded each time to phenothiazine treatment.

On admission the patient was combative and incoherent, and his thinking processes were grossly disorganized. His initial behavior was reminiscent of the continuous pacing of a caged wild animal. The patient sat in his urine, smeared food about the seclusion room, and assumed bizarre postures. He became mute within several hours of admission.

Over the next 12 days the patient's catatonic excitement and hostility gradually decreased, and he began speaking again in two- or three-word phrases. Physical findings were within normal limits except for almost total immobility. The patient lay motionless and resisted attempts by the examiner to initiate any motion; e.g., efforts to flex an extremity were met with active extension efforts by the patient. During his 12th hospital day the patient suddenly developed respiratory distress (tachypnea and cyanosis) followed rapidly by hypotension and cardiac arrest. He was rushed to a nearby hospital emergency room, where he was pronounced dead on arrival.

A complete post mortem examination indicated that aspiration pneumonitis was the cause of death. Gastric contents were also found in the larynx. Toxicological findings were negative except for blood levels of chlorpromazine consistent with the prescribed dosage (600 mg orally every 24 hours).

Dr. Bort is an Instructor, Department of Psychiatry, University of Michigan School of Medicine, Ann Arbor, Mich., and a Staff Psychiatrist, University of Michigan Alcoholism and Drug Abuse Clinic, Riverview Building, 900 Wall St., Ann Arbor, Mich. 48105.

DISCUSSION

Standard psychiatric textbooks generally discuss the management of catatonic patients in terms of psychological and somatic treatments. Any mention of the high risk of aspiration in acutely ill, withdrawn catatonic patients is notably absent.

Cameron (1) reported a high mortality rate from aspiration pneumonitis in both acutely and chronically ill patients and described appropriate nursing care procedures related to proper positioning. To facilitate proper drainage of tracheobronchial secretions, high-risk patients should be positioned on their sides, with the foot of the bed elevated six to nine inches, a pillow placed between their legs and against their backs, and no pillow used under the head. Findings by Ribaudo (8), which indicated that the acidity of the gastric aspirate rather than the amount of the aspirate or amount of solids in the aspirate determined morbidity and mortality in aspiration pneumonitis should also be considered. Related studies in another high-risk group, parturient patients undergoing Caesarian section (9, 10), demonstrated the feasibility of using antacids to raise the gastric pH to a relatively safe level (pH greater than 2.4). This appears to markedly reduce the risk of morbidity when acid aspiration inadvertently occurs.

It appears that this latter approach should be adopted as part of the routine treatment of severely withdrawn catatonic or other immobilized schizophrenic patients. In addition to appropriate positioning to allow adequate oropharyngeal drainage, liquid ant-

acid medication, such as magaldrate suspension, should be given in dosages of 15 milliliters every 3 hours until the phase of the illness during which the patient is immobilized has passed.

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Siblings as Therapists: A Behavioral Approach

BY NANCY BROWN MILLER, M.S.W., AND DENNIS P. CANTWELL, M.D.

Although many family therapy programs have taught behavior modification techniques to parents, few have involved siblings as therapeutic agents in the treatment of retarded, emotionally disturbed, or physically handicapped children. The authors describe their experiences with two families in which siblings were taught to use simple procedures to encourage desired behaviors in a disturbed brother or sister. They conclude that further investigation is needed to refine sibling training techniques and suggest that involvement in treatment programs may have beneficial influences on the normal siblings of disturbed children.

MOST SCHOOLS OF THOUGHT in child psychiatry emphasize that the nature of the interactions between a child and his family is an important factor that may contribute to the development of psychopathology in the child. However, they differ on which elements of the interactions are important and how family members may or may not be involved in treatment of the affected child (1). Practitioners who regard family members as agents in the genesis of psychiatric disorders in children usually exclude family members from treatment and work individually with the child. If the parents are worked with, they are worked with by separate therapists who focus on parental problems that may have contributed to the development of psychopathology in the child. Little or no time is spent giving parents practical suggestions on how to deal with their child's problem.

Family therapists, using a systems-oriented approach, treat all family members together, but most use a framework based on the same underlying principles as those therapists who work with parents and child separately. Recently, however, the behavioral school has taken the position that direct modification of a child's behavior in his natural environment by family members under the direction of a clinician might be more fruitful than the traditional office-oriented approach. Early efforts at using parents as therapeutic agents concentrated on discreet maladaptive behaviors of the child that were modified by operant techniques taught to the parents (2). Recently there has been a shift to the "parent-child interaction system" as the target for intervention. There is now concern not only for immediate behavioral change, but also for maintenance of new desirable behavior by all family members (3–5).

Direct family intervention at this level is aimed at providing parents with techniques and skills that will

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The authors are with the Neuro-Psychiatric Institute, University of California, Los Angeles, 760 Westwood Plaza, Los Angeles, Calif., 90024, where Ms. Miller is Social Work Training Coordinator, University Affiliated Facility, and Dr. Cantwell is Director of Professional Education, Division of Child Psychiatry and Developmental Disabilities.

enhance their parenting ability. Practitioners of direct family intervention hold that intervention aimed solely at changing the behavior of a child over a short period of time will have only limited success. However, intervention programs geared toward the improvement of parents' general competence will not only cause the child's negative behavior to decrease, but will result in an overall positive change in family interaction. The most successful exponent of this approach has been Gerald Patterson at the Oregon Research Institute in Eugene, Ore. He has parents attend a 10- to 12-week training program in parenting skills that requires parents to master one skill level before progressing to the next one (6).

Even sophisticated behavioral programs have all but neglected the use of siblings as agents of therapeutic change in the family. This paper will describe the treatment programs of two families in which siblings were involved as therapeutic agents. We will focus on the effectiveness of siblings in the treatment programs and problems encountered in using them as therapeutic agents.

CASE REPORTS

The impetus for systematically involving siblings in child treatment came from our experiences with two families in which siblings were successfully undoing the parents' efforts to carry out behavioral programs with target children. One 3-year-old boy was having about 12 temper tantrums a day. The parents had learned to ignore the tantrums by leaving the room, but the rate was not decreasing. The mother discovered that whenever she left, the child's 14-year-old sister entered the room and held him because she felt sorry for him and couldn't stand to hear him cry. Two sessions were held with the sister to explain social learning principles, and with great reluctance she was able to change her behavior. The tantrums decreased to two a day within 2 weeks.

In the second family, a 9-year-old autistic boy engaged in a high rate of what the family called "crazy talk." The parents had learned to ignore his inappropriate speech, but his four older sisters thought it was quite funny and "talked crazy" back to him. After the parents tried unsuccessfully to gain the siblings' cooperation, three sessions were held with them in the home to teach them how—and why—to ignore this behavior and to give him extra attention for talking appropriately. There was a significant decrease in the amount of "crazy talk," and the girls were also able to generalize their learning to other situations, such as whining and temper tantrums.

Case 1. Sally, age 4, is the youngest of five children, with three brothers and one sister. She was born prematurely with a congenital facial anomaly, and her early development was slow. When referred to us she was speaking in one- or two-word phrases that were unintelligible except to her family, would not play alone but insisted on being beside her mother most of the time, and engaged in frequent whining. Observations of family interaction showed that the whole family treated her with excessive indulgence. For example, when Sally moved around the room or played with toys, at least one member of the family would remind her to be careful. If she dropped a toy, someone would pick it up for her. The family reinforced her limited language skills by responding to her gestures and one-word interactions and by frequently anticipating her wishes. She was very shy and passive and showed little affect.

The treatment program consisted of working with the parents for four sessions to teach them principles of normal child development and social learning. Videotape sessions were held to give the parents feedback on their interaction with Sally. The siblings were then brought into the program; they observed the mother playing with Sally and she explained social learning principles to them in the therapist's presence. Videotape sessions were again held and the family members were able to observe their progress (7).

By the end of the 10-week program, Sally was speaking in four- and five-word sentences that were clearly intelligible. She would frequently say something and then ask, "Can you understand me?" The emphasis throughout the program was on facilitating Sally's development by shaping her speech and encouraging her to take risks and to play alone for longer periods of time. Eventually she was able to play alone for as long as 45 minutes. Follow-up observations 6 months after treatment indicated that all gains had been maintained and that Sally had become more outgoing and assertive in her family interactions.

Case 2. Andy, 11, was diagnosed as moderately retarded with autistic features. He had four older siblings: two sisters, 20 and 18, and two brothers, 16 and 15. Presenting problems included bizarre behavior, swearing, running away from home, stealing food from the refrigerator, and extreme hyperactivity.

The parents reported that the oldest daughter was willing to help take care of Andy but the other siblings were generally uncooperative. Efforts to involve them in Andy's care resulted in many family conflicts.

All of the siblings were willing to participate in the program. Treatment was begun while Andy was in a 5-day-a-week inpatient program and continued after he went home.

During the first two sessions, social learning principles were taught to the family through didactic materials and role playing. One behavior selected for intervention was taking food from the refrigerator, which occurred about 20 times a day. If a sibling saw this behavior occur, he or she would call the parents to intervene or would yell at Andy to stop. This would frequently be followed by chasing and giggling. A time-out procedure was established; anyone who saw a transgression would announce, "No stealing food, Andy. Three minutes of time out," and then take him to a predesignated chair in the corner of the hall, tell him to sit, and stand silently behind him. The first day, his 16-year-old brother caught him and carried out the intervention; about 1 hour later his 15-year-old brother repeated the procedure. About 3 hours later, Andy was observed as he went to the refrigerator, opened the door, closed it, and then went to the time-out chair and sat down. The problem did not recur.

One of the most effective interventions in this family was reprogramming the time each family member spent with Andy. All of the siblings had complained that their own time was frequently impinged upon when they were told to take

care of him. A schedule was made so that each sibling had 2 regular days "on call" during which he or she might have to help with Andy. On other days they were free to plan their own activities. In addition, each sibling was asked to spend 15 minutes a day in a one-to-one activity related to Andy's interests, such as playing with cars or going for a walk. During these sessions they were to encourage eye contact, coherent speech, and appropriate social behaviors. This aspect of the program was very effective in decreasing family arguing and in increasing positive interactions between Andy and his siblings.

BENEFITS OF SIBLING INVOLVEMENT

Several positive results of involving siblings in treatment programs have been identified.

1. Teaching new skills. Older siblings frequently "teach" younger siblings. The teaching is usually haphazard, sporadic, and based on the interests and attention span of the "teacher." By teaching siblings what to teach and how to teach, it may be possible to use this natural tendency to bring about actual gains for the target child. The tasks taught may be self-help skills such as dressing or eating, or more abstract skills such as recognizing colors, letters, or words.

2. Consistency: providing a united front. If all members of a child's environment react to selected behaviors in the same way, there may be more rapid learning of desired behaviors and a faster decrease of undesirable behaviors. This is true whether the planned intervention is praise, ignoring, or punishment. A consistent, predictable environment is particularly important for children with developmental disabilities. The advantages for children who require a longer time to learn of a family environment in which everyone works together to produce behavioral change are obvious.

3. Increased positive interactions. Negative interaction frequently results because siblings feel that their own lives are being impinged upon by the needs of the target child, who may also receive more parental attention and "get away" with more. By structuring both the amount and content of the time siblings spend with the target child, they can have successful, positive experiences together and know that the family is working together in a constructive, consistent manner.

PROBLEMS IN SIBLING TRAINING

1. Attention span. Sessions should be well planned and made up of a variety of short segments that alternate didactic teaching with active role playing and behavioral rehearsal. Length of sessions will vary with the age and behavior of the siblings, but generally 1 to 1½ hours is a maximum limit.

2. Sharing family secrets. We observed on several occasions that siblings did not feel comfortable talking about each other or their parents. This was particularly clear in one family in which the father had apparently spanked the target child rather severely, contrary to instruction. One of the siblings blurted this fact out, and the father gave him a harsh look and denied it. Such situations are difficult for both parents and children, and it is important to establish rules at the very beginning of the program, including a rule that all behavior in the family that is observed by anyone is subject to discussion in the meetings.

3. Following through on assignments. Siblings occasionally forget to write down a required assignment unless they recognize its importance. It may be necessary, especially in the early stages of treatment, for the therapist to call the family between sessions and talk to each family member about his progress in the program. The mother or father or a specified sibling may be "manager" of the program for a week to see that assignments are carried out.

4. Presence of target child. Trying to carry out an organized, efficient session can be very difficult when the referred child is present, particularly when the problem behaviors are hyperactivity and short attention span. In Andy's family, the therapist decided early that one target behavior should be independent play, but the program was only partially successful in achieving this goal.

It would be ideal to have the child present for only a part of each session to allow the family to practice new behaviors. However, finding a babysitter for many problem children is a difficult endeavor. A babysitter in the clinic may provide a workable solution to this problem.

5. Participation by family members. The dominating parent and the passive child, or other comparable combinations, present problems that require the therapists's active intervention during sessions. Siblings often "talk for" each other, and parents often "talk for" the children. The therapist needs to be in control of who talks when to assure equal participation by all family members.

DISCUSSION

More than 80% of the children growing up in the United States have siblings (8). Although much has been written about the importance of peer relationships to child development (9) and such terms as "sibling rivalry" and the "middle child syndrome" are used as if they represent some specific situation or syndrome, almost nothing of substance is known about the influence of siblings on each other (10). It is only natural to speculate that sibling relationships are important because of the amount of time and space they share and because of the natural hierarchy that exists simply because of birth order.

The interaction between siblings duplicates in microcosm most of the significant social interaction experiences that the child faces as he or she goes through adolescence and adulthood. Patterns of loyalty, and helpfulness and protection, or of conflict, domination,

and competition may be first learned by a child in relationships and interactions with siblings (9). These patterns may later be generalized to other social relationships. Because of the intense long-term relationships that siblings have with each other, we may assume that they influence and control, or attempt to control, each other on a daily basis. It seems logical, then, that siblings ought to be involved in behavioral modification programs that use family members to effect therapeutic changes in family interaction.

We have described the results of involving siblings in the treatment programs of two families. Both positive results and problems ensue when siblings are involved actively in treatment programs. The programs described here are only a beginning effort to understand the nature of sibling relationships and maximize positive social interaction among family members. Much further research is needed to develop and refine sibling training techniques.

More research is also needed on the effect of involving siblings in the training program on the siblings themselves. Although it is generally known that both intellectually retarded and physically handicapped children are at risk for the development of behavior disorders (11, 12), it is less well known that the normal siblings of these children are also at significant risk for the development of behavior disorders. The mechanisms of this situation are not well known, but some investigators have suggested that the development of behavior disorders in normal siblings of handicapped children is due to the distortion of family relationships that occurs as a result of a handicapped child in the family (12, 13). If this is the case, involving siblings in training programs might help prevent the development of behavior problems in the siblings themselves. As demonstrated in one family discussed above, a positive aspect of involving siblings in the training program is the development of increased positive interactions between the sibling and the target child. If negative interaction between the sibling and the target child is one of the causes of behavior disorders in the sibling, we might expect increased positive interaction to prevent or ameliorate behavior problems of siblings.

We hope that this paper will stimulate other professionals to investigate methods for effective involvement of siblings in behavior change programs.

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LETTERS TO THE EDITOR

Death, Creativity, and Depression

SIR: In "The Madness of Art" (October 1975 issue), Professor Leon Edel beautifully described the phenomenon of chronic depression in creative individuals, which he feels is causally related to a preoccupation with death. While death has been the theme of many masterful aesthetic works and many artists do seem very concerned with this subject, I suggest that this interest can better be explained as a secondary elaboration, not the source of depression. If this were so, chronic frustration could be the basis for a prototypical psychodynamic formulation.

My hypothesis is based on one of the few "facts" about creativity upon which all students agree, Henri Poincaré's four stages of the creative process—preparation, incubation, illumination, and verification.

The creative artist always carries with him an awareness of those things he wishes to create. His first task is gathering background. The preparation then settles into appropriate (although sometimes unusual) perspective during the incubation period. This is necessary before illumination can take place. It is during the incubation phase, when he has achieved an appreciation of that which he wishes to create, that the artist faces great stress. He constantly refines his approach to the creative end; he reworks his preparation and receives partial rewards. He has a compelling need to repeat this process until it yields results. Realizing the potential of his desires, he values the striving process, but the overall effect is that he feels inadequate in his attempt to achieve illumination. The artist then accepts a resulting chronic depression.

When a single illumination does take place, it rewards the artist's choice of lifestyle. Even then, however, there are several other parallel means by which the artist perpetuates his depression. One of these is his heightened awareness of death. By virtue of a sensitive appreciation of the possible, the artist sees creative potential in coming to terms with death. This challenge becomes another frustration that can heighten depression. Thus death can be viewed as more "grist for the mill." It is the character and cognitive style that cause the frustration and depression.

TERRY E. PASSMAN, M.D.
New Orleans, La.

Rauwolfia Derivatives and Breast Cancer

SIR: Studies published in 1974 by investigators from the United States (1), Great Britain (2), and Finland (3) showed that the incidence of breast cancer in hypertensive women over 50 treated with rauwolfia derivatives is significantly higher than that found in parallel control series. These data suggested a causal link between rauwolfia and breast cancer. The results of a U.S. study published in 1975 (4) did not support this hypothesis.

The first three studies identified the problem and indicated the need for further research and for caution in the use of rauwolfia in women at risk for breast cancer. There are some important points to be clarified, such as dosage, time of exposure, and causal mechanisms (5). If the possibility of carcinogenic action is related to the prolactin release caused by rauwolfia, an important problem could arise for psychiatrists (6) since prolactin is known to increase in some people receiving phenothiazines.

The research division of the Hôpital St. Michel Archange in Quebec and the psychiatric department of Laval University in Quebec are preparing a special investigation to explore the hypothesized link between the use of reserpine and other neuroleptics and the incidence of breast cancer in women.

Some preliminary data from this study may be of interest. We have found that the percentage of women operated on for breast cancer during a 28-year period before the introduction of rauwolfia derivatives and other neuroleptics (1925–1953) was not significantly different from that found after the introduction of these drugs (1954–1974).

From 1954 to 1974, 53 women were operated on in the same hospital for breast cancer. Their mean age was 59 years (range, 31–90). For each case of breast cancer, a female patient from the same hospital matched for year of operation and for age (within 5 years) was selected as a control. The mean age in the control group was 59.4 years (range, 32–85). The control patients had been operated on for minor surgical complaints. The preoperative exposure of these two groups to reserpine was examined, and it was found that the breast cancer group had not been exposed to reserpine more often than the control group.

We also compared the incidence of breast cancer in two groups of 70 women each, one treated with reserpine and other neuroleptics and the other with different neuroleptics excluding reserpine. The mean age in the former group was 59.5 years (range, 30–82) and in the latter 60 years (range, 32–83). The length of reserpine treatment ranged from less than 6 months (N=30) to 9 years (N=3). Four women in each group were operated on for breast cancer. Thus the incidence of breast cancer in the two samples was the same. We should emphasize that some of the reserpine patients were treated with high doses (i.e., reserpine was used as an antipsychotic) and some were given reserpine as an additional drug prescribed for hypertension.

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A. Jus, M.D.
R. Pineau, M.S.
N. Hazzi, M.D.
Quebec, Que., Canada

"Primary" Prevention?

Sir: After a careful review of the excellent article "We Help Each Other: Primary Prevention for the Widowed" by William F. McCourt, M.D., and associates (January 1976 issue), I question the use of the term "primary." Wouldn't "secondary" and in some cases "tertiary" have been more appropriate, especially when it is stated that "We perceived the recently bereaved as being in a state of acute crisis"?

Incidentally, on page A7 of the same issue, I noted that there is no committee on preventive psychiatry and I wonder why this is so.

Vincent Lentini, M.D.
Colorado Springs, Colo.

On Jungian Psychology

Sir: I have noted a growing receptivity in psychiatry and among psychiatric residents in the last decade to Jung's theories. In order to determine whether this opinion is widely held in America, I submitted a detailed questionnaire to 357 directors of psychiatric residency programs and to 116 American members of the International Association of Analytical Psychologists; 48% of the former and 44.8% of the latter returned completed questionnaires.

Program directors stated that Jungian concepts were included in supervision of case material in 21% of the residencies. They also thought that psychiatric residents were receptive to Jungian analysis when first introduced to it; a mean of 6.5 was found on a scale from 1 (strongly reject) to 7 (strongly receptive). Program directors indicated that 23% of residents showed increased receptivity. The program directors saw increasing receptivity in 17% of psychiatrists and 20% of psychologists.

Jungian analysts were extremely optimistic about receptivity to Jungian psychology, believing there was a 96% increase in the past decade among university students, 63% among psychiatric residents, 57% among psychiatrists, and 72% among psychologists. Ninety-five percent of Jungian analysts favored greater participation in psychiatric training programs. While they perceived no change over the last decade in the receptivity of academic psychiatry to Jungian theory, they thought receptivity would increase during the next decade.

Jungian analysts ranked the concept of the collective unconscious and Jung's dream work as his most important contributions. They listed *Two Essays on Analytical Psychology* (1), *Memories, Dreams, Reflections* (2), and *Man and His Symbols* (3) as the three most important books to recommend to psychiatric residents.

There currently seems to be a zeitgeist for subjective inner world experiences as well as greater tolerance for a wide variety of therapies and a decreasing domination of psychoanalysis. There is also a greater awareness of the non-Western world, oriental religions, and the esoteric. It is my opinion (which the facts neither support nor refute) that there is a growing understanding of the practical value of Jungian psychology in psychotherapy, of dream analysis, and of having an alternative view of depth psychology. The evidence, to the extent that the opinions I have summarized are valid, suggests that there has been and will continue to be a growing influence of Jungian psychology in American psychiatry, psychotherapy, and analysis.

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Harry A. Wilmer, M.D., Ph.D.
San Antonio, Tex.

Editor's Note. Dr. Wilmer presented an expanded version of these comments as part of the "Centennial Tribute to C.G. Jung" at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Another Leaderless Group

Sir: As a member for over two years of a leaderless group that began while its members were psychiatric residents in various stages of training, I read with much interest "History and Analysis of a Leaderless Group of Professional Therapists" by Winslow Hunt, M.D., and Amnon Issacharoff, M.D. (November 1975 issue). The group I belonged to consisted of six men in two San Francisco training programs, who ranged in age from their late twenties to early thirties.

The core of the final group were residents in a small community mental health program that assigned trainees to catchment areas in different parts of the city; they consequently had little contact with one another. The residents met irregularly to discuss their assignments and alternatively to gripe or praise their relatively unstructured and unsupervised program. By the end of the first six months a group had formed, a regular meeting time had been designated, and a meeting place was selected. At the end of the first year, two members transferred to another program and a member from that program replaced one of the original members. New trainees were invited to participate, but by the second year the group had in effect closed.

The group was used for feedback about patients, supervisors, and coworkers as well as ourselves. All six of us were in analytically oriented psychotherapy. The tacit group commandment, "Thou shalt not leave treatment," was an important cohesive force. Our frustration, anger, resistance, ad-

vances, and retreats were elaborated in the group. Intragroup rivalry focused on the prominence of our analysts in the community. Not-so-subtle jokes were made about whether the "sicker" group members needed daily analysis or were receiving "supportive" biweekly psychotherapy and whether the "healthier" could undergo daily analysis or were independent enough to need only biweekly treatment.

Five of the six members were single, and the group served an important weekly socializing and friendship function. In the second year, as members became closer, dinners and brunches alternated with weekly meetings. However, the group was possessive of its members and we were both pleased and upset when members entered serious relationships.

As members finished residency training and became job seekers or military "obligated volunteers," the group suffered many shocks. Intragroup rivalry was apparent in regard to academic appointments, private practice partners, and acquisition of patients. Three of the original six members declared conscientious objector status and the ethics of entering or not entering military service were hotly debated. The group continues in its fourth year.

MAJ. MELVIN BLAUSTEIN, MC, USA
APO San Francisco

Regression and Borderline Patients

SIR: In "Psychotherapy of Borderline Patients: The Influence of Theory on Technique" (October 1975 issue), Henry J. Friedman, M.D., raised the issue of the usefulness of regression in the treatment of borderline patients. In a community mental health setting where we attempt to minimize state hospital referrals, we see a significant number of individuals referred for such treatment where the principal problem appears to be massive regression. Many of these patients have the characteristics of the borderline syndrome.

Dr. Friedman is to be congratulated for drawing attention to this issue. I would be most interested in a more extensive discussion of regression, encompassing the clinical situations where regression is likely and the therapeutic techniques that can effectively counteract or limit regression. Perhaps Dr. Friedman could provide further information or references on this topic.

JAMES SPENSLEY, M.D.
Sacramento, Calif.

Dr. Friedman Replies

SIR: One common theme that emerges from examination of regressive states in borderline patients, whether they occur during psychotherapy or in patients' personal lives, is an intense relationship with an individual or therapist who has failed to set realistic limits on the nature of the interaction, either therapeutic or personal.

In my review of histories of patients who have shown massive regression, there have been some recurrent themes. While an exhaustive summary is impossible due to space limitations, these include 1) an inexperienced therapist, 2) excessive reliance and emphasis on transference, not as a naturally occurring phenomenon but as something to be provoked and sought out, 3) a tendency to confuse the patient's illness and regressive tendencies with a total inability to cope, and 4) a therapist who struggles to work with a patient who has inadequate motivation to change.

Clinically, borderline patients tend to get lost in the transference so that they in essence forget the actual purpose of their treatment. One of the main therapeutic tasks related to this tendency is a need for the therapist to be active in keeping the realistic aspects of the doctor-patient relationship in clear view for the patient and for himself.

The goal of limit setting in the psychotherapy of borderline patients is not to prevent all regression. This, of course, would not be possible even if it were desirable. The aim is rather to keep the regression at a workable level that is compatible with constant efforts to develop insight. This is not as simple with borderline patients as it is with more healthily neurotic individuals who have greater degrees of ego strength.

Unfortunately, the false dichotomy between expressive psychotherapy and supportive psychotherapy has persisted. Neither is effective for borderline patients—"letting it all come out" in expressive psychotherapy tends to lead to disorganization in borderline patients, and strictly supportive treatment fails to lead to adequate insight and emotional growth. The answer to this difficult clinical problem posed by borderline disorders seems to me to lie in increased flexibility in the application of psychoanalytic understanding of the variability of ego states between neurotic and borderline patients. If he recognizes this difference, the experienced clinician will make specific adjustments in his therapeutic technique that will decrease the regressive pressure on the patient when it is essential to do so. An inventory of such crucial points would require an additional paper. However, the general principle of applying less stress to the patient in tense, difficult clinical situations will suffice to allow most treatments to continue without disruptive regression. This maneuver will strengthen the therapeutic alliance and allow for further insight-oriented work at a point of less stress in the patient's life or transference.

References on the limit-setting aspects of working with borderline patients are scarce (1, 2). The content of the regression has attracted far more interest in the literature. It is my impression, however, that in actual clinical work the principles of limit setting have been widely accepted because they do work and permit the outpatient treatment of patients who otherwise would require lengthy and debilitating hospitalizations.

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HENRY J. FRIEDMAN, M.D.
Boston, Mass.

Psychodynamics in Medical Education

SIR: I was much puzzled by "The Selection of a Psychiatric Curriculum for Medical Students: Results of a Survey" by Warner Johnson, M.D., and John Snibbe, Ph.D. (May 1975 issue). Among the 10 topics consistently ranked as most

important were suicide evaluation, the dying patient, psychophysiologic disorders, the doctor-patient relationship, drug and alcohol abuse, sexual problems, and differential diagnosis. I think it would be impossible to learn about these topics and develop clinical judgment and evaluative acumen without an understanding of the basic psychodynamic issues.

I realize that medical students, medical residents, and even psychiatric residents are shocked by the emergence of unconscious fantasies, and that they seek quick diagnosis and fast results. Nevertheless, psychoanalytic psychology should be taught in a fashion integrated with clinical teaching in small groups, so that both patients' and residents' resistances can be analyzed and understood. Medical training should not encourage a flight from unconscious material. Psychiatric educators must make a serious effort to teach psychodynamic principles that are meaningful and relevant to those patients actually being seen by the residents.

I am not at all sure that psychologists, general psychiatrists, or other physicians even know what psychodynamics is (the science of motivation and control), or that there is a dynamic, genetic, and adaptive balance of forces left over in the psyche from a primitive infantile mental life. This psychic trauma imposes countless distortions onto the current life of the individual, inhibits his ability to cope, and is obfuscated by many social crises, family illnesses, and much individual suffering.

HELEN STEIN, M.D.
Fresno, Calif.

Dr. Johnson Replies

SIR: We share Dr. Stein's concern about the findings of our survey. We feel it is important to respond to her letter because it probably represents the feelings of many dedicated teachers who find that course material important to them may be less valued or needed by others.

Unfortunately, psychiatric education in medical school has not been very well regarded by our medical colleagues. If Castelnuovo-Tedesco's findings (1) are representative of physicians' opinions about the quality and relevance of psychiatric training in medical school, then we educators must return to the proverbial drawing board. Indeed, it would be well for us to reevaluate totally our past teaching efforts and make greater use of modern educational principles—even if it means making dramatic changes in our instructional practices.

We believe that an important first step toward placing psychiatric teaching on a more objective footing is to define clearly the curriculum to be taught. Our study was meant to do just that. It was, in essence, a needs assessment survey determined by a consensus of many physicians and mental health professionals. This technique reduces the effect of individual biases, which can easily distort curriculum selection. Like Dr. Stein, we were somewhat surprised at the survey findings and their implications for psychiatric education. Nevertheless, the results of the study were quite clear about what should and should not be included in a psychiatric curriculum for today's medical students.

One can, of course, dismiss the study and insist that future presentations in psychiatry be modeled after those of the past or in accordance with personal preferences. In our opinion, to do so would be a disservice to our students and future colleagues. We cannot justify the assumption that any single system of theory or course work that is important for us is necessarily relevant for nonpsychiatric physicians. As educators, it is our task to prepare our students to meet *their* future practice needs. With our track record, we need all the help available in devising a useful curriculum. It is very likely that practicing physicians can educate us about what psychiatric knowledge and skills are needed most in their day-to-day practice of medicine.

Apparently, many of our teaching efforts have not proven useful, and formal courses in psychoanalytic theory and psychodynamics rank among them. It is important for us to review why these courses and others have not been well received. Dr. Stein suggests that the phenomenon is due to resistance or a "flight from the unconscious." Perhaps this is so, but we feel that antiquated teaching methods, inadequate preparation of students to deal with complicated theory, and lack of practical clinical relevance are other possible explanations.

We are currently investigating the problem from the medical student's perspective and hope to have more data available soon. Curriculum selection is a task of great importance. At the present time we feel that it can be best accomplished by consensus.

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WARNER JOHNSON, M.D.
Los Angeles, Calif.

A Medical Student's Experience with Akathisia

SIR: Because the unpleasant but treatable extrapyramidal side effects of antipsychotic drugs are not often experienced by medical personnel, and because one of these side effects, akathisia, is not infrequently misdiagnosed, I would like to describe an akathisia reaction I experienced after receiving 1 mg of haloperidol intramuscularly during a neuroendocrine research study.

At 8:00 p.m. I had an intravenous heparin block inserted in an arm vein, from which blood was withdrawn at intervals. At 9:00 p.m. I received 1 mg of haloperidol intramuscularly. I felt drowsy by 9:20. By 9:30 the drowsiness had dissipated and was replaced by a diffuse, slowly increasing anxiety. My uneasiness soon began to focus on the idea that I could not possibly sit still for the rest of the experiment. I imagined walking outside; the idea of walking was particularly attractive. I could not concentrate on what I had been reading. Contrary to my expectations, the anxiety was not relieved by the removal of the IV at 10:00 p.m. As soon as I could move, I found myself pacing up and down the lab, shaking and wringing my hands. Whenever I stopped moving, the anxiety increased. Performing the simple tasks of centrifuging and pipeting the blood samples was difficult, and it took maximum concentration to sit still long enough to complete the task. I imagined bicycling home rapidly—the thought of rapid motion was again appealing.

The reaction peaked at 10:30 p.m., about the time I finished with the samples. I walked very quickly to my bicycle, which brought some relief from the anxiety. At home, I walked rapidly several times around the apartment complex

and then ran up and down the long driveway. Once inside my apartment, I did a little jig, moving my arms and legs quickly. By midnight the intensity of the reaction was decreasing. To have something to do, I gladly washed a large pile of dishes that were not mine—normally not my favorite activity. I was able to get to sleep by 2:00 a.m., two hours past my usual bedtime.

Two features of this akathisia reaction impressed me. First, the intensity of the dysphoria was striking. With the possible exception of going on stage on an opening night, I cannot remember any feeling of anxiety so intense. Second, the sense of a foreign influence forcing me to move was dramatic. Long before I thought of akathisia, or even discovered that moving made me less anxious, I was pacing and wringing my hands. Had I been asked why I was behaving this way, I can imagine I would have looked at my hands and feet in amazement and responded, "I don't know."

I hope that my opportunity to experience and describe this most unpleasant extrapyramidal side effect will alert others to the necessity of its rapid detection and treatment. I believe that warning patients of the possibility of this reaction is particularly important. The sense of a foreign force driving one to move, with the concomitant anxiety, might be extremely disconcerting to an uninformed patient who is already struggling with psychosis.

KENNETH S. KENDLER
Palo Alto, Calif.

Depressive Disorder in Childhood

SIR: "Clinical Criteria for Psychiatric Diagnosis and *DSM-III*" by Robert Spitzer, M.D., and associates (November 1975 issue) reports an important step toward bringing psychiatric diagnosis into the mainstream of medical diagnosis. I would like to suggest that clinical diagnostic criteria also be applied to psychiatric diagnosis of prepubescent children. Modification of the *Research Diagnostic Criteria* (1) is necessary in order to apply the criteria to children. Several recent studies (2–4) have proposed criteria for diagnosing the affective disorders in prepubescent children. An example of the diagnostic category of "childhood depression" that could be used in *DSM-III* is shown below:

Depressive Disorder in Childhood

A. Both dysphoric mood and self-deprecatory ideation. The dysphoric mood includes hypersensitivity, crying spells, negativity, "difficult to please," and statements or appearance of sadness, loneliness, unhappiness, hopelessness, and/or pessimism. The self-deprecatory ideation includes beliefs of persecution, desire or attempts to run away or leave home, death wishes, suicidal thoughts or attempts, and feelings of being worthless, useless, "dumb," stupid, and/or ugly. Dysphoria and self-deprecation must be prominent and relatively persistent but not necessarily the most dominant symptom.

B. At least two of the following eight symptoms are required to have appeared as part of the episode.

1. Aggressive behavior (agitation).
2. Sleep disturbance.
3. Change in school performance, including frequent complaints from teachers of daydreaming, poor concentration, or poor memory, loss of usual work effort in school subjects, and loss of interest in nonacademic school activities.
4. Diminished socialization, to include decreased group participation or social withdrawal.
5. Change in attitude toward school, including school phobia.
6. Somatic complaints, including nonmigraine headaches, abdominal pain, or other somatic concerns.
7. Loss of usual energy (includes nonschool activities).
8. Unusual change in appetite or weight.

C. Dysphoria and self-deprecation must be a *discrete change* in the individual's usual behavior and must be present for longer than one month. Momentary shifts from normal to a state of dysphoria and self-deprecation (usually associated with acute turmoil) are excluded.

D. During the episode the individual must have impaired functioning socially, with family, at home, or in school because of symptomatology.

The use of diagnostic clinical criteria in *DSM-III* will help to standardize research in the diagnosis and treatment of childhood psychiatric disorders.

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ROGER A. BRUMBACK, M.D.
Bethesda, Md.

Dr. Spitzer Replies

SIR: We are delighted that Dr. Brumback considers the incorporation of specific clinical diagnostic criteria in *DSM-III* an important step in the development of our field. What amazes us is that we have yet to hear anyone raise the issue of the potential dangers of this approach! Perhaps we are uncomfortable about suggesting something that is apparently so devoid of controversy. As to Dr. Brumback's specific proposal, inclusion in *DSM-III* of "depressive disorder in childhood," the subcommittee of the Task Force on Nomenclature and Statistics that deals with disorders specific to childhood has given this matter considerable thought and is familiar with the references cited.

There is controversy regarding the presence of clinical depression in children and, if it exists, whether or not it is fundamentally different from depression in adults. We feel that whenever clinical depression is judged to be present in a child, use of either the adult major or minor depressive disorder categories should be sufficient. We are not convinced that depression in a young person is a fundamentally different disorder than it is in adults. (Actually, the criteria Dr. Brumback proposes are very similar to the criteria for minor depressive disorder in the *Research Diagnostic Criteria*.) This would be similar to the practice of using the adult diag-

nosis of phobic or obsessive-compulsive disorder when either condition appears in a child.

It may be that there is a specific form of depression that occurs only in children and that we are mistaken in not including such a category in *DSM-III*. Only research studies that employ operational criteria for defining diagnostic categories can answer this question.

ROBERT L. SPITZER, M.D.
New York, N.Y.

Was "Sociopathy" Ever a Diagnosis?

SIR: " 'Sociopathy' and Its Synonyms: Inappropriate Diagnoses in Child Psychiatry," by Dorothy Otnow Lewis, M.D., and David Balla, Ph.D. (July 1975 issue) contains the strangest logic I have ever seen in the *Journal*. The authors recommend abandonment of the diagnosis of sociopathy because it "has lost its value." It is strange indeed to see such a suggestion in the *Journal*, since as far as our Association is concerned, sociopathy is not and never has been a diagnosis. It has never been a synonym for antisocial personality, which *is* a diagnosis. I was particularly perplexed to read that "Established in the adult nomenclature, [which it never was] the diagnosis of sociopathy infiltrated the new section of Behavior Disorders of Childhood and Adolescence." Because I wrote that section and have never accepted the diagnosis of sociopathy nor permitted it to be used on my service, this statement leaves me at a loss.

The authors cited two cases of adolescents dismissed by hospitals as "sociopathic." As a result of this dismissal, the correct diagnosis was missed and treatment was not instituted. On the basis of these examples, Drs. Lewis and Balla wish to delete a section of our diagnostic manual.

I must heartily agree that diagnosis in this area is often regrettably sloppy. However, any experienced clinician has seen instances in which the diagnosis of mental retardation has been incorrectly made in a child with a sensory or language deficit. Would the authors suggest that we should delete the diagnosis of mental retardation from our diagnostic manual for this reason? The diagnosis of retardation may also result in failure to treat and may have adverse consequences for the life of the child so diagnosed. The fact is that we *need* the diagnosis of mental retardation. During my lifetime, the accepted term for this diagnosis has changed from feeblemindedness to mental deficiency to mental retardation, but the concept was and is a necessary one.

In the same way, we need to recognize a deficit in the capacity of some persons to relate with empathy to others. This deficit commonly results in a failure of socialization. This is the essence of the antisocial reaction in *DSM-I* (1) and the antisocial personality in *DSM-II* (2). It is *one* of the elements that contribute to instances of antisocial behavior and crime. Since this is a fundamental defect in character structure and has far-reaching and, at times, catastrophic consequences, we cannot ignore it.

An important cause of this failure of socialization—the lack of an opportunity to develop empathy with a parent person in early life—was recognized by Loretta Bender (3), who was cited by Drs. Lewis and Balla, although they do not seem to understand what she wrote. It results in a guiltless, shameless, and often hostile aggressive personality. Attempts to shape a suitable diagnostic category for this type of defect, the authors point out, have a long history.

The concept of "moral insanity," which they mention, was subject to attack on religious grounds. In the words of John Ordronaux, Professor of Medical Jurisprudence at Columbia University, "The only disease to which the moral nature is subject is sin." This concept was replaced by "psychopathic personality" and in *DSM-I* by antisocial reaction, which stressed the lack of empathy of these individuals, describing them as "maintaining no real loyalties to any person, group, or code."

"Sociopathy" does not appear in either *DSM-I* or *DSM-II*. "Sociopathic personality disturbance" appears in *DSM-I* as a heading that includes antisocial reaction, dyssocial reaction, sexual deviation, and addiction (both alcoholism and drug addiction). In *DSM-I* all homosexuality (which our Association has recently decided is not a mental illness) was classified as a sociopathic personality disturbance. If we accept Drs. Lewis and Balla's term, then before our Association voted, all homosexuals were sociopathic.

"Behavior disorders of childhood" in *DSM-II* arose not from any theoretical preconceptions, but empirically from the repeated experimental finding that the same six symptom clusters exist in populations in various clinics in both this country and Japan. Lewis and Balla will find reference to these studies in my book *Behavior Disorders of Childhood and Adolescence* (4). These are three behavior disorders that come within the range of what they call "sociopathy."

The "group delinquent reaction" listed in *DSM-II* is a pattern of peer group rebellion often associated with strong group loyalty. In such instances it is a *socialized* response within a rebellious group. Particularly vulnerable is the adolescent boy who developed the capacity for empathy with others under his mother's nurture in his early years, who comes from a large and impoverished family living in a disorganized delinquency area, and who lacks an adequate father figure.

Two patterns of unsocialized delinquency are listed. They represent the classical alternatives of fight and flight. The "unsocialized aggressive reaction" is a pattern of attack in a world seen as hostile and develops most typically in the child who has good muscles, who has been and feels rejected, who has been treated inconsistently but has also had some degree of overprotection or shielding from the authorities in his aggressive behavior, and who consequently has the courage to attack. The "runaway reaction" involves repetitive running away from home overnight or longer and is prone to develop with the more severely rejected child who feels utterly beaten down, has a very bad self-image, and seeks to run from his problems.

Contrary to the negative treatment attitudes that Drs. Lewis and Balla said their two cases met, these three behavior disorders are treatable and probably offer a more favorable prognosis in general than the two cases they cite as missed diagnoses. To be sure, they are not effectively treated with drugs or traditional psychotherapy, but in the structured environment of a good training school, group delinquents may be rewarding to work with. The unsocialized delinquents who lack the capacity for empathy with others must be worked with patiently in a structured situation in terms of their egocentric self-interest. At younger ages, unsocialized children typically respond to the structure, consistency, and behavior modification methods of a children's psychiatric ward. Adolescents may require the structure of a training school or a security hospital. The Robert F. Kennedy Youth Center in Morgantown, W. Va., successor to the old National Training School for Boys in Washington, D.C., has reported a good degree of success using a structured token

economy with unsocialized aggressive boys. I have also seen many good results of treatment at the Iowa Training School for Boys.

Let us not throw out the baby with the bathwater. It does no good to delete needed and appropriate diagnostic categories because they are misused.

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RICHARD L. JENKINS, M.D.
Iowa City, Iowa

Dr. Lewis Replies

SIR: I prefer at the present time not to reply to Dr. Jenkins's letter, since I am working on a book that will provide a more comprehensive statement.

I have, by the way, made it a point to discuss his letter and other issues about delinquency personally with Dr. Jenkins. He therefore knows that I have not ignored or avoided his views.

DOROTHY OTNOW LEWIS, M.D.
New Haven, Conn.

Focusing on Television

SIR: I would like to suggest that the constant shifting of visual frames in television shows is related to the hyperkinetic syndrome. Television has emerged as the single major cognitive experience during the developmental years of huge numbers of children. Apart from the vapid and violent content of the programs, there are incessant changes of camera and focus, so that the viewer's reference point shifts every few seconds. This technique literally programs a short attention span and probably accounts for the almost hypnotic attraction television has for many of us.

A child conditioned to experience the world through television is then placed in a classroom where he is expected to learn from a static visual frame of reference. I suggest that the hyperactive child is attempting to recapture the dynamic quality of the television screen by rapidly changing his perceptual orientation. I also wonder if it is not possible that amphetamines control this behavior by providing a subjective experience comparable to the fleeting worlds of television and hyperkinesis.

Research with large samples and matched controls could test the hypothesis that with whatever other preconditions there may be, the hyperkinetic syndrome results from excessive television viewing during the child's early development. If this were confirmed, we would have additional reason to limit or proscribe television. However, this would probably be no more effective than other public health exhortations. Another approach might be to prevail upon television producers to modify techniques so that their programming is less noxious in style, if not content.

MATTHEW P. DUMONT, M.D.
Belmont, Mass.

A Television Stereotype

SIR: As a respite from the constant perusal of the psychiatric literature, I like to watch television occasionally. Within a three-week period, I, like millions of Americans, was treated to the following morsels. On November 12, 1975, "Starsky and Hutch" tracked down a psychopathic murderer dressed in aluminum foil ("to keep out the cosmic rays"). On November 13, "Harry O" attempted to infiltrate a psychotherapy group to uncover the murderer of two people. On November 16, "Bronk" sought a deranged, senseless bomber. On November 30, "Bronk" trailed two unstable, impulsive, screaming female revolutionaries who had kidnapped a wealthy man's son; one of the women was a drug addict.

My purpose is not to assail violence in television programming. Rather, I wish to point out that all of these stories link crime and mental illness so closely that I am afraid television viewers in general will begin to make an absolute association between mental illness and criminal behavior.

My sample of programs may have been biased by chance network programming schedules, and the shows I have described may be the only ones that link crime and psychiatric illness this season.

I would not deny that some mentally ill people do commit bizarre and sensational crimes, but TV programs appear to be including such people in their scripts far out of proportion to their actual numbers. If so, then we in psychiatry should attempt to give a counterbalancing, or at least a mitigating, education to the public on this topic. Otherwise, serious misconceptions may develop that could prove (if they have not already done so) detrimental to the welfare and rehabilitation of our patients. Obviously, this would also undo the considerable effort expended over many years toward removing the stigma of mental illness. Perhaps this letter will alert other psychiatrists to the potentially serious consequences of this type of television programming.

DANIEL A. DANSAK, M.D.
Washington, D.C.

Correction

The correct title of Paul Briquet's book referred to in "The Semantics of Hysteria Continued" (Letter to the Editor, January 1976 issue) is *Traité Clinique et Thérapeutique de l'Hystérie*. The staff regrets this error.

BOOK REVIEWS

Mental Health and Law: A System in Transition. U.S. Department of Health, Education, and Welfare Publication 75-176, *by Alan A. Stone, M.D. Rockville, Md., National Institute of Mental Health (Washington, D.C., U.S. Government Printing Office, distributor), 1975, 266 pp., $2.65 (paper).*

Many private practice psychiatrists are only half aware of the attack on established methods of psychiatric functioning by new law cases and legislation designed to make commitment more difficult to obtain, to press for deinstitutionalization, to protect more fully the civil rights of mental patients, and to establish new patients' rights. Institutional psychiatrists are more sensitized to the fact that they are being challenged to justify their decisions and to prove that they provide enough of the right kind of treatment to patients who are capable of benefiting from treatment. They are being required to demonstrate that their predictions of treatability and dangerousness are accurate enough to merit the authority they wield.

Dr. Stone, Professor of Law and Psychiatry at Harvard University, is acutely aware of the tensions and turmoil that exist in the mental health system because of the changing relationship between law and psychiatry. The National Institute of Mental Health's Center for Studies of Crime and Delinquency commissioned Stone to write a monograph on the present state of the interaction of the legal and mental health systems to focus more attention on transition phenomena. The result is what the author and the sponsor call a monograph, but it does not have the narrow limits that are sometimes associated with that term; it is a book, a remarkably far-ranging and comprehensive book, on the current status of legal psychiatry.

Stone has been thoroughly immersed in the transition he describes. As a result of a longtime debate with Prof. Alan Dershowitz, his civil-liberty-minded colleague at Harvard Law School, he has had to think through the problems of criteria for commitment and the usefulness of commitment. (Stone and Dershowitz have not yet come to a meeting of the minds.) As chairman of APA's Commission on Judicial Action, Stone has been the chief voice in deciding in which legal controversies APA should intervene and on whose side. (Although I serve on the commission, I have tried not to let this bias my appraisal of the book.)

Stone describes a mental health system that operates not in isolation, as many psychiatrists are accustomed to think, but in relationship to the criminal justice system and the welfare system. He states,

> What has happened in the last two decades is that in the name of reform, the professionals within each of these social institutions have taken on the roles, functions, and goals of each other.

In the field of psychiatry, he says, it could be argued that inflated claims concerning technical services that are provided have led to a huge array of mental health technicians, few of whom in fact have significant technical skills. Rather, much of what they do is the providing of personal care, attempting to engraft a meaningful human relationship on what poses as a technical service.

Another important discussion in this book deals with standards in mental health care. Stone feels that the 1973 federal legislation authorizing Professional Standards Review Organizations (PSROs) will force psychiatry to come up with tangible standards that will guide the courts in dealing with the appropriateness of commitment and treatment. Thus, he feels, one of legal psychiatry's greatest problems will be solved.

Among the important topics the author deals with are dangerousness, civil commitment, the right to treatment, the right to refuse treatment, institutional labor, mental retardation, the juvenile system, the aging, and sexual psychopaths and defective delinquents ("quasi-criminals"). There is also an excellent and up-to-date discussion of criminal responsibility and competency to stand trial. The final chapter deals with the role of lawyers in the mental health system. Because of a time lag between the writing of the manuscript in 1974 and its publication in 1975, Stone has added an appendix to bring the reader up-to-date with the current activity in legal psychiatry. He states that in this 12-month period, "There have been new legislative enactments, hundreds of reported cases, thousands of publications, and innumerable changes in administration and policy."

The book maintains a middle ground, recognizing that a great deal of psychiatric authority is exerted in ways that are not useful and infringe on the individual as well as the reality of psychic suffering and the need for help that sometimes require a paternalistic approach.

On almost every page of this book there are such thought-provoking statements as the following:

> When there are no alternatives, the concept of voluntariness is a charade.
>
> Dangerousness, like beauty, is to some extent in the eye of the beholder.
>
> One factor [often overlooked but worth mentioning] is the medically debilitating effect of not providing due process safeguards.
>
> It is ironic that States who exercise the power of removing children from their parents because of neglect will expose children whom they accept responsibility for to even greater depths of neglect.

Stone undoubtedly knows that there is no way to force his fellow psychiatrists to pay attention to the relationship between law and psychiatry until an outside agency—the government, the patient advocacy bar, personal injury lawyers, or reforming legislators—makes the situation impossible to ignore. However, if he cannot force psychiatrists to face up to all of the obligations and responsibilities that go with the practice of psychiatry, he has made it easier for them to be-

come better informed and to think more. He has described problems, advanced a number of useful solutions, given his own opinions, and brought an appreciable measure of clarity to a scene that has been obscure.

It is important that this book be widely circulated and widely read.

JONAS ROBITSCHER, J.D., M.D.
Atlanta, Ga.

Too Much Anger, Too Many Tears: A Personal Triumph Over Psychiatry, *by Janet and Paul Gotkin. New York, N.Y., Quadrangle/New York Times Book Co., 1975, 395 pp., $10.95.*

The subtitle of this book reveals its content. This is a detailed account of a woman's experience as a psychiatric patient constructed retrospectively by the former patient and her husband. They acknowledge their bitterness and their intent to attack "a new ever-hungry monster of mental health care." Their audience is the lay public, and their target is psychiatry and psychiatrists.

The story is well written, although sometimes a bit tedious. It conveys the 10 years of intense pain that began for Janet Gotkin when, as a highly successful overachiever, she was unable to tolerate separation from home on entry to a prestigious women's college. She was aware of a profound resentment at attending a school she did not wish to attend; she felt that her life had been a charade aimed at pleasing her parents. Their hopes for her older brother had been completely dashed by innumerable battles that culminated in his flight from the home. All of her parents' expectations were then focused on Janet, who could neither say no nor rebel directly or indirectly.

To this point the story is clinically familiar. Janet's description of the pain she experienced during her year at college makes for compelling reading. She turned to the college counseling service for help; here we meet her first therapist: "Dr. Kurtzman was an ugly man . . . . fat, manicured fingers . . . my first and lasting impression was of a sloppy, sated, lewd old man." He was of no help to her, so she turned to Dr. Wyatt: "Perhaps if he had shown any real interest in me, I might have opened myself a little, . . . but he didn't seem very concerned about my silence or very interested in hearing me when I did speak."

She then took a medical leave of absence from school and was referred to Dr. Berman. He had a 15-minute interview with her and her parents together during which he told Janet's father that he would be away for two months but would see Janet when he got back. Two months later she came to see him. He said, "As you no doubt know, the treatment will be psychoanalysis. You will come five times each week, 50 minutes each session. I will give you a schedule of appointments today. Do you have any questions?" Terror stricken, she obeyed his order to lie on the couch and tell him whatever entered her mind; then she slashed her wrists in his washroom. He brought her parents in immediately, dismissed her as "most unresponsive and uncooperative," and sent her off to the first of many hospitalizations.

All of the psychiatrists Janet met thereafter, with only one dubious exception, are disposed of predictably in this book. There is the "shithead" who was more frightened of her than she was of him. There is "another dull-eyed resident"; there is "the psychiatric resident, a silly-looking young man"; and there is the ward psychiatrist, "a glum-looking man in a well-tailored suit."

Most of all there is Dr. Sternfeld. (All of the names of doctors and hospitals are fictitious in this book to preserve anonymity and, one might suspect, to avoid libel suits.) Dr. Sternfeld was the "eclectic psychiatrist" who, over a period of 8 years, loaded her with huge doses of every psychotropic drug available, prescribed innumerable ECTs, and, in desperation, suggested insulin coma but could not find a hospital that used it anymore. According to the patient, neither he nor she ever recognized that her relationship with him was a precise duplicate of her relationship with her parents. With him she was once more the "golden girl" who lived out his every aspiration while repressing the murderous rage arising out of her dimly felt awareness that she was sacrificing her understanding of her own autonomous, worthwhile self. Dr. Sternfeld cared about Janet and displayed remarkable devotion, persistence, and dedication. He knew about drugs and ECT and made a point of the personal touch, including holding her and suffering with her—like a parent.

The patient perceived Dr. Sternfeld as a controlling, omniscient parent who slowly but surely became increasingly helpless himself in the no-win battle with a daughter whose dependency and rage were more than she could tolerate. No wonder she repeatedly slashed her wrists (as a means of feeling alive) and overdosed, always arranging beforehand to be found out.

After 10 years of suffering, Janet finally took a huge dose of thioridazine, was comatose for 5 days, and emerged with a sense of joy at being alive. In fact, she felt reborn. It was as though she had to die in order to live—death was the ultimate cleansing that enabled her to make the separation from her parents and from her psychiatrist. She effected her own release and, understandably, accepted it as a mystery, a miracle of sorts, and as proof that she had never been ill but only made so by self-serving professionals. For 5 years now she has been entirely well, living the ordinary trying experience of everyday life with her husband, who struggled along with her during the last 5 years of her illness, and with her daughter, who was born after her recovery.

Now that she is well, Janet and Paul Gotkin's bitterness for all of the bad treatment she received takes two channels. One is the total denial that Janet was ever sick. They reason that sickness is something of the body and that to become sick of the mind results from the diagnosis and brainwashing of psychiatrists whose livelihood depends on making certain that patients accept psychiatrists' perverted view of humaneness and humanity. The second channel is the organization of a group called Mental Patients' Resistance, whose task is to "inform the public of the exploitative nature of institutional psychiatry."

This book joins a number of the same genre. Their value often consists in their bringing to the lay public an appreciation of the suffering of the mentally ill. They usually provide information that is to be found in detail only in the psychiatric and psychoanalytic literature and is therefore unknown to the public. It is all to a fine purpose that the public be informed of the agony of mental illness by one who is ready to open herself about her experience; the truth of pain needs no defense. However, the cause of pain requires much more than a self-serving condemnation of all of psychiatry. I suppose that it is possible in a world in which anything can happen that Janet had the misfortune to fall upon a selected group of the worst psychiatrists and the worst private and public mental hospitals. The psychiatrists, psychoanalysts, psychiatric residents, nurses, and aides that I have known

and worked with in all settings have been notable for their efforts to understand and help rather than dismiss and punish. I am not saying that Janet can be all wrong, but all of her possible helpers could not have been so malevolent as she describes them.

It is in Janet's interest to maintain the fiction that the whole cause of her misery lay outside of herself. By institutionalizing the enemy she can achieve even greater distance from herself. The fantasy of rebirth after an extremely serious suicide attempt is not unusual. It becomes, in this book, a remarkable demonstration of the survival value of externalization. It affords a way to make political capital at the expense of a profession and all of its helpers. Once more a large segment of the public is invited to malign psychiatry and its workers. It is too bad that the richness of the writing in this book is entirely in the service of a myth of nonillness made into illness by the cruelty, inhumanity, and avarice of all psychiatrists. Perhaps it is consonant with the times we live in that all difficulties are assigned to the enemy out there, thus disposing of individual responsibility.

JAMES MANN, M.D.
Waban, Mass.

Rypins' Medical Licensure Examinations: Topical Summaries and Questions, 12th ed., *by Arthur W. Wright, M.D., with the collaboration of a review panel. Philadelphia, Pa., J.B. Lippincott Co., 1975, 999 pp., $25.00.*

This old friend of worrisome pre-examination nights has now appeared in its 12th edition. It is a veteran of 40 years on the medical scene, and this edition has been refurbished and updated with four of the chapters rewritten by new authors.

Still divided into two broad areas, basic medical sciences and clinical sciences, the subject matters covered are those most frequently asked in the various licensing examinations. The examinations covered now are not only the various state board exams but also the national board exams and the more recent Federal Licensing Examination (FLEX). Foreign medical school graduates who are required to take the Examination Certifying Foreign Medical Graduates (ECFMG) will find the book particularly helpful. Sample questions are appended to each chapter.

This volume presents a compact, concise, and orderly survey of the several fields of medicine in which one is sure to be examined. Special emphasis is placed on up-to-date knowledge of cell structure and function based on electron micrograph studies, and investigations with the ultracentrifuge are to be found in several of the chapters. The chapter on psychiatry, titled "The Synopsis of the Scope, Goal, and Content of Psychiatry," is presented by Howard P. Rome and Gordon Moore of the Mayo Clinic.

There is a chapter on public health and community medicine, which seems particularly important in this day of community involvement and with the possibility of national health insurance in the offing. The chapter covers material that was not taught to older clinicians; it just might interest everyone.

One can get an idea of the general medical qualifying examinations by reading the first chapter of the book, which is authored by Dr. Wright. Types of examinations and the constitution of the various licensing bodies are discussed here. The FLEX is certainly new to older graduates; it was not introduced until 1968. In addition, examples of various types of questions, including multiple choice and essay, are to be found in this chapter.

In the entirely new segment on behavioral science, written by John Paul Brady and Fred and Mary Ann Strider, a whole host of subjects is considered: genetics; biochemical aspects of behavior; the life cycle of learning; society, man, and the physician; and theories of personality development comprise a partial list. All of these subjects reflect modern medical thinking and give evidence of its involvement in the broad spectrum of human events.

Although no one will require the reading of this book in order to pass medical specialty board examinations, anyone who contemplates taking any medical licensing examination would do well to keep it handy and assimilate what it has to offer. The work evokes a bit of nostalgia as we think back to what we specialists used to know about the various elements of general medicine and brings forth regret about what we have forgotten. Anyone interested in recalling things he was taught years ago and learning the new findings that have been added to medical lore will find this presentation most interesting. The fact that one can read it without the threat of an examination hanging over him will enable him to savor it even more.

F.J.B.

An MMPI Handbook, Vol. II: Research Applications, revised ed., *by W. Grant Dahlstrom, George Schlager Welsh, and Leona E. Dahlstrom. Minneapolis, Minn., University of Minnesota Press, 1975, 567 pp., $25.00.*

For those who enter into any of the numerous areas of research to which the Minnesota Multiphasic Personality Inventory (MMPI) may be applied, this book is the master key. It is the second part of a two-volume revision of *An MMPI Handbook: A Guide to Use in Clinical Practice and Research*, which was originally published in 1960. The first volume of this revision, titled *Clinical Interpretation*, gathered all of the important information about methods and procedures relevant to the interpretation of the test. This second volume is a data bank that contains all of the important knowledge pertaining to the research use of the MMPI that has accumulated since the publication of the original handbook.

This second volume, like the first, has the virtue of being useful to beginning MMPI students as well as to experts because it can save countless hours of searching the literature in an area one plans to investigate by providing complete bibliographies. It also introduces all of the important ideas about applying the test to a particular area of research. For sophisticated MMPI users this is a reference book.

The book is well organized and clearly details the important research uses of the MMPI, e.g., in recognizing emotional disturbances or in predicting the presence of some behavior or attribute of psychological functioning. It also compiles the recent data and ideas for applying the test to the investigation of medical problems and describes its applications to the system of criminal justice and to educational and vocational questions. The authors also review the important literature concerning studies in which the test has been incorporated into experimental investigations of personality and behavior. They discuss the major sources of variance in the test and describe its usefulness in treatment evaluation.

The great usefulness of the MMPI lies not in predicting the old nosology (as it was originally constructed to do). The test

is useful because of all that has been learned about it over the years. It is useful in the effort to recognize a variety of traits, qualities, and complex patterns of personality and behavior. The 10 clinical scales of the MMPI have become indices of numerous characteristics other than psychiatric diagnoses. Beyond the 10 clinical scales and 3 validity scales, which are described in the first volume, this second volume includes the items and directions for scoring the items for the 455 special scales that have been generated from the original item pool. These scales are indices of a variety of characteristics—from promiscuity to success in baseball and from headache proneness to pharisaic virtue.

Both volumes of *An MMPI Handbook* provide access to the full usefulness of the MMPI. They bring all of the important ideas into one publication. The second volume includes a bibliography of over 6,000 significant references to the MMPI through the end of 1973.

DAVID F. BERRY, PH.D.
Hartford, Conn.

Evaluating Treatment Environments: A Social Ecological Approach, *by Rudolf H. Moos. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1974, 377 pp., $15.95.*

This is a carefully developed, highly useful book that introduces the reader to an extensive collection of techniques for the evaluation of mental health treatment environments. The book and the technical approaches to evaluation it describes are the outgrowth of research and investigations carried out in a number of quite different kinds of inpatient and community settings in the United States and abroad.

Moos and his associates see themselves as following the tradition of such earlier social psychologists as Stanton and Schwartz, whose descriptive studies of inpatient treatment settings first pointed to a relationship between changes in caregiving ecology and changes in patient behavior. Moos has gone beyond the empirical observations of the social-behavioral scientist to design tools that will measure the often divergent points of view of the participants in the therapeutic process—both patients and staff.

How are treatment settings perceived by patients and how are they perceived by staff? What are the implications for effective treatment of various patterns of patient-staff perceptual dissonance and consonance with regard to the treatment environment? What are the implications for both the immediate treatment situation and for the patient's well-being after discharge from the hospital? How can the evaluation process itself be used as a tool to help caregivers make the necessary changes that will render their programs more effective? Moos suggests a process and gives examples of situations in which his group has successfully used evaluation feedback to produce constructive change and more effective clinical programs.

The book appears to be written primarily for the behavioral science researcher who is interested in studying what makes mental health treatment environments work so variably in different patient populations. However, it has something useful to say to other groups as well. The mental health administrator and program developer will find the approaches to evaluating the ecology of the treatment environments of practical help in establishing more effective treatment environments. Those concerned about questions of the cost effectiveness of various ways of organizing the treatment setting will find the book helpful in unraveling some of the professional mystification of milieu therapy and in developing guidelines or criteria for program operations.

In an increasingly cost-conscious age, the ecological approach implicit in this book suggests ways of using the powerful effects of treatment milieus by means that are more precise and systematic and that strengthen the art of treatment and clinical management with science.

JEROME A. COLLINS, M.D.
Lynn, Mass.

Psychotherapeutic Approaches to the Resistant Child, *by Richard A. Gardner, M.D. New York, N.Y., Jason Aronson, 1975, 377 pp., $15.00.*

As any psychiatric trainee knows, the learning of child psychiatry is an unsatisfactory business. The journals and texts are full of case descriptions and theory, but there is a striking lack of information on how to use the technology of child psychiatry.

One of the results of this defect is that the techniques of child psychiatry have very little generalizability to closely related fields, e.g., general psychiatry, pediatrics, education, and child welfare. Too many child psychiatric techniques have the unfortunate and autistic definition of "activities done by a child psychiatrist." The specialty remains arcane, and overworked child psychiatrists are distressed at the degree to which other professionals seem unwilling to work psychotherapeutically with children.

Among those who have attempted to fill in this defect, Richard Gardner has made a number of excellent contributions. His mutual storytelling technique, which makes use of videotape or a tape recorder, has been a highly practical therapeutic device, and his books for children and parents on divorce and minimal brain dysfunction have eased the therapeutic tasks of working with these problems. His most recent book, *Psychotherapeutic Approaches to the Resistant Child*, is no exception. It is particularly valuable for those who wish to acquire or expand child psychotherapeutic skills. This does not mean that the book is not useful for the experienced child psychiatrist—it is—but it will be of even greater interest for trainees, pediatricians, social workers, and others who wish to learn simple yet effective ways of communicating and working with school-age children.

One of the greater difficulties faced by the trainee and the inexperienced professional is to decide which aspect of a child's behavior to focus on. When is a game just a game and when is it symbolic of the child's problems? When is the aggression of toy soldiers indicative of intrapsychic or family conflict and when is it simply an expression of play?

One of Gardner's greatest contributions is his creation of simple and semi-structured games that provide a means of eliciting children's verbal fantasies. In his games the therapist has an opportunity to listen to fantasy material on a regular basis instead of irregularly, as in nonstructured play therapy. This provides a means of obtaining verbal content and, more importantly, a means of focusing the therapist's attention on process—the patterns of perceiving, processing, and coping that are essential to understanding the child's behavior and relationships at home, at school, and with peers.

From a theoretical perspective, Gardner's techniques are primarily oriented toward the psychology of the ego. Although his theory is broadly psychoanalytic, the techniques he has developed illuminate ego function with special clarity.

This book would have been even better if the underlying

theory had been explicated more fully. In particular, I was frequently reminded of Anna Freud's classic, *The Ego and the Mechanisms of Defense* (1). For child psychiatrists in particular, Gardner's book should be read with Anna Freud's. Gardner's extensive clinical examples provide excellent documentation of the defense mechanisms she describes, in particularly denial in fantasy and denial in word and act.

The next question for the inexperienced therapist is, What do you do with this information once you've got it? Gardner's contribution to this problem is one of the rules of his storytelling games—the therapist gets a chance to make up his own stories. This has a number of major practical implications. It gives the therapist a chance to select themes from the child's productions and to feed back his own understanding (or lack of it) of what the child has said. It also gives the therapist an opportunity to create modifications and outcomes that offer improved adaptive solutions for the child and gives the therapist noncritical ways of challenging the child's defenses. Throughout the clinical vignettes one can see Gardner challenging his patients' use of magical and omnipotent approaches to problem solving. In his stories he tells his patients that practice and effort are required to achieve desired goals, that trying is necessary to success, and that children who do not learn how to develop personal, social, and work skills are going to be unhappy and have problems.

Finally, the value of Gardner's ego psychology techniques is that they are fun. Children like the games, including seeing or hearing themselves on video or tape, and therapists like playing them. They offer the therapist expanded opportunities to be creative and to make use of his own unconscious, sublimations, and adaptive skills in a format that can be channeled and titrated according to the patient's needs. Thus the play and the work of child psychotherapy are linked together according to the level of the child's development and the therapist's skill and experience. Professional growth and development can occur as an integral part of psychotherapeutic practice.

In summary, this book, although short on theoretical linkages, is a major contribution to applied psychotherapeutic work with children. In my opinion, it should become a standard text for teaching technique for all of the child professions.

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WILLIAM M. BOLMAN, M.D.
Honolulu, Hawaii

Neurology of Infancy and Childhood, *edited by Sidney Carter, M.D., and Arnold P. Gold, M.D. New York, N.Y., Appleton-Century-Crofts (Prentice-Hall), 1974, 195 pp., no price listed.*

This monograph consists of a well-organized series of chapters by different authors dealing with all of the disease categories commonly involved in the nervous system during infancy and childhood. It is presumably designed to provide an easy and practical source of information for the clinician who is a nonspecialist in neurology and who wants guidance regarding clinical management of a particular problem. The monograph is successful in this regard. Each chapter is also strengthened by a well-organized list of references.

As is so often the case, one can perhaps fault this book on its lack of homogeneity as a result of multiple authorship, but this does not detract from its usefulness. It is therefore recommended for physicians in fields allied with neurology who have occasional contact with problems in this area, but it is not designed as a reference text for the neurologist or the neurosurgeon.

ARTHUR A. WARD, JR., M.D.
Seattle, Wash.

Female Sexuality and the Oedipus Complex, *by Humberto Nagera, M.D. New York, N.Y., Jason Aronson, 1975, 139 pp., $10.00.*

This addition to the burgeoning literature on female sexuality is flawed because it fails to integrate recent advances in our knowledge in this area and relies too heavily on ill-defined and questionable concepts. Dr. Nagera's schematization of the female Oedipus complex holds that, in contrast to boys, who proceed directly from the anal to the oedipal stage, girls experience a phallic-oedipal stage. Dr. Nagera fails to consider the evidence that both boys and girls experience a phallic stage and that this stage is associated in both boys and girls with a narcissistic investment in the entire body at a time when the genitals are becoming the dominant erotogenic zone.

According to Dr. Nagera, in her phallic-oedipal phase the girl experiences her clitoris as a penis and her mother as the object of her libidinal investment. This reverses in the oedipal phase, when the father is sought as the object and the mother is negatively invested. To accomplish this, the girl has to renounce their longings for the mother. As we know and Dr. Nagera confirms, the choice of object may vary and accounts for the constellations referred to as the positive, negative, and inverted oedipal complexes.

Variations in object choice as well as in the final outcome of the Oedipus complex and the female's sexuality and psychology (a distinction that is blurred by Dr. Nagera) are determined by the complex interaction of the biological strength of the girl's drives, the maturation of her ego, and the experience that influences her fantasies about genitalia and her attitudes toward activity, passivity, masculinity, and femininity.

Dr. Nagera's views of female sexuality are misleading because he fails to include observations that do not support his assumptions. His assertion that clitoral masturbation is phallic and that the clitoris has to be renounced if femininity is to be established is contradicted by evidence that the clitoris is an erotogenic zone that is only sometimes perceived as a substitute for a penis and that, rather than renouncing it, the female has to incorporate it into her sexual experience.

Dr. Nagera's equation of femininity with passivity and masochism is also very questionable. The normal female does not seek pain. Rather, she accepts the pain associated with some female functions such as defloration, menstruation, and parturition as the inevitable accompaniments of some of her experiences as a woman. To describe the behavior of women as passive is inaccurate. Homemaking and child care require a great deal of activity, as do many of the other things that women do. It is true that the sexual behavior of women is different from that of men, but this difference is more appropriately described as receptive than passive. Gratifying

intercourse is not an experience in which something is done to a woman; it is one in which she actively participates.

Dr. Nagera's case material illustrates some of the weaknesses of his theoretical formulations. The masochism of some of his patients is pathological and related to unresolved oral and anal issues. Although he acknowledges that their masochism is excessive, he states that a certain amount is necessary for normal femininity. The pathological aggresivitv of some of his patients is not determined by their unwillingness to renounce the pleasure of their clitoris but to their anger and hatred, which are related to many factors, including experiences in which men were overvalued and women were humiliated and demeaned.

We have a great deal to learn about the psychology of women and the marked tendency of societies throughout history to undervalue women (albeit ambivalently). To better understand these phenomena, we need more detailed observations of the influence of anatomical and biological factors on fantasies and other psychic representations related to maleness and femaleness and the interaction of these factors with social and economic forces. Dr. Nagera's contribution tends to retard rather than advance our knowledge because of his failure to critically examine some of the concepts he invokes.

REBECCA Z. SOLOMON, M.D.
Hartford, Conn.

Prometheus Revisited, *by William H. Sheldon, Ph.D., M.D. Cambridge, Mass., Schenkman Publishing Co., 1975, 248 pp., no price listed.*

Those familiar with the name of William Sheldon should be informed of the basic difference between his previous important studies on the human constitution in relation to clinical matters, such as *Varieties of Delinquent Youth* (1), and the present volume. Published almost 40 years ago as *Psychology and the Promethean Will*, this book is provocative, pointed, exciting at times, and frustrating. Written in an incisive, aphoristic style, it conveys the strong message of a physician who, disenchanted with today's state of civilization, temporarily renounces the role of the scientist to take over that of philosopher and theologian.

Underlying the many points brought up in the book is the main theme of Prometheus (i.e., forethought) versus his brother Epimetheus (i.e., afterthought)—the daring and radical versus the conservative and organizational. For Sheldon, the ultimate function of religion is to harmonize Promethean and Epimethean urges by attempting to integrate affect with cognition. Unfortunately, in his view, religion is hindered by the pitfalls of power, money, and sexual morality. Worst of all, the conflict between the Promethean and Epimethean trends contributes to the rise of what Sheldon terms "waster personalities," i.e., the many people affected by human disorientations who give up the use of their forebrain and become addicted to advertising and slogans. As a remedy to this rather gloomy situation, Sheldon advocates a fusion of religion and social psychiatry. He then proceeds to discuss the main tenets of five dimensions (or "panels," as he calls them) of feeling and awareness: 1) material relationships, 2) social interrelationships, 3) reproductive relationships, 4) need for a humanistic religion, and 5) aesthetic fulfillment. He then discusses clinical issues in regard to each of these panels. Throughout the book the criticism of psychoanalysis and of its founder is pervasive and stringent. At times the criticism is convincing, e.g., the attribution to the mind of an unconscious consciousness is a logical absurdity. At other times the criticism appears unfounded, for example, the author's blending of Freud and American psychoanalysts and his irony about women psychoanalysts. At the root of Sheldon's opposition to psychoanalysis may very well be his devotion to Jung, to whom he publicly acknowledges his debt and from whom he borrowed the concepts of persona and anima.

Sheldon's criticism of our present institutions is sometimes hard to accept, notably, his criticism of nursery schools, children's clinics, and child aid societies, all of which he finds guilty of producing "normal," socialized, extroverted children, i.e., potential wasters. Sheldon's comparison of his Promethean and Epimethean types with his typological models of endomorphic (viscerotonic), ectomorphic (cerebrotonic), and mesomorphic (somatotonic) are hard to follow, especially in view of the fact that most individuals are a mixture of these types.

On the other hand, there are appealing points in this book, such as the emphasis on the primeval character of the female in comparison with the male and Sheldon's statement that, in contrast to the modern tendency to push young people into mature roles, many children need more time to mature sexually and in other ways.

Has the author succeeded in his endeavor? Will he be able to convince the reader of his theses or will the effect mainly elicit nostalgia for things past? Whether he is quixotic or prophetic, Sheldon's candor in this book has appeal. Even if he is a voice in the desert, it is a voice that needs to be heard once in a while.

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GEORGE MORA, M.D.
Poughkeepsie, N.Y.

Psychiatric Aspects of Neurological Disease, *edited by D. Frank Benson, M.D., and Dietrich Blumer, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1975, 307 pp., $17.50.*

A basic tenet in nineteenth-century thinking was that the brain is the organ of the mind. This implied a special relationship between psychiatry and neurology and resulted in the hybrid specialty of neuropsychiatry. In the early part of the twentieth century this special relationship loosened; it was even denied. Emphasis shifted to the learned aspects of behavior and to interpersonal and social forces as determinants of aberrent behavior. In more recent years the tide has turned again as the result of a deeper and broader understanding of the neuronal and biochemical determinants of behavior. For example, certain neuroleptic drugs not only ameliorate schizophrenic psychosis but can permanently derange the metabolism of biogenic amines, leading to persistent dyskinesias; asymmetries of function in the cerebral hemispheres may result not only in dramatic disturbances of language function with left-hemisphere lesions but in more subtle and less well understood impairments in recognizing stimulus equivalence with lesions in the right hemisphere.

The editors of this volume offer a multi-authored review of clinical problems in the traditional borderlands of neurology and psychiatry. A brief listing of the topics will suggest the breadth of coverage to the potential reader. The volume opens with a brief but lucid review by Norman Geschwind that emphasizes why psychiatrists must know the neuroanatomy of behavior. Lipowski then proposes a reclassification of the so-called organic brain syndromes. Three chapters address themselves to the problems of diagnosis and treatment of dementia, and three other chapters address the difficult questions of defining the deficits in behavior with lesions of the frontal and temporal lobes and the much-debated but still poorly understood relationship between temporal lobe epilepsy and personality disorders. C. David Marsden and colleagues provide a lengthy and well-documented review of the problems of spontaneous and drug-induced movement disorders in psychotic patients. The succeeding chapter considers dementia and episodic affective disorders in Huntington's chorea.

This is a well-planned and well-executed volume that should be read by all residents in psychiatry as well as by psychiatrists preparing for Boards and those exposed to a broad spectrum of psychiatric disease. Neurologists, although more often exposed to the problems presented here, will likewise find a great deal of interest in the book.

I find only one major fault in this volume, and the publishers are to blame. For some reason that defies comprehension the table of contents lists the chapters but not their page numbers. The reader looking for a particular chapter must thumb through until, by chance, he eventually finds the opening pages. This bizarre decision surely merits the now-defunct Rowan and Martin Flying Finger of Fate Award.

DAVID D. DALY, M.D.
Dallas, Tex.

The Psychobiology of Depression, *edited by Joseph Mendels. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1975, 172 pp., $15.00.*

This book deals with an area of great importance to psychiatry, yet it leaves me with a feeling of disappointment. The disappointment is not a result of any fault in the book. Rather, it is caused by the honest reporting of hopes yet to be fulfilled.

Approximately 20 years ago two accidental observations of great value were made: reserpine could induce depression; and iproniazid, a monoamine oxidase inhibitor, seemed to bring on euphoria. These findings had far-reaching clinical implications. With the addition of the tricyclic antidepressants and lithium salts, an important step forward in the treatment of affective disorders was accomplished. Besides their clinical value, there were additional results of these seminal findings: an amine hypothesis was formulated to help conceptualize the biology, biopathology, and pharmacotherapy of affective disorders; and a large cadre of dedicated investigators focused their energies on testing, extending, and revising that hypothesis.

Dr. Mendels, one of the leading investigators of the hypothesis, edited this "state of the art" volume, which explores the testing of the amine hypothesis, the implications of it, its relationship to other biological issues, and problems of research. The book presents many fascinating issues. It deals with such new areas of investigation as postsynaptic events, alterations in cell membrane function, previously unstudied metabolites, and the role of neuroendocrines. The book also discusses the genetic implications of the work and evaluates such techniques of investigation as the study of red blood cell and platelet function and sophisticated clinical strategies. The many facets of a monumental research effort are revealed by the various authors.

What emerges from all this work? There is a multitude of interesting findings, but validation and significance are often lacking. A number of worthwhile results, however, are evident, e.g., patients with affective disorders can now be classified in terms of response or nonreponse to specific drugs. This finding not only has clinical value but also improves our understanding of the biological basis of affective disorders. The genetic part of the theory has also been developed. However, the research itself has not led to great clinical advances. Theoretically, the original amine hypothesis has not been confirmed. At best, it survives as a partial formulation of numerous complex factors.

Our knowledge of biological aspects of affective disorders has been enhanced by many bits of knowledge. Some of these may become cornerstones for significant advances. However, the work of the last 20 years has not led to an integrated theory, nor have striking new clinical strategies emerged. To quote Dr. Mendels, "Much of the work which has been done remains controversial and needs elaboration and expansion. New concepts must also be sought."

NORMAN TABACHNICK, M.D.
Sepulveda, Calif.

World History of Psychiatry, *edited by John G. Howells, M.D., D.P.M. New York, N.Y., Brunner/Mazel, 1975, 728 pp., $25.00.*

This volume presents a country-by-country summary of psychiatric history. Most of the contributors are academic or institutional leaders in the countries they report on, and there is a lot of inevitable repetition as well as some signs of national pride (e.g., the writers from many countries suggest that witch trials were more common in other countries than in their own). Recent events are generally regarded as the most propitious, even by the editor, who deplores the tendency. Understandably, the most outstanding chapters are the work of historians, notably Veith on the Far East, Simon on ancient Greece, Margetts on early Canada, Fischer-Homberger on Germany and Austria, and Mora on Italy. The bulk of the other chapters are little more than accounts of institutional and legal changes. Nevertheless, even the most summary chapters give a sense of psychiatric development as it has occurred in sharply different times and places, especially the fascinating chapters on Africa and the Arab countries.

Several impressions from this book strike me. There have been few fundamental discoveries in psychiatry, and the times of their introduction have varied considerably. Psychiatry appears to have been a development of the last 20 or 30 years in many countries. However, the order in which changes appear has not varied very much. For example, mental hospitals are established, then stabilized, then turned toward a therapeutic milieu, and then toward the community. General hospitals are gradually involved in psychiatric work, psychoanalysis is introduced, and then a sociological approach is taken.

Expansion in psychiatry is worldwide, but child psychiatry lags in almost every country. There is less dependence on German and English training and more on centers in North

America. Ironically, as the prestige of dynamic psychiatry falls in the United States it appears to be advancing almost everywhere else. It would be tragic if the United States should have less and less of what other countries seek here because we have come to resemble the German and English centers they leave behind.

Little good is written of psychoanalysis in these chapters, despite the general claim that it is continuing to grow in countries where it is not forbidden for religious or political reasons and the fact that it is occasionally recommended for therapists. Mental retardation receives the cursory attention one has come to expect from psychiatrists; its institutional aspects alone are taken up, and only briefly.

I believe that this is the first world history of psychiatry to be assembled. Indeed, it appears to be the first time the psychiatric histories of a number of countries have been reviewed at all. This is a volume well worth publication and well worth reworking every several years.

LESTON L. HAVENS, M.D.
Boston, Mass.

Humanizing the Psychiatric Hospital, *edited by Alexander Gralnick, M.D. New York, N.Y., Jason Aronson, 1975, 283 pp., $10.00.*

This volume is a collection of papers reflecting the program at High Point Hospital in Port Chester, N.Y. Sixteen of the 19 papers have been previously published. The institution is described as a psychoanalytically oriented hospital serving adults and adolescents, most of whom are chronic schizophrenic patients. The papers are grouped into three categories: clinical theory, clinical applications, and clinical research.

The first portion of the book is a very human account, largely in a personal style, of the attempts of Dr. Gralnick and his medical staff to build and develop a humane and clinically effective hospital in the private sector. In the second portion, there are several papers describing various clinical problems encountered in the hospital and the sincere and skillful efforts of the staff to deal with them. The research papers in the third part of the book include a frank appraisal of the results of the program, which are often disappointing, and an excellent study of the impact of staff attitudes on treatment effectiveness.

The theme of humanization that is intended to unify the book does not appear to be exhaustively addressed. Although there is no doubt as to the humane qualities of the professional staff, the book does not satisfactorily deliver on its implied promise to specify the unique manner in which this hospital designedly offsets the alienating and dehumanizing experiences that its patients have undergone in their troubled pasts. In fact, the book describes a highly unified staff functioning in a structured manner that may border on benevolent authoritarianism in dealing with patients.

The implication seems to be that the patient's experience in this humane, highly ordered "family" will serve as a significant corrective emotional experience to offset the psychotogenic effects of prior life outside of this hospital. Sadly enough, the follow-up studies candidly presented in the third portion of the book fail to convincingly support this thesis. Indeed, it is highly questionable that six months of hospitalization—the seldom-achieved optimal period cited in the book—can be expected to significantly reverse the noxious impact of the past lives of psychotic patients, even though family therapy, also emphasized in the book, is concurrently provided.

It is with regard to the place of inpatient hospitalization in the total necessary spectrum of therapeutic experiences that the volume is critically inadequate. Community psychiatry is depicted as a kind of enemy of the proprietary psychiatric hospital, although any responsible community psychiatry program regards the specialized psychiatric hospital as a valuable portion of the total spectrum of services, not an antedeluvian relic to be totally eliminated or replaced. The prime justification for community psychiatry seems to be the guarantee of an articulated array of clinical and associated services to the mentally ill. This is certainly a necessary improvement over the discontinuity of isolated institutions carrying out their programs, however humane and clinically sound, with little or no acknowledgment that vital services to the patient must go beyond what is carried on within the protective confines of the hospital.

Dr. Gralnick and his valiant staff need not fear the incursions of community psychiatry if their concern for humanization of patient care leads them to consider the patient's total therapeutic experience in the community and to link arms with community psychiatry rather than set themselves at odds with it.

HARRY R. BRICKMAN, M.D.
Los Angeles, Calif.

Progress in Community Mental Health, Vol. III, *edited by Leopold Bellak, M.D., and Harvey H. Barten, M.D. New York, N.Y., Brunner/Mazel, 1975, 332 pp., $15.00.*

The volume does an excellent job of reviewing the current state of community mental health theory and practice. As do all such compendiums, it varies in quality, but all of the contributions are useful.

The book is divided into four sections: The Changing Scene; Developments in Community Mental Health; Some Special Programs, and Neglected Populations; and Evaluation and Research. The chapter on "The Catchment Area Concept," by Gary L. Tischler and associates from the Department of Psychiatry and Epidemiology and Public Health and the Institution for Social and Policy Studies at Yale University, is an excellent discussion of a very controversial subject. The chapter by H. Richard Lamm on "Treating Long-Term Schizophrenic Patients in the Community" is a clearly written exposition that contains a great deal of hard-won clinical wisdom.

The most outstanding chapter in the book is that by Jack Zusman, "Program Evaluation and Quality Control in Mental Health Services." He says,

> The importance and universal usefulness of program evaluation efforts are so far among the great myths of mental health and other human service fields. . . . While huge sums of money have been spent to evaluate service programs, individual efforts have been widely scattered and often undertaken by individuals with relatively little preparation. Impact of these programs appears to be close to zero. . . .
>
> Few of the proponents of program evaluation have addressed the fundamental question of why program evaluation studies *should* have an effect on practice. In a world where financial and professional rewards go to the administrator of the big agency with good public rela-

tions, where the consumer is unable to judge the quality or value of the clinical service he gets, and where professional ethics are becoming progressively weaker, where is the force that can be expected to move an agency away from providing poor or useless service toward good and effective service? (pp. 233, 234)

From this highly skeptical beginning, Dr. Zusman goes on to discuss the various categories of evaluation activity, such as effort, system, structure, process, and outcome evaluations. He also examines evaluations as a political process and talks about undertaking an evaluation study from the administrator's point of view. He examines cost, administrative issues, and the evaluator's professional role.

Overall, this book can be recommended without reservation for both the advanced practitioner and the student of the community mental health undertaking in the United States.

H.G. WHITTINGTON, M.D.
Denver, Colo.

Mental Hospitals: A Study in Organizational Effectiveness, *by Clagett G. Smith and James A. King. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1975, 181 pp., no price listed.*

For many years mental hospitals have been the center of controversy, particularly over the question of whether or not they really help their patients. Many authors have written that mental hospitals do more harm than good. Despite the many publications on the subject, little solid research has been accomplished. However, the authors of this book present a systematic organizational study of the mental hospital.

The research operations were carried out over a five-year period. A total of 18 hospitals were selected to represent the national population as nearly as possible. The size of the hospitals varied from a small 275-bed hospital to two of the largest in the country (both had more than 11,000 patients).

Some of the areas covered in the authors' analysis include measurements of hospital effectiveness, work environment of the whole staff, communication and coordination, hospital goals, and decision-making processes. A very impressive aspect of the research is the fact that discharged patients were contacted and interviewed to assess their current mental status and community adjustment. By this method, some measure of how well hospitals were meeting the goal of successful rehabilitation was made. This aspect of research is all too frequently ignored when treatment goals are evaluated.

The authors found that such organizational factors as goals, structure, and administrative behavior did not have the direct effects they had originally expected. However, they state that with proper use of both organizational and administrative processes a truly therapeutic community can be realized.

This book has a message for all mental hospital workers—from the chief administrator to the ward personnel. Its findings should be made known to legislators as well because the authors' unmistakable message is that with adequate planning and funding, mental hospitals can be made to function efficiently for the benefit of the people they seek to help.

A major criticism I have of this book is that it is very difficult to read because of the tremendous amount of data presented. Many readers may feel so overwhelmed with the scientific material that they may not stay with the book to reach the conclusions. However, I would emphasize that if one expends the necessary time and effort, reading this book will be a rewarding experience.

GEORGE W. BARTHOLOW, M.D.
Omaha, Neb.

Collusion for Conformity, *by Andrew Slaby, M.D., M.P.H., and Laurence Tancredi, M.D., J.D. New York, N.Y., Jason Aronson, 1975, 203 pp., $10.00.*

This book sets out to analyze the role of nonconformity in the culture of the United States. The authors seek to examine the structure, nature, and rationale of the various institutions and mechanisms devised by society for controlling individual behavior. Slaby and Tancredi review the issues of privacy, drug use, sexual behavior among consenting adults, suicide, and euthanasia. In the final two chapters they discuss conduct affecting others and give their conclusions. They advise better societal control mechanisms for minimizing harmful behavior by the individual toward others as well as for allowing the individual the optimal degree of freedom for happiness, creativity, and social progress.

The authors take a utilitarian ethical position; shifts in society's attitudes toward drug use, sex, and euthanasia are interpreted in terms of socioeconomics. Thus society's current awareness of the need for zero population growth promotes tolerance for homosexuality, contraception, and abortion, which were all once regarded as seriously deviant behaviors subject to strict control by the church, psychiatry, or the law. They argue strongly for full disclosure of the real issues behind societal control of deviant behaviors.

> The critical point is that control of behavior, particularly that which can be considered victimless behavior, should be clearly for policy reasons rather than reasons that result in the distortion of the ends to be achieved by [such] social systems as psychiatry and morality. (p. 201)

Slaby and Tancredi also advocate what they call a "balancing test":

> What is really required at this time is a test for balancing the individual's rights of privacy and freedom against the harm or potential harm that such behavior would have to the society. Such a balancing should include objective evidence of the harm that might be induced by certain behaviors. (p. 70)

The authors do not define how such a test should be constructed, who should administer it, or how the results should be applied.

Their arguments tend to be weighted heavily toward the liberal position in their discussions of the right to die and the right to privacy. In pointing out the "tremendous social benefits" arising from "maximum privacy," for example, they appear to overlook completely the harm caused by such secret societies as the Ku Klux Klan and the Weathermen. In their conclusion about euthanasia they state,

> It might be expected that as we become tacitly aware of the needs of society that some modes of active euthanasia, such as administering a lethal dose to a patient who consents to euthanasia, or even possibly the elimi-

nation of grossly deformed children, might become societally condigned [sic]. (p. 174)

This sounds uncomfortably reminiscent of the rationalizations used by Nazi Germany to eliminate those judged unfit and unworthy.

The style of the writing is cumbersome due to the authors' preference for long sentences. Pope's admonitory couplet would be fitting here:

Words are like leaves; and where they most abound,
Much fruit of sense beneath is rarely found.

Besides the professorial sentences that weary the reader, the authors repeat throughout the book the hackneyed terms "in effect," "of course," "in fact," "by the same token," and "nonetheless." They also reveal carelessness when they say that "Thou shalt not kill" is the fifth Commandment (p. 156); it is the sixth. Finally, the publisher should fire the proofreader: there were obvious errors on pp. 40, 41, 44, 46, 50, 80, 90, 96, and 104, at which point I stopped looking.

FRED B. CHARATAN, M.D.
New Hyde Park, N.Y.

A Child Is Being Beaten: Violence Against Children, An American Tragedy, *by Naomi Feigelson Chase. New York, N.Y., Holt, Rinehart and Winston, 1975, 225 pp., $8.95.*

In considering child abuse in a clinical setting or a discussion of a book, one cannot avoid noting that feelings of such intensity are stirred up that both the helplessness and the wish to lash out at the institutions, systems, and individuals charged with the responsibility of protecting children are made understandable.

Ms. Chase, who is a free-lance writer, was alternately depressed, horrified, and fascinated by the idea of a book on this subject; she clearly experienced what almost all of us feel when confronted with this phenomenon. Her response is not atypical either: she lashes out at those charged with protecting children. Being human, readers are likely to feel the lashing is deserved until it comes to their own institution, discipline, or profession.

Chase considers the economic system to be responsible for the institutionalization of child abuse. She states that children are neglected as a matter of social policy "even if that policy is due to lack of resources." She is scornful of early reformers and of present-day individuals and agencies that work with these children and their stressed families. A newspaper that launched a sensational and somewhat hypocritical crusade after the death of a child does not fare any better in her opinion. Bungling courts, indifferent legislators, a foster-care system that does not afford a child the love and care that is needed, and overloaded social agencies offer no solutions. Chase also derides individual treatment because, in her view, the real problem is the social neglect of all children, particularly poor and minority children.

Chase draws conclusions from her study and makes recommendations for the provision of the following: jobs for all, because the joblessness of the father and his absence from home is an important cause of child abuse; compensation for women who work at home as a way of reversing the downgrading of the role of the mother; good child-care arrangements for women who work outside of the home; decent housing; income redistribution; free health care; education in infant care; family education; an expanded day-care system; family help centers; prohibition of corporal punishment in all schools and institutions; and free abortion on demand (despite the fact that she equates abortion with infanticide).

These recommendations are humane, but some of them would encounter insurmountable opposition. All of them come at a time of major retrenchment in publicly supported social, economic, and health programs. To what use can recommendations be put if they cannot be implemented? Furthermore, Chase is adamant that child abuse is a social problem; one wonders why she finds it so difficult to view it as both a social problem and a problem that occurs in individuals. This book presents us with the curious phenomenon of a very negative view of psychoanalysis from an author who repeatedly cites evidence from psychoanalysts and their works, not to mention that the title of the book is the title of one of Freud's most famous papers (1).

REFERENCE

1. Freud S: "A child is being beaten": a contribution to the study of the origin of sexual perversions (1919), in Complete Psychological Works, vol XVII. Translated and edited by Strachey J. London, Hogarth Press, 1955, pp 175–204

JACOB SWARTZ, M.D.
Boston, Mass.

Zen Meditation Therapy, *by Tomio Hirai, M.D. Tokyo, Japan, Japan Publications (Elmsford, N.Y., Japan Publications Trading Co., distributor), 1975, 103 pp., $3.25 (paper.)*

Psychophysiology of Zen, *by Tomio Hirai, M.D. Tokyo, Japan, Igaku Shoin, 1974, 141 pp., no price listed.*

These two small books complement each other. *Zen Meditation Therapy* is a rather low-pitched presentation of the neurophysiological and psychophysiological aspects of Zen. It supplies answers about Zen meditation in a Western scientific frame of reference. In an elementary manner, it covers many issues that may bore anyone who has already studied the beneficial effects of true mental and physical relaxation. It presents Zen (Zazen) as a therapeutic process rather than a religious experience and aims at providing knowledge of the techniques of reaching the ideal mental state of meditation. This ideal state is defined in terms of the type of brain wave activity, cardiac rate, respiratory rate, and galvanic skin response existing in this condition.

The reader will learn many new Japanese terms, such as "Hishiryo," the state of concentrating without thinking, the state of Zen enlightenment. Hishiryo is presented as the goal for the Western meditator. Our built-in cultural resistance to Zen because of its Eastern religious ties is approached in this brief book; it tries to make Zen more scientific and less religious and thus more acceptable to the Western reader. The book is easy to read and is recommended to the physician interested in exploring and/or developing a technique for applying relaxation and learned autonomic control therapeutically.

Psychophysiology of Zen is an attempt at a more scientific presentation of the subject, an effort to make it appealing to the Western scientific mind. Under such headings as "Physiology of Zen Meditation," "Results of Stimulation Experiments in Zen Meditation," and "Changes in Respiratory Functions During Zen Meditation," the author presents sum-

maries of research data accumulated. The very critical will find many questions unanswered in these chapters; the inquisitive will find many stimulating answers.

There is a chapter on "Zen Meditation and Feedback Training, or the Voluntary Control" that looks briefly at Zen meditation versus biofeedback. This chapter encapsulates the differences between the Eastern and Western approaches to relaxation and autonomic control as a therapeutic process: "The start of the study is different in East and West, but the results are the same, as is clear from our study and those of Biofeedback training or voluntary control." This book is recommended to those who have just begun to study biofeedback therapy.

PHILIP C. ROND, M.D.
Tallahassee, Fla.

Love and Ecstasy, *by Arthur D. Colman, M.D., and Libby Lee Colman, Ph.D. New York, N.Y., Continuum (Seabury Press), 1975, 200 pp., $8.95.*

This attractive little book is an essay about human closeness. Its focus is on the "merger felt by two people who are intimate together." Although the authors occasionally allude to sexual intimacy, this does not appear to be their major concern. Their thesis is that ecstasy is a special state of consciousness "in which the sense of one's 'I,' one's personal boundary, is lost through merger with something or someone else." The main message of the book is to suggest that ecstatic merger can best be experienced in two-person relationships. Psychopathological aspects of merger, as during psychosis, nightmares, or intoxication, are mentioned only in passing.

It is extraordinarily difficult to achieve a satisfying verbal definition of love, not to mention ecstasy. The Colmans—she is an English teacher and he is a psychiatrist—tackle this problem in the first two chapters, which deftly blend concepts from philosophy, neurophysiology, and mysticism.

Their central argument appears in the third chapter, titled "The Dyad in Ecstatic Tradition." Here they debate the risks and rewards of romance, religion, and other aspects of ecstatic feeling; quote Martin Buber and Kahlil Gibran; and raise more questions about love than they can hope to answer.

The fourth chapter presents a hypothesis about the infantile origin of ecstatic consciousness based partly on psychoanalytic theory and partly on the authors' own studies of the psychology of pregnancy. Some highly original and very poignantly expressed ideas about ecstasy in childhood, adolescence, and adult life follow in the next three chapters, and the essay closes with an optimistic redefinition of ecstasy as both a feeling and a quality of interpersonal relationship that is to be sought and treasured.

This book is not a scientific treatise aimed at satisfying readers who want to follow logical sequences toward a conclusion. Rather, one gets the sense of two people who are in love talking about what they know, believe, or want to be true. This creates an undulating style that is absolutely delightful at times but also runs the risk of fluctuating between dogma and obscurity. For example,

> We emerge from childhood into adolescence with the sense of an ecstatic past which, if we dare, can be reopened. We fall in love with visions, taste the bliss of orgasm, plumb the private depths of other minds. We nibble at the past and let portions of it become part of our future as well. Occasionally we find a person in whom everything can be combined, who heals our earliest and deepest wound, whom we can love. And then we can struggle and pray for the ecstasy of discovery to last long enough so that a social fabric can be developed which will allow the potential for ecstasy to continue and for our personal love tradition to develop.

An earnest and almost religious quality sets this book in contrast to the more flamboyant and even frankly pornographic publications about ecstatic experience that are available today. For this and other reasons I would have liked to see the Colmans' book produced in a less expensive paperback edition.

PETER F. OSTWALD, M.D.
San Francisco, Calif.

Transpersonal Psychologies, *edited by Charles T. Tart. New York, N.Y., Harper & Row, 1975, 485 pp., $16.50.*

This volume is one of a series of recent studies that reflect a burgeoning interest in the related problems of modifications of consciousness and meditative approaches to religious experience. The study of states of consciousness has long had its place in the range of psychological studies as a legitimate area of interest and investigation for scientific psychology. This book approaches this study from the point of view of the transpersonal (i.e., spiritual or religious) psychologies. The various authors attempt to articulate the attitudes toward religious experience and meditative techniques of a variety of religious traditions and to relate them to more familiar Western scientific categories. In my opinion the enterprise falls far short of success.

The study of consciousness and altered states of consciousness has been the focus of a number of scientific approaches. Neurophysiologists have studied the patterns of physiological functioning of nervous tissue in relation to states of consciousness and arousal. This approach has extended itself in more specialized studies of patterns of brain activity related to altered states of consciousness during sleep and dreaming. The alterations of cortical and reticular activity involved in states of arousal and attention have also been given considerable attention. Changes in the level and specificity of awareness in conditions in which specific areas of brain tissue have been pathologically affected have been a perduring interest of neurologists since the last century. The influence on the central nervous system of drugs that modify the level and the organization of conscious experience has also been an active and important area of scientific research.

The study of conscious experience and awareness has certainly not been foreign to the interests of psychiatrists and psychoanalysts. The study of consciousness and unconsciousness has been an immediate and pressing concern of psychoanalysts from the earliest days of psychoanalytic involvement. I am reminded that one of the special interests of the late David Rapaport—one of the most prestigious and influential of analytic thinkers—was precisely in the altered states of consciousness. Rapaport put considerable effort into the study of the organization of conscious processes, particularly in terms of their cognitive functioning. One of the central points of his work is an attempt to elaborate a theory of consciousness and conscious activity within the psychoanalytic framework. Rapaport himself undertook the empir-

ical observation of states of consciousness other than the alert waking state, particularly such states as automatic writing, daydreaming, hypnogogic reveries, and dreams. Out of this came his work on cognitive structures, which has enriched not only psychoanalysis but the study of human cognition in general.

From a broader perspective, interest in altered states of consciousness is close to the clinical concerns of every psychiatrist and psychoanalyst. In the work of the analyst the induction of an altered state of consciousness that allows for a perceptible shift in the organization of the patient's thought processes is part of the therapeutic regimen. Forms of regression and their influence on the formation of symptoms and on patient behavior is an area open to the experience of every clinical psychiatrist.

Another important area of consideration of altered states of consciousness relates to the recent attempts to implement and study the effects of meditation on forms of psychopathology and mental disequilibrium. In general, one can say that the outcome of such studies has indicated that there are indeed identifiable therapeutic benefits in meditative practice in terms of an inner calming, the alleviation of anxiety, the diminution of certain forms of defensive organization and response, alleviation of compulsive drug taking, and a number of other beneficial outcomes. However, the serious study of such phenomena and their utility in the overall therapeutic enterprise remains to be established and certainly deserves further attention by the psychiatric community.

Unfortunately, if one looks for information or help in any of these important areas from this book he will certainly be disappointed. The most helpful section is that by Dr. Tart himself, in which he points out the limitations of a restrictive scientific methodology and argues quite persuasively that the enterprise of understanding human consciousness and experience cannot be limited to the constraining scientific paradigms imposed by the Western scientific tradition. The problem here is that he seems to be beating a dead horse because even the behavioral sciences have grown out of the largely positivistic and deterministic phase of their scientific development. There seems to be an implicit assumption in the tenor of Dr. Tart's argument that if one can dispose of such positivistic, scientific canons, just about anything goes. There is no attempt here to grapple with the problem of what does go and how one approaches the problems of validation and theoretical understanding when dealing with transpersonal psychologies.

The rest of the chapters of the book are given over to accounts of the meditative experience within various religious traditions. The chapters vary considerably in quality, amount of information, and bias. The more general difficulty in these accounts is that they tend to present a relatively "theologized" account of their respective psychologies that addresses itself little if at all to the scientific interest and cognitive framework of those of us who are mired in the Western psychological tradition. Thus the explicit aim of the book—to translate the mystical traditions of religious orientations into the language of science—seems to me to be far from adequately achieved.

For an account of how these traditions approach the problem of understanding the meditative phenomena they inculcate, however, these accounts are fairly succinct and reasonably focused.

W.W. MEISSNER, S.J., M.D.
Cambridge, Mass.

This Month's Reviewers

JONAS ROBITSCHER, J.D., M.D., is Henry R. Luce Professor of Law and the Behavioral Sciences, Emory University School of Law, Atlanta, Ga.

JAMES MANN, M.D., is Professor of Psychiatry, Boston University School of Medicine, Boston, Mass.

DAVID F. BERRY, PH.D., is Staff Psychologist, Institute of Living, Hartford, Conn.

JEROME A. COLLINS, M.D., is Director, Greater Lynn Community Mental Health Program, Lynn, Mass., and Lecturer on Psychiatry, Laboratory of Community Psychiatry, Harvard Medical School, Boston, Mass.

WILLIAM M. BOLMAN, M.D., is Professor of Psychiatry and Pediatrics, University of Hawaii School of Medicine, Honolulu, Hawaii.

ARTHUR A. WARD, JR., M.D., is Professor and Chairman, Department of Neurological Surgery, University of Washington, Seattle, Wash.

REBECCA Z. SOLOMON, M.D., is Supervisor of Psychotherapy, Institute of Living, Hartford, Conn., and Supervising Analyst, Western New England Institute for Psychoanalysis, Woodbridge, Conn.

GEORGE MORA, M.D., is Medical Director, Astor Home and Clinics for Children, Rhinebeck, N.Y.

DAVID D. DALY, M.D., is Professor of Neurology, Southwestern Medical School, University of Texas Health Science Center, Dallas, Tex.

NORMAN TABACHNICK, M.D., is Staff Psychiatrist, Veterans Administration Hospital, Sepulveda, Calif., and Clinical Professor of Psychiatry, University of California, Los Angeles, Calif.

LESTON L. HAVENS, M.D., is Professor of Psychiatry, Massachusetts Mental Health Center, Harvard Medical School, Boston, Mass.

HARRY R. BRICKMAN, M.D., is Program Chief, Los Angeles County Mental Health Services, and Clinical Professor of Psychiatry, University of California, Los Angeles, and University of Southern California Medical Schools, Los Angeles, Calif.

H.G. WHITTINGTON, M.D., is Clinical Director, Malcolm X Center for Mental Health, Denver, Colo.

GEORGE W. BARTHOLOW, M.D., is Chief, Psychiatry Service, Veterans Administration Hospital, and Professor of Psychiatry, University of Nebraska College of Medicine, Omaha, Neb.

FRED B. CHARATAN, M.D., is Chief of Psychiatry, Jewish Institute for Geriatric Care, Long Island Jewish-Hillside Medical Center, New Hyde Park, N.Y.

JACOB SWARTZ, M.D., is Professor of Psychiatry, Boston University School of Medicine, and Training and Supervising Analyst, Boston Psychoanalytic Society and Institute, Boston, Mass.

PHILIP C. ROND, M.D., is Director, Division of Mental Health, Florida State University Health Service, Tallahassee, Fla.

PETER F. OSTWALD, M.D., is Professor of Psychiatry, University of California, San Francisco, School of Medicine, San Francisco, Calif.

W.W. MEISSNER, S.J., M.D., is Assistant Clinical Professor of Psychiatry, Harvard Medical School, and Chairman of the Faculty, Boston Psychoanalytic Institute, Boston, Mass.

Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Psychiatria Fennica, *edited by K.A. Achté. Helsinki, Finland, Psychiatria Fennica, 1975, 391 pp., 90 Finnish markkas (paper).*

Drinking Habits Among Alcoholics, Finnish Foundation for Alcohol Studies, Vol. 21, *by Salme Ahlström-Laakso. Helsinki, Finland, Finnish Foundation for Alcohol Studies (New Brunswick, N.J., Rutgers Center of Alcohol Studies, distributor), 1975, 216 pp., $8.00.*

How To Divorce Your Wife: The Man's Side of Divorce, *by Forden Athearn. Garden City, N.Y., Doubleday & Co., 1976, 160 pp., $6.95.*

Brain and Behavior: A Textbook of Physiological Psychology, *by Hugh Brown. New York, N.Y., Oxford University Press, 1976, 389 pp., $12.00.*

Sex After Sixty: A Guide for Men and Women for Their Later Years, *by Robert N. Butler, M.D., and Myrna I. Lewis, A.C.S.W. New York, N.Y., Harper & Row, 1976, 159 pp., $6.95.*

Annual Review of the Schizophrenic Syndrome, 1974–1975, *edited by Robert Cancro, M.D. New York, N.Y., Brunner/Mazel, 1976, 552 pp., $20.00.*

Nightmares and Hobbyhorses: Swift, Sterne, and Augustan Ideas of Madness, *by Michael V. DePorte. San Marino, Calif., Huntington Library, 1974, 160 pp., $10.00.*

Neurotransmitter Balances Regulating Behavior, *edited by Edward F. Domino and John M. Davis. Ann Arbor, Mich., NPP Books, 1975, 233 pp., $12.50.*

In Search of Love and Competence: Twenty-Five Years of Service, Training, and Research at the Reiss-Davis Child Study Center, *edited by Rudolf Ekstein, Ph.D. Los Angeles, Calif., Reiss-Davis Child Study Center (New York, N.Y., Brunner/Mazel, distributor), 1976, 361 pp., $12.50.*

Abnormal Psychology, *by Amerigo Farina. Englewood Cliffs, N.J., Prentice-Hall, 1976, 201 pp., no price listed (paper).*

Freud: The Fusion of Science and Humanism: The Intellectual History of Psychoanalysis. Psychological Issues Monograph 34/35, *edited by John E. Gedo and George H. Pollock. New York, N.Y., International Universities Press, 1976, 438 pp., $12.50 (paper).*

Positive Addiction, *by William Glasser, M.D. New York, N.Y., Harper & Row, 1976, 152 pp., $7.95.*

Review of Child Development Research, Vol. 5, *edited by E. Mavis Hetherington, John W. Hagen, Reuben Kron, and Aletha Huston Stein. Chicago, Ill., University of Chicago Press, 1975, 590 pp., $17.50.*

Personality Theory: The Personological Tradition, *by Robert Hogan. Englewood Cliffs, N.J., Prentice-Hall, 1976, 211 pp., $5.95 (paper).*

Only Human: Teenage Pregnancy and Parenthood, *by Marion Howard. New York, N.Y., Continuum (Seabury Press), 1975, 251 pp., $8.95.*

The Hippocampus, Vol. 2: Neurophysiology and Behavior, *edited by Robert L. Isaacson and Karl H. Pribram. New York, N.Y., Plenum Press, 1975, 441 pp., $24.50.*

Epilepsy After Non-Missile Head Injuries, 2nd ed., *by Bryan Jennett, M.D. Chicago, Ill., William Heinemann Medical Books (Year Book Medical Publishers, distributor), 1975, 176 pp., $20.00.*

Mental Handicap, *by Brian Kirman and Joan Bicknell. Edinburgh, Scotland, Churchill Livingstone (New York, N.Y., Longman, distributor), 1975, 473 pp., $39.50.*

Psychoanalytic Theory: An Exploration of Essentials, *by George S. Klein. New York, N.Y., International Universities Press, 1976, 321 pp., $17.50.*

The Family as Educator, *edited by Hope Jensen Leichter. New York, N.Y., Teachers College Press, 1976, 129 pp., $7.00.*

Society, Stress and Disease, Vol. 2: Childhood and Adolescence, *edited by Lennart Levi, M.D. New York, N.Y., Oxford University Press, 1975, 534 pp., $67.50.*

The Leader in the Group, *edited by Zanvel A. Liff, Ph.D. New York, N.Y., Jason Aronson, 1975, 315 pp., $15.00.*

Understanding and Living with Brain Damage, *by Patrick E. Logue, Ph.D. Springfield, Ill., Charles C Thomas, 1975, 104 pp., $8.50.*

The Nuremberg Mind: The Psychology of the Nazi Leaders, *by Florence R. Miale and Michael Selzer. New York, N.Y.,*

Quadrangle/New York Times Book Co., 1976, 302 pp., $10.95.

Drug Abuse and Social Issues: Clinical Aspects. Second International Symposium on Drug Abuse, Jerusalem, May 28–31, 1972, *edited by L. Miller and S. Einstein. New York, N.Y., S. Karger, 1975, 259 pp., $35.75 (paper).*

Introduction to Personality, 2nd ed., *by Walter Mischel. New York, N.Y., Holt, Rinehart and Winston, 1976, 552 pp., no price listed.*

Human Adaptation: Coping with Life Crises, *edited by Rudolf H. Moos. Lexington, Mass., D.C. Heath and Co., 1975, 438 pp., $5.95 (paper).*

The Human Context: Environmental Determinants of Behavior, *by Rudolf H. Moos. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 437 pp., $18.95.*

The Insanity Defense: A Blueprint for Legislative Reform, *by Grant H. Morris. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1975, 126 pp., $10.00.*

Controversial Issues in Human Relations Training Groups, *edited by Kenneth T. Morris, Ph.D., and Kenneth M. Cinnamon, Ph.D. Springfield, Ill., Charles C Thomas, 1975, 133 pp., $9.75.*

A.M.: How To Teach Yourself Meditation, *by Ted Nicholas. Wilmington, Del., Enterprise Publishing Co., 1975, 200 pp., $9.95.*

Alcohol in Colonial Africa. Finnish Foundation for Alcohol Studies, Vol. 22, *by Lynn Pan. Helsinki, Finland, Finnish Foundation for Alcohol Studies (New Brunswick, N.J., Rutgers Center of Alcoholic Studies, distributor), 1975, 121 pp., $5.50.*

Consultation-Liaison Psychiatry, *edited by Robert O. Pasnau, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1975, 304 pp., $16.00.*

The Psychology of Childhood and Adolescence, *by C.I. Sandström; translated by Albert Read. Baltimore, Md., Penguin Books, 1976, 252 pp., $3.25 (paper).*

Bereavement: Its Psychosocial Aspects, *edited by Bernard Schoenberg, Irwin Gerber, Alfred Wiener, Austin H. Kutscher, David Peretz, and Arthur C. Carr. New York, N.Y., Columbia University Press, 1975, 367 pp., $15.00.*

The Amazing Power of Hypnosis: What It Can Do for You, *by Wesley Shrader. Garden City, N.Y., Doubleday & Co., 1976, 189 pp., $7.95.*

The Practice of Pediatric Neurology, Vols. I and II, *edited by Kenneth F. Swaiman, M.D., and Francis S. Wright, M.D. St. Louis, Mo., C.V. Mosby Co., 1975, 1,082 pp., $119.50.*

Giambattista Vico's Science of Humanity, *edited by Giorgio Tagliacozzo and Donald Phillip Verene. Baltimore, Md., Johns Hopkins Press, 1976, 480 pp., $16.50.*

The Homosexual Matrix, *by C.A. Tripp. New York, N.Y., McGraw-Hill Book Co., 1975, 304 pp., $10.00.*

Primitive Internalized Object Relations: A Clinical Study of Schizophrenic, Borderline, and Narcissistic Patients, *by Vamik D. Volkan, M.D. New York, N.Y., International Universities Press, 1976, 333 pp., $13.50.*

The Home Birth Book, *by Charlotte and Fred Ward. Washington, D.C., INSCAPE Corp., 1976, 149 pp., $6.95 (paper).*

The Benzodiazepines—Patterns of Use: An Annotated Bibliography. Addiction Research Foundation Bibliographic Series 9, *compiled by C.E. Weise and S.F. Price. Toronto, Ont., Canada, Addiction Research Foundation, 1975, 197 pp., $7.00 (paper).*

Psychological Anthropology, *edited by Thomas R. Williams. The Hague, The Netherlands, Mouton (Chicago, Ill., Aldine Publishing Co., distributor), 1975, 636 pp., $27.50.*

Malnutrition and Brain Development, *by Myron Winick, M.D. New York, N.Y., Oxford University Press, 1976, 155 pp., $9.95.*

Life History Research in Psychopathology, Vol. 4, *edited by Robert D. Wirt, George Winokur, and Merrill Roff. Minneapolis, Minn., University of Minnesota Press, 1975, 354 pp., $19.50.*

OFFICIAL ACTIONS

Fall Committee Meetings

Following is a summary of the proceedings of the fall committee meetings of the American Psychiatric Association, which were held in Washington, D.C., in October 1975.

The Council on Medical Education and Career Development

RICHARD I. SHADER, M.D., CHAIRPERSON

THE COUNCIL met in March, May, and October 1975. Two new members of the Council, Drs. Paul Fink and Nancy Roeske, were present at the fall meeting.

The Council and its components actively pursued the development, clarification, and implementation of guidelines for continuing education, now a requirement for maintenance of active membership in the Association. A modification of the American Medical Association's guidelines has seemed a desirable format. This would permit the necessary degree of overlap between APA requirements and those of other bodies (e.g., AMA, state medical societies and relicensure authorities, and district branches). The preliminary draft of the guidelines was reviewed and amended to extend Category I coverage to participation in approved educational programs of the Canadian Psychiatric Association. It was also decided that it was essential to have a procedure for resolving disputes over program approval that might occur between individuals and district branches or district branches and area councils. The Council voted that if such disputes occur they will be settled by the Office of Education of APA, with the advice of the Committee on Continuing Education.

The Council decided that an Office of Education would be essential to the furtherance of the educational goals of APA. A new director having the status of a deputy medical director within APA is being sought.

Considerable discussion was focused on the future of certification in administrative psychiatry by APA; the Council recognizes and affirms the value of such certification. The Council again reaffirmed its understanding that APA is not a certifying organization. Therefore, the Council voted again to have the Board of Trustees assist in efforts to have this certifying function taken over by the American Board of Psychiatry and Neurology. The Council deferred action on the request of the Commission on Certification in Administrative Psychiatry to award honorary certificates until the commission submits criteria for such certificates and an outline of the selection process. The remainder of the commission's report was received with thanks.

The *Commission on History*, David Musto, M.D., chairperson, has withdrawn its request for additional funds for the preparation of a history syllabus by Dr. Daniel Blain. It was decided that commission members will assist in the development of relevant history questions that will be made available to the American Board of Psychiatry and Neurology and to the psychiatric self-assessment examination to incorporate into their examinations as appropriate.

The *Committee on Medical Education*, Larry Silver, M.D., chairperson, requested formation of a task force on medical student mental health services. It is felt that comprehensive medical student mental health services should be provided at each medical school and should include the availability of psychiatric services for students as well as supportive counseling, preventive counseling, and advice to students relating to problems or crises within their family or extended family. The Council voted that a task force be established to explore 1) which medical schools provide such services and to what extent; 2) who provides such services when they are available and whether they are a part of the Dean's Office or of the Department of Psychiatry; 3) who finances the salaries and operational expenses for such services; 4) whether guidelines are established relating to confidentiality and responsibility; and 5) where students go for such services when there are none provided by the medical school. Since these issues also relate to the Council on Mental Health Services, it was further proposed that this task force be a joint effort; the committee and the Council on Mental Health Services would each select and finance two

members for the task force. Liaison with the Task Force on Suicide Prevention (Council on Mental Health Services) was recommended.

The *Committee on Continuing Education*, Robert A. Senescu, M.D., chairperson, reported that it is premature to further specify content areas for Category I credits in the guidelines for continuing education; the council agreed.

The Council reviewed a proposal for a colloquium, entitled "How to Teach About Handling Psychiatric Emergencies," to be held July 22–25, 1976, and endorsed the topic and format. The Council felt strongly, however, that the colloquium should be conducted only if this can be done at no additional expense to APA; the Council supports staff time and travel. The subcommittee requested and received approval from the Program Committee for space and time on the program of the annual meeting to present a forum on continuing education for psychiatrists who teach in family medicine residency programs. There will be a formal presentation followed by small group activities focusing on curriculum planning, use of resources, content, methodology, and evaluation. Participants may choose the small group activity in which they wish to work.

The *Subcommittee on Education of Psychiatrists*, Donald Naftulin, M.D., chairperson, requested approval for a one-day conference to be held at the annual meeting in Miami, Fla., on Sunday, May 9, prior to the beginning of the meeting. The conference will consist of simulations in continuing education accreditation, discussion of program substance, and methods of evaluating educational needs. The afternoon will consist of program substance, subdividing into area meetings in which the district branch continuing education chairperson can develop area continuing education committees and discuss needs, areas, and accreditation processes. The Council endorsed this meeting, but only if there were no additional expense to APA.

The Council did not approve the current version of a position statement on foreign medical graduates prepared by the *Committee on Graduate Education*, Bryce Templeton, M.D., chairperson, and instructed the committee to work with the *Subcommittee on Foreign Medical Graduates*, Tsung-Yi Lin, M.D., chairperson, to improve this document. The Council approved the committee's recommendation that APA give high priority and provide staff support to the development of a research proposal to define the goals and objectives of psychiatric residency training. The concerns about the diagnosis and treatment of alcoholism and drug abuse that were expressed in the recent report of the Speaker-Elect should be included in this review. The Council discussed the importance of neurology training during the four years of psychiatric training. Fiscal difficulties were considered. The Council reaffirmed the Board- and Assembly-endorsed principle of flexibility in the opportunities for training in neurology and in the clinical care of medically ill patients and recommended that APA request that the American Board of Psychiatry and Neurology create a firm policy that will increase the potential for reciprocity in training for psychiatrists and neurologists.

Acting on the recommendation of the Committee on Graduate Education that the report of the *Task Force on Research Training*, Frederic G. Worden, M.D., chairperson, not be published in its present form, the Council charged the committee to review the report in more detail and to make further recommendations to the Council at a later date.

The plans for a meeting in Kansas City, Mo., to be held by the American Board of Psychiatry and Neurology and attended by Drs. Shader, Senescu, and A. Freedman and representatives of ABPN, were discussed. The purpose of this meeting will be to initiate multiorganization discussion of certification and recertification. The Council concluded that a task force on recertification should be established to ensure that a thoughtful, forward looking, and balanced view toward recertification can be developed. In appointing this task force, the Council will select some of its own members, consultants, and members of components who have already been working hard on this matter.

The Council responded to a request from the Board of Trustees to develop a method of selection and procedures for nomination of APA representatives to the ABPN. The Council recommended that APA procedures 1) request that the ABPN develop qualifications for the position of director on the Board; 2) adopt these qualifications as criteria to be followed in selecting APA nominees; 3) have the Medical Director develop a list of potential nominees and then submit to the Board of Trustees a minimum of 10 names of individuals who meet the accepted qualifications; and 5) have the Board of Trustees establish a rank order for the nominees by written ballot. It was also recommended that the steps of this process be published annually in *Psychiatric News* and include adequate information about the size and composition (APA, AMA, ANA) of the Board, aggregate data about the sex and race of the directors of the Board, the role of AMA in the process, Board procedures once nominations are received, information about reappointment and length of service on the Board, and a list of present members of the Board including brief biographical data. The Council feels that such an open process would more closely meet the various professional interests of our membership.

The Council thanked Dr. Paul Fink for sharing copies of three recent papers on psychiatry as a primary care specialty. His reports were helpful in focusing the deliberations of the Council on this vital matter. The issues involved in defining psychiatry's relationship to primary care are many and complex and are defined in part by the contexts in which the discussion occurs and in which the primary care is provided. The Council believes that the definition of primary care, which is not fixed at this time, will evolve to better approximate the nation's health care needs and realities.

In preparing testimony in the Senate for the proposed HR 5545, the Council recommended that APA take the following positions.

1. Psychiatry is a primary care specialty, and psychiatrists meet all six criteria of the Association of American Medical Colleges for the primary care physician.

2. With public education and increased availability and acceptability of psychiatric treatment, the public demand for psychiatric services has risen; therefore, an irreducible minimum of residency slots (probably not less than 10%) should be allocated to psychiatry. The planned reduction in the number of foreign medical graduates will probably result in a serious shortage of psychiatrists (the field of psychiatry has had an unusually high proportion of foreign medical graduates).

3. There will be increased need for the teaching of psychiatry to physicians in training and for continuing education in psychiatry in the other primary care specialties.

The Council approved the ABPN statement that individuals who from now on receive training in British programs that meet the rules of American training facilities will receive full credit so they can become eligible to take Board exams.

The Council reviewed the important role of audio and audiovisual materials in the continuing education of the membership. A major source of such materials is the APA annual meeting and other appropriate meetings and functions. In or-

der for APA to develop mechanisms for editorial control of these materials once the economic and proprietary features of this issue are resolved by the Committee on Public Information (Council on Internal Organization), the Council recommended that the Committee on Continuing Education work in liaison with the Committee on Public Information to initiate the development of appropriate policies.

After reviewing the letter of July 22, 1975, to Dr. Marmor from Elaine Knutsen, M.D., consultant to the Task Force on Suicide Prevention (Council on Mental Health Services), regarding the mental health needs of the members of the psychiatric profession and their families, the Council recommended that any further study generated by the task force be referred for action to the Council on Mental Health Services and that liaison be established on such projects between the Council on Mental Health Services and the Committees on Graduate Education and Continuing Education of this Council.

The Council reviewed the importance of the annual meeting in the continuing education efforts of the Association. It was felt that the priorities of the Program Committee have not always been consistent with those of the components of this Council; it was therefore recommended that Dr. Shader discuss with the chairperson of the Program Committee ways to fulfill the mandates of the components of this Council through efficient use of time at the annual meeting.

The Council plans two luncheon meetings during the APA annual meeting in Miami, Fla.

The Council on Mental Health Services

JAMES C. JOHNSON, M.D., CHAIRPERSON

DURING THE YEAR, several task forces completed their work and were discharged with thanks.

The *Commission on Childhood and Adolescence*, Viola Bernard, M.D., chairperson, concentrated primarily on the formation of a new Council on Children, Adolescents, and Their Families. They have developed a charge for this council as well as suggestions for several task forces. Upon recommendation of the Council and the Assembly, the Board of Trustees approved establishment of the new council, to begin in April 1976.

The commission has also maintained collaborative efforts with relevant APA components. It has been intimately involved with the Commission on Judicial Action in its approach to various suits involving child abuse and children's rights.

The *Committee on Financing of Mental Health Care*, William H. Goldman, M.D., chairperson, circulated a draft of its report to the Assembly, to the Board of Trustees, and to appropriate others in July, with feedback to the committee to come via the Assembly through its district branches, area councils, and the Assembly Task Force on Delivery of Mental Health Services. Dr. Goldman discussed the report with the Assembly at its November meeting. When the committee met November 30 with representatives of the Assembly Task Force on Mental Health Services to review the July 1975 draft report, two main issues on which there were differences were clarified. A compromise on these issues was reached through an agreement to further edit section two, "Principles of Psychiatric Coverage Under National Health Insurance," and section three, "Recommended Benefits," so that they would reflect more closely the Assembly task force's position. A general statement on peer review was added to section three. The revised sections were approved in principle by the Board of Trustees in December and referred to the Assembly Executive Committee for approval in February.

It is envisioned that the final report will be published in two separate parts: a relatively brief brochure including the historical background together with sections two and three, and a separate appendix including the supporting data and references.

The Council reviewed the reports of the June and October meetings of the *Committee on Peer Review*, Donald Langsley, M.D., chairperson; the committee also met in February 1976.

The committee proposed several alternatives concerning the establishment of an Office of Peer Review, seen as high priority by both the Council and the committee. The Assembly endorsed and the Board of Trustees approved implementation of one of these alternatives in which one member of the Peer Review Committee will visit the district branches during the next two years to help develop an effective peer review mechanism in each district branch. The committee has identified the chairpersons of components dealing with peer review or PSRO in all of the district branches, established an informational network by soliciting information about operations at the local level, and circulated the minutes of the national committee and other timely information of interest to these chairpersons.

Additional work has been done on model criteria for inpatient services, and criteria for outpatient services are being developed. The committee has developed a three-tiered system of concurrent review with explanatory narratives. It is considering development of a manual on peer review that would be useful to the district branches.

The committee has had constructive meetings with Blue Cross in California and has been involved in a Blue Cross Federal Employees Benefit-National Institute of Mental Health project headed by Dr. Steven Sharfstein. It is hoped that this project will provide additional data on patterns of utilization and cost of mental health services.

Dr. Langsley chairs a new Select Committee on Psychiat-

ric Services Evaluation (SCOPSE-II), a projected NIMH-Department of Defense study of schizophrenics in six metropolitan areas that will use the peer review concurrent review approach.

The *Committee on Federal Government Health Services,* Stewart Baker, M.D., chairperson, discussed the potential impact of the Privacy Act, which became effective in September 1975. The Federal Aviation Administration leans heavily on the quality of information derived directly from the private medical sector and is concerned that this quality will be adversely affected in the near future by the risk of disclosure of medical information to the patient on demand. FAA feels it has an ethical obligation to quickly alert the private medical sector to this new system of contingencies and that the ultimate impact of this act on FAA operations will be to lessen the quality of medical information obtained. The Council endorsed the committee's recommendation that this matter be referred to the Joint Commission on Government Operations for opinions and procedural recommendations.

There has been a shift in personnel planning by the Army that will double the number of women in uniform while decreasing the size of the Army. This new program includes increased emphasis on pregnancy prevention, rape prevention, improved identification of appropriate job skills for women, improving and modifying the stereotype of women in uniform, and other special considerations. The Council endorsed the recommendation of the committee that this question be referred to the Committee on Women for their review and opinions.

The *Task Force on Private Psychiatric Hospitals*, John H. Houck, M.D., chairperson, has completed its work and submitted a final manuscript and was discharged with thanks. This scholarly and informative report not only elucidates the present status of private psychiatric hospitals in the delivery system but also highlights remaining issues to be investigated. The tentative conclusions were that the private psychiatric hospital accounts for 6% of the total patient care episodes in the United States; this is a fairly consistent figure. The population profile served by the private psychiatric hospital is different in important aspects from that of the population served by the public mental hospital. This reflects the persistence of a two-tiered system of care for mentally ill patients requiring hospital services. These differences in care are unresolved issues. Patterns of delivery of care are shaped by mechanisms of payment for such services, but there is no presently available system of evaluation. Further attention to these unresolved areas will be a continuing concern of this Council.

The Council recommended, and the Board of Trustees approved, publication of the report of this task force along with the reports of the Task Force to Study the Status of Psychiatric Services in Nursing Homes and Other Extended Care Facilities and of a previous Task Force on Public Mental Hospitals.

The *Task Force to Study the Status of Psychiatric Services in Nursing Homes and Other Extended Care Facilities*, Seymour J. Veiner, M.D., chairperson, completed its work and was discharged with thanks. The following recommendations were made by this task force.

1. A component on psychiatric matters relevant to the aged, long-term health care facilities, and residential care facilities should be established. The Council plans to establish a task force to study staffing in various types of facilities.

2. There should be appropriate communication about matters pertinent to the aged and facilities for their care during APA's ongoing liaison with executive and legislative branches of the federal government. This recommendation was referred to the Joint Commission on Government Operations.

3. There should be communication with other interested organizations such as the American Medical Association, the American Nurses' Association, etc., indicating APA's interest in the psychiatric requirements of the aged, particularly in nursing facilities. This recommendation was referred to the Council on Professions and Associations for study by the Committee on Psychiatric Nursing and the Committee on Liaison with the American Hospital Association.

4. The APA Central Office should have a compendium of educational and source material available to interested parties. This was referred to the APA Librarian for comment.

5. District branches should take cognizance of the status of psychiatric services to the aging and to nursing homes in their areas, and legislative committees of district branches should be made aware that geriatric matters must not be neglected in their educational endeavors with legislators at local and state levels. These matters were referred to the Assembly for discussion by legislative representatives and in the district branches.

The *Task Force on Suicide Prevention*, Matthew Ross, M.D., chairperson, completed its charge, submitted a final report, and was discharged with thanks.

Statistical evidence prior to final analysis and refining indicates that the suicide rate among psychiatrists is higher than among other physicians and that the suicide rate among female psychiatrists is particularly high.

The task force had proposed a psychological autopsy study. This proposal, together with appropriate background material, was referred to the Council on Research and Development for its consideration.

The *Task Force on Collaboration of the Psychiatrist and Consumer in Psychiatric Programs*, June J. Christmas, M.D., chairperson, has completed its work and was discharged with thanks. The Council endorsed their recommendation that citizen involvement at the local community level should be through development of citizens' liaison councils and this was referred to the Assembly for implementation. The Council was unwilling to endorse the recommendation that APA develop a National Citizens Advisory Council with broadly representative membership, feeling that a national council was probably not necessary and might well be ineffective compared to one at a local or state level. The full report was circulated to the Reference Committee and there will be further discussion of it at the next meeting of that committee.

Stanley Holzberg, M.D., will chair a new *Task Force on Rehabilitation*, established by the Council at its October meeting. The task force will provide liaison for APA to counterpart elements of organizations, agencies, and institutions that have requested such liaison, as well as appropriate others, and develop a proposal for a structure within APA to act both independently and in conjunction with other organizations to contribute psychiatric input into all types of rehabilitative service.

The Council had been charged with updating the present position statement on "Organizational Structure of State Mental Health Programs." Dr. William Spriegel, after conducting a survey of district branches for the Council and in conjunction with Dr. James Lowry, prepared a revised position statement reflecting the opinions voiced by the district branches. The statement was endorsed by the Assembly and approved by the Board of Trustees in December and was published in the March 1976 issue of the *Journal*.

The Council was also charged with making recommendations relative to the different administrative responsibilities of nonmedical administrators and psychiatrists and attempting to provide a procedure for resolving possible conflicts within institutions where such a pattern exists. The recommendations of the Council were endorsed by the Assembly and approved by the Board of Trustees in December. Implementation will include a revision of Standard 15 of "Standards for Psychiatric Facilities" and establishment of an Assembly task force or survey of district branches to gather documentation or strong presumptive evidence of the incidence and prevalence of such conflicts.

The Council on Research and Development

LOUIS JOLYON WEST, M.D., CHAIRPERSON

DURING THE PAST year, the Council met three times—in April, at the annual meeting, and during the fall committee meetings. In May, Charles Gaitz, M.D., joined the Council, John J. Schwab, M.D., resigned, and Leigh M. Roberts, M.D., replaced E. Ivan Bruce, M.D., as the Assembly liaison. Continuing members of the Council during 1975 were Bernard C. Glueck, M.D., vice-chairperson, Lester Grinspoon, M.D., and Walter W. Shervington, M.D. James H. Carter, M.D., continued as observer/consultant. Nelson Hendler, M.D., was the Falk Fellow assigned to the Council. Other Falk Fellows and APA Fellows attended various meetings as observers.

After the report of the *Task Force on Lithium Therapy*, Irvin M. Cohen, M.D., chairperson, was completed, the task force was dissolved with thanks. In view of the importance and timeliness of the report, the Council recommended early publication; the report was published in the September 1975 issue of the *American Journal of Psychiatry*. The Council also saw a need for periodic reassessment of indications and contraindications for lithium therapy and may propose appointment of a new task force in two or three years to prepare a progress report.

The *Task Force on Methadone Therapy and Narcotic Antagonists*, Roger E. Meyer, M.D., chairperson, presented its final report to the Council. The report consisted of a summary of an earlier, multiauthored review. While informative, it had been largely superseded by several recently published articles. The Council therefore decided that the report should not be published as an official APA document, particularly as the field is still in a state of flux. In view of the fact that federal guidelines on this issue are changing, and because of discrepant views (not yet resolved by data) on this controversial subject, the Council dissolved the task force with thanks.

The *Task Force on Evaluation Research in Community Mental Health Centers*, Gerald L. Klerman, M.D., chairperson, was dissolved with thanks for its work. In 1974 it recommended that the APA Central Office join with NIMH to develop a commission to conduct ongoing evaluations of community mental health centers. Because of changes in NIMH policy, this was not accomplished; thus the task force could go no further with its mandate.

The *Task Force on Psychiatric Aspects of the Aerospace Program*, previously approved but never activated, was reconsidered. The Council noted the uncertainty of the relevance of the task force's charge to the broad APA activities and the insufficiency of available data. The Council decided that this task force should not be activated. It was thought that at some future time the Council should consider the advisability of establishing a new task force on the subject of "Man and Machine—Problems of Adaptation." Such a task force could study the entire area of psychiatric problems encountered in intensive care units, sensory and social isolation, the effects of weightlessness, etc. Aspects of the aerospace program could fit into such a larger study.

The *Task Force on Meditation*, James Morgan, M.D., chairperson, submitted a one-page report as a possible position statement for APA. After a list of references is prepared, this report will be submitted to the Reference Committee for approval of publication. Based on its findings, the task force recommended that well-controlled studies be conducted to evaluate the specific usefulness, indications, contraindications, and dangers of various meditative techniques. Such research should also compare various forms of meditation with one another and with psychotherapeutic and psychopharmacological modalities. The task force was dissolved with thanks and with the note that future developments in this area might require the establishment of a new task force in one or two years to review the state of affairs at that time.

The *Task Force on Effects of Television on Mental Health*, James H. Ryan, M.D., chairperson, completed its report and was dissolved with expressions of thanks for its work. The report (which includes several reviews said to represent more than 3,000 references) will be studied by the Council, and copies will be sent to selected experts for critical comment.

Activation of a Task Force on Research on Psychosurgery has been deferred pending study of Dr. John Donnelly's report on his Task Force on Psychosurgery (Council on Emerging Issues), soon to become available.

Other components of the Council reported progress toward their respective goals. Claudewell S. Thomas, M.D., began work on his Board-authorized study of "Role Con-

flicts of Psychiatrists." Dr. Thomas is collaborating with scientists at the UCLA Survey Research Center to develop the questionnaire and research protocol for the study.

The *Commission on Drug Abuse*, Daniel X. Freedman, M.D., chairperson, in preparation for its scheduled termination in 1976, expressed concern about its dissolution without provision being made for something to take its place. A serious need was seen for a mechanism through which APA can respond rapidly to inquiries on drug abuse from various sources or to internal requirements relevant to formulation of APA policy. Over the past five years the Commission on Drug Abuse has been used primarily by the APA Medical Director when urgent information and/or position statements were required in reaction to various critical issues arising in government agencies (including Congress) regarding the use of drugs of all types, especially those subject to abuse. The commission feels that several important responsibilities remain, e.g., keeping the membership fully informed about substance abuse and providing information and advice to various health-related and other agencies, both governmental and nongovernmental, that are (or should be) concerned about the drug abuse problem.

The commission sees five major areas of concern that need to be covered after the present commission is dissolved: 1) government liaison regarding scheduling of drugs, laws regulating treatment, and professional standards (e.g., training and/or use of paraprofessionals); 2) constant monitoring of changing policy issues on the use of drugs, including indications and contraindications for commonly used drugs in psychiatric practice; 3) keeping abreast of and being prepared to deal with potential problems in the future (e.g., the introduction of new substances that may find their way to the street and be abused, proposed new regulations for existing compounds; 4) changing patterns of alcohol and polydrug abuse (especially among teenagers) and how to deal with them; and 5) evaluation of both "standard" and "novel" therapies for drug abuse (e.g., methadone and narcotic blocking agents, drug-free therapeutic communities, meditation).

Based on these needs, the Council recommended to the Board of Trustees that a new Commission on Substance Abuse be established, to report directly to the Board, following the model of the Judicial and Legislative Commissions. Such a commission could itself develop suggestions for specific task force activities that might fall within the province of any of the several councils. A proposal could then be made to an appropriate council suggesting creation of a specific task force.

A progress report was received from the *Task Force on Biofeedback*, Martin T. Orne, M.D., chairperson, Bernard C. Glueck, M.D., Council liaison. This task force is focusing on the clinical significance of work on biofeedback as it relates to psychiatry. It is also defining the limitations of present knowledge about biofeedback. The task force feels that further research is needed before it can report comprehensively on the appropriate uses of biofeedback in psychiatry and in other areas of medicine as well. Areas of special interest are 1) the behavioral significance of biofeedback, 2) cortical and subcortical CNS representations of biofeedback, 3) relevance of biofeedback procedures to psychiatric illness, and 4) relevance of biofeedback procedures to medical problems not specifically psychiatric (e.g., epilepsy and hypertension). The aim of the task force's report will be to clarify the appropriate therapeutic uses of biofeedback at the present time, to delineate those areas which require more detailed research before justifying routine clinical applications, and to identify those areas where clinical application seems unjustified or contraindicated in light of available evidence.

This year the *Task Force on Ethics in Research*, Samuel B. Guze, M.D., chairperson, L.J. West, M.D., Council liaison, Robert J. Ursano, M.D., Falk Fellow, published a letter in the *American Journal of Psychiatry* requesting research protocols to study as precedents for the development of guidelines for ethics in research; response was negligible. As a result, the Council developed a new charge for consideration by the task force. This charge includes 1) review and analysis of ethical guidelines for psychiatric research formulated by other organizations and agencies, 2) delineation of ethical principles appropriate to psychiatric research design, with emphasis on such matters as informed consent and confidentiality, and 3) the provision of a set of sound ethical guidelines for psychiatric research that protect the rights of human subjects and at the same time allow the development of new knowledge.

The *Task Force on Racism in Psychiatric Research*, Claudewell S. Thomas, M.D., chairperson, Walter W. Shervington, M.D., Council liaison, is developing a questionnaire to be sent to various institutions doing research, asking them to identify the names of researchers who themselves represent minority groups. A second questionnaire will then be sent to the researchers so identified in order to gather information about various aspects of their research within the general context of how racism in all of its forms might be affecting their research activities, including problems of obtaining support. The results of these questionnaires will form the basis of a report to be produced by the task force. During this year the task force submitted a grant proposal to the Center for Minority Studies at NIMH to support this study but subsequently decided to withdraw the application.

Because of the large scope of its work, the *Task Force on Nomenclature and Statistics*, Robert L. Spitzer, M.D., chairperson, Bernard C. Glueck, M.D., Council liaison, Conrado Weller, M.D., Falk Fellow, has eight members, four official consultants, and over 30 special consultants. The task force has produced several documents to date: 1) a critique of *DSM-II* (with changes proposed for *DSM-III*); 2) an initial draft of *DSM-III* classifications, and 3) first drafts of texts for the sections on schizophrenic disorders, mood disorders, anxiety disorders, hysterical disorders, personality disorders, disorders arising in childhood, disorders of basic biological functions, stress disorders, impulse disorders, and miscellaneous matters. Still to be drafted are sections on organic mental disorders, alcohol and drug dependence, and paranoid disorders.

On June 10–12, 1976, the APA and the University of Missouri will cosponsor a conference on "Improvements in Psychiatric Classification and Terminology: A Working Conference to Critically Examine *DSM-III* in Midstream." This conference will provide a unique opportunity for invited experts in the areas to be covered in *DSM-III* to discuss and critique the task force's work to date. The information derived from the conference will also be used to develop an improved *DSM-IV*.

The Reference Committee, and subsequently the Board of Trustees, approved a recommendation by the Council that this task force be given every encouragement and support to produce *DSM-III* as speedily as is consistent with comprehensiveness and excellence. Accordingly, funds have been shifted within the Council's budget to provide additional support for this task force in the present year, and $25,000 has been approved for its work next year.

Four new task forces were approved by the Council this year.

1. The *Task Force on Electroconvulsive Therapy*, Fred Frankel, M.D., chairperson, Lester Grinspoon, M.D., Council liaison, quickly became embroiled in the national controversy surrounding ECT. At its first meeting, the task force concluded that the present crisis over ECT, including legislative actions being considered by several states, represents a major crisis for all of medicine. The response of the American Psychiatric Association to this particular issue of legislative control over medical practice will be very important and may provide a model for future activity.

The task force decided that its final report should include statements on the clinical efficacy of ECT, unestablished or experimental uses, adverse effects, methods of administration, basic science, social, moral, and legislative issues, training and education, and areas for further study. These statements should be based on all currently available empirical evidence (gleaned from a search of the literature, open hearings, a questionnaire study of the APA membership, and data collected about ECT use at high-caliber U.S. institutions).

While gathering and formulating this information will be expensive, the Council strongly supported the prospect of its speedy accomplishment. The council felt that this is an issue vital to all members of the APA, whether or not they use ECT in their practice. Because it is of such unusual importance, immediate funding is essential in order to develop the necessary information and successfully represent the Association and the various district branches in present and prospective legal and legislative issues surrounding ECT. Therefore, the Council urgently recommended that APA either help obtain the needed funds from other sources or provide the funds itself without delay.

2. The *Task Force on Research on Aging*, Carl Eisdorfer, M.D., Ph.D., and Lissy F. Jarvik, Ph.D., M.D., cochairpersons, Charles M. Gaitz, M.D., Council liaison, was given a charge by the Council chairperson. The task force has two years in which to address some fundamental issues in research, education, and treatment of the elderly. Members of the task force are Robert Butler, M.D., Eric Pfeiffer, M.D., and Alexander Simon, M.D.

3. Establishment of the *Task Force on Ethical Considerations in Behavioral Therapy*, Lee Birk, M.D., chairperson, Lester Grinspoon, M.D., Council liaison, was approved by the Council in April but tabled in October. The American Psychological Association currently has a multidisciplinary commission working on this subject. The Council wants to review the report of that group before proceeding with work by its own task force.

4. A *Task Force on Poverty and Requirements for Psychiatric Resources* was approved for establishment under the Council's auspices in response to the report of the Task Force on Delivery of Psychiatric Services in Poverty Areas (Council on National Affairs). The issues raised by the report were services for children and adolescents, services for the elderly, professional roles and skills, and professional rewards.

Before work began on this task force and after further consideration of these four issues, the Council concluded that a more efficient way to deal with three of them was to establish a new Task Force on the Role of the Psychiatrist in Poverty Areas under the Council on Mental Health Services, and the Council recommended this to the Board. The remaining issue, services for the elderly, will be referred to the new Task Force on Research on Aging.

Other Council actions during the year included 1) a request for establishment of a Task Force on the Aging Psychiatrist, which was referred to the Council on Internal Organization, 2) a decision not to establish at this time either a Task Force on Children and Religious Cults or a Task Force on Acupuncture, and 3) a recommendation that the Association (under the Division of Membership Services and Studies) sponsor the analysis of data already gathered on psychiatric manpower issues rather than initiating a new task force on manpower at this time.

The Council on National Affairs

HAROLD M. VISOTSKY, M.D., CHAIRPERSON

REVIEWING ITS CHARGE, role, and functions the Council affirmed or reaffirmed 1) the responsibility of monitoring the quality and output of the components of the Council and taking a strong organizational role in making direct input, 2) a commitment to activity, and 3) a responsibility to direct components to determine areas of common concerns. In considering several requests for reconstituting task forces as committees, the Council determined that there is a need to develop a policy regarding the development or evolution of permanent organizational components to assure representation of the underrepresented groups. The Council agreed that there should be a developmental period (the task force period) during which the minority professional membership is identified and organized into a coherent entity to make policy statements relative to common interests of the ethnic professionals represented by the committee charge. In accord with this proposed policy, the Council approved recommendations from the Task Force on Indian Affairs and the Task Force on the Mental Health of Spanish-Speaking People in the United States for restructuring as committees. These recommendations were approved by the Reference Committee and the Board of Trustees. Each component of the Council was urged to carefully consider the report of the Task Force on Mental Health Services to Poverty Areas and to forward its recommendation to the Council. Dr. Visotsky requested recommendations to the Task Force to Study Arbitrary Discrimination Against Persons with Previous Psychiatric Treatment.

The Council received the final report of the *Task Force on Third Party Intervention in Community Crisis*. Burr Eichelman, M.D., who had assumed responsibility for evaluating questionnaires circulated to a random sample of the membership, reported that 50% of the respondents have encountered community crisis (more than 25% have been actively involved in conflict resolution), and only 10% of the respondents were trained for these experiences (more than 50% indicated willingness to be trained).

The Council recommended that 1) Dr. Eichelman report the study in an article to be submitted to the *Journal* for publication, 2) a copy of the paper be directed to the Council on Medical Education for use in resident training and continuing education seminars, and 3) through an announcement in *Psychiatric News* and to district branches, a roster of interested members be developed.

During the year Johanna Clevenger, M.D., succeeded Morton Beiser, M.D., as chairperson of the *Task Force on Indian Affairs*. A unique feature of the task force has been its liaison activities with American Indian groups and projects. These include the Association of American-Indian Physicians, a component of the American Academy of Child Psychiatry, the Association of American Indian Affairs, the Association of Native American Medical Students, the Indian Health Service, and the American Indian and Alaskan Native Mental Health Research and Development Center. The task force was successful in obtaining funds from the Ittleson Family Foundation for a conference on the mental health of Indian youth. The conference, held in Portland, Ore., in April 1975, attracted a large number of Indian and non-Indian participants and was regarded as a very successful meeting. A monograph of the proceedings is being developed. Another major undertaking was the preparation of a summary of the evaluation of available programs and resources for American Indians; this report was developed under a contract with the Indian Health Service.

Several activities of the *Task Force on the Mental Health of Spanish-Speaking People in the United States*. Angel G. Gomez, M.D., chairperson, have served as a unifying and cohesive force for Spanish-speaking psychiatrists. In particular, a newsletter was published that included information on the activities of the task force and other matters of interest to Spanish-speaking psychiatrists. Efforts have been directed toward appointing additional Spanish-speaking psychiatrists to the various organizational components of the Association and planning the Simon Bolivar Lecture for the annual meeting.

The *Committee of Black Psychiatrists*, Charles B. Wilkinson, M.D., chairperson, has maintained active liaison with the Solomon Carter Fuller Institute and the Center for Minority Group Mental Health Programs. In keeping with the theme of the 1976 annual meeting, the committee selected a historian, Dr. John Hope Franklin, to receive the Solomon Carter Fuller Award and invited him to deliver the Fuller lecture. The task force communicated with the Task Force on Nomenclature and Statistics about the inclusion of racism in the diagnostic nomenclature and with the Assembly's Task Force on Key Conference Recommendations regarding procedures for assuring adequate representation of racial minority-group members. In response to President-Elect Gibson's inquiries, the committee recommended that all minority group members be considered in appointments as observer-consultants.

A number of position papers were solicited and received for discussion at the initial meeting of the new *Task Force of Asian-American Psychiatrists*, Lindbergh S. Sata, M.D., chairperson. These papers were solicited from a broad cross-section of the membership, focused on topics related to Asian-Americans and Asian refugees and mental health issues, and enhanced the productivity of the meeting. The task force has initiated a survey of identifiable Asian psychiatric resources. The need to document Asian mental health delivery system models for interested Asian psychiatrists was recognized as a potential function for the task force. Other areas of study included 1) the geographical distribution of Asian psychiatrists as related to the restrictive regulations for licensure, 2) the special requirements of continuing education for racially diverse multilingual Asian psychiatrists, and 3) the resettlement of Vietnamese refugees, which was partially resolved in the recent formation of an advisory council, composed entirely of Vietnamese and Cambodians, that assures relevant representation and input for all future refugee activities. In addition, individual task force members have been in contact with a variety of Asian community agencies that are actively developing supportive and rehabilitative services for the refugees. The recognition of the lack of Vietnamese psychiatric resources within APA and the awareness that a variety of projects have already been initiated by Asian com-

munity agencies led to the recommendation that the Association be available but unintrusive. The task force will attempt to communicate with relevant Asian groups to avoid the misconstruing of our actions by sensitive community groups. It was further agreed that two key roles of the task force would be active support of community efforts to provide humanistic services to the refugees and serving as a conduit for pertinent information received regularly by the APA Central Office to the task force members. It was further suggested that, while actual statistics are not currently available to the task force, approximately two-thirds of the refugee population is of middle-class origin, and for those conversant in French, French-speaking psychiatrists could be used if psychiatric intervention is indicated.

Before the formal opening of the fall committee meetings, the *Committee on Women* held a dinner meeting that was attended by the majority of women members of the various components in addition to two guests from the American Psychological Association. Several reports dealt with liaison activities with other components and the overall increase in activities and contributions of the women members of APA. Other activities reported during the proceedings of the committee meeting were Dr. Virginia Davidson's development of a handbook for assistance in the organization of committees on women in the district branches, and the development of a bibliography on the needs of residents. Drs. Nancy Roeske, Elaine Hilberman, and Anne Seiden reported on their respective research projects. The Council approved the recommendation submitted by chairperson Elissa P. Benedek, M.D., that APA publish Dr. Hilberman's monograph on rape. This recommendation was approved by the Reference Committee and the Board of Trustees.

Aristide H. Esser, M.D., has served as the acting chairperson of the *Task Force on Ecopsychiatric Data Base*. Liaison was established with the Committee on Comprehensive Health Planning to develop position statements—one related to the quality of life and the other related to health planning. Both statements were approved by the Council and referred to the Reference Committee, which took no action on the latter statement because the language was incorporated in the report of the Committee on Comprehensive Health Planning. The position statement on the quality of life was approved, pending editorial changes, by the Reference Committee. Other areas of study of the task force included alternative environments for troubled youth and environmental issues related to unemployment.

The *Committee on Comprehensive Health Planning*, Robert L. Williams, M.D., chairperson, was reconstituted from a task force. Additional responsibilities and activities have stemmed from the passage of the National Health Planning and Resources Development Act. To assist in the work of the committee, the Board of Trustees approved the creation of a staff position. Mr. Sam Muszynski was appointed to this position and has worked closely with the committee in following the planning of the proposed program. A comprehensive position statement developed by the committee was approved by the Council and subsequently by the Reference Committee and the Board of Trustees.

The Council on International Affairs

NORMAN ROSENZWEIG, M.D., CHAIRPERSON

THE COUNCIL CARRIED out several highly successful projects during this past year, including hosting activities at the 1975 annual meeting in Anaheim, Calif., and participation in the first Pacific Congress of Psychiatry in Melbourne, Australia, May 11–16, 1975. The task force that planned the APA participation at the congress was chaired by Dr. Alfred Auerback, and Dr. Floyd Cornelison provided leadership for the preparation of the APA portion of the program. A delegation of Swedish psychiatrists who attended the Anaheim meeting were beneficiaries of a special program planned by a task force under the leadership of Dr. Robert Garber.

Special arrangements for foreign guests at the 1976 annual meeting in Miami, Fla., are being made with the assistance of the Local Arrangements Committee. Immediately after the annual meeting on May 15–18, 1976, a Joint Meeting with the Caribbean Psychiatric Association and the Section on Psychiatry, Neurology, and Neurosurgery of the Puerto Rican Medical Association is being held in San Juan, Puerto Rico. Victor Bernal y del Rio, M.D., is chairperson of the Program Committee, and Jose Fumero, M.D., is supervising local arrangements.

The VI World Congress of Psychiatry will meet in Honolulu, Hawaii, August 28 to September 3, 1977. Shervert Frazier, M.D., is chairing the Organizing Committee and Gerald Sarwer-Foner, M.D., the Program Committee. An office for this congress has been established at APA headquarters, with Ms. Rosa Torres serving as coordinator.

The Board of Trustees approved APA cosponsorship of the Congress of the World Federation of Mental Health in Vancouver, B.C., Canada, in August 1977.

The *Task Force on Liaison with International and Foreign Psychiatric Organizations*, Nathan S. Kline, M.D., chairperson, is developing a roster of resource people who have extensive knowledge of a country or region. The Association could consult with these people on short notice when problems or questions arise concerning a particular area.

The *Task Force on Transcultural Aspects of Ethnocentricity Among Psychiatrists*, Ramon Parres, M.D., chairperson, continues to refine the questionnaire being used in their interviews designed to measure ethnocentricity. Preliminary interviews have been done in Canada, Mexico, New Jersey, and Louisiana.

The *Task Force on Psychiatry and Foreign Affairs: Psychological Aspects of Near Eastern Studies*, William D. Davidson, M.D., chairperson, held its final meeting in October 1975. During the past two years, the task force participated in meetings at Princeton University, the University of London, Georgetown University, and the U.S. State Department. Its insights and approaches to some of the problems in the Middle East were valuable contributions.

Two new task forces are being initiated this year. The *Task Force on Cross-Cultural Perspectives of Psychiatric Practice*, Joseph T. English, M.D., chairperson, held its first meeting in Washington, D.C., on Jan. 23, 1976. Rita Rogers, M.D., will chair a new *Task Force on Psychiatry and Foreign Affairs; the Cyprus Conflict.*

The *Inter-American Council of Psychiatric Associations* held a business meeting in Mexico City on January 26 and 27, 1976. The first printing of the Spanish edition of the *Psychiatric Glossary*, royalties to the IACPA, will be distributed only in Mexico until a suitable translation can be done for the other countries in Latin America.

The Council on Professions and Associations

RUTH I. BARNARD, M.D., CHAIRPERSON

THE NEW MEMBERS of the Council are Leon Roth, M.D., and Warren Williams, M.D. The latter was appointed to complete the term of Lewis L. Robbins, M.D., who was elected trustee-at-large.

The *Committee on Liaison with American College of Physicians*, Charles P. Neumann, M.D., chairperson, is concentrating on reviewing its past role and functions, determining its goals, and identifying more precisely the current problems of joint concern to APA and ACP. Dr. Neumann has completed his term of office, having served as an active member for six years and as a most productive chairperson for the past three years.

The *Task Force on Confidentiality as It Relates to Third Parties*, Maurice Grossman, M.D., chairperson, has completed its work and its report has been published. A new Committee on Confidentiality will be established within the Council on National Affairs in May 1976. This new component will provide a continuing mechanism to discover, disseminate information about, and coordinate efforts to deal with problems of confidentiality on a national basis.

The Council continues to monitor the Insurance Coding Project developed by this task force. Dr. Grossman and Dr. Richard Johnson are pursuing this issue with representatives of the Health Insurance Council (HIC) in an effort to find a way to provide insurance companies with the information they need to process patients' claims for psychiatric treatment while preserving an adequate degree of confidentiality. A coding system has been worked out and is ready for a field trial with a substantial number of insurance companies that are members of HIC. There has been difficulty, however, in getting support and participation in this trial from a sufficient number of district branches. The project is neither complicated nor expensive, and it is very important that whatever problems the district branches are having in understanding and cooperating in the field trial be solved before we lose the excellent cooperation we have had with HIC.

The *Committee on Psychiatry and the Law*, G.J. Sarwer-Foner, M.D., chairperson, undertook the development of a model commitment law, as charged by the Reference Committee and the Council. When consensus has been reached, the recommendations of the committee will be put in proper legal language by attorneys. This work is progressing actively and is the main current thrust of the committee. Freedom, involuntary hospitalization, the ethics of psychiatry, the right to individual freedom and liberty, the right to be protected and treated, as well as the right to refuse treatment are among the concerns and issues the committee is dealing with. Dr. Stanley Portnow is the committee's representative for further testimony (if necessary) before the Senate regarding APA's stand favoring retention of insanity as a defense as it relates to proposed Senate Bill 1 and Senate Bill 1400.

The committee expressed thanks for the contributions of Professor Henry H. Foster, Jr., and Ephraim Gomberg from the Family Law Section and the Section on Criminal Law of the American Bar Association and asked them to continue in the work of the committee.

The *Task Force on the Right to Treatment*, Jonas R. Rappeport, M.D., chairperson, was to have completed its work this year; its position statement was accepted by the Reference Committee and the Executive Committee of the Board of Trustees in June. However, the Assembly did not accept the statement in its entirety, and the task force will therefore be continued until a position statement acceptable to all can be formulated.

The report of the *Task Force on Religion and Psychiatry*, Abraham N. Franzblau, M.D., chairperson, "Psychiatric Services to Religious Institutions and Viewpoints on Religion," is in press. The final phase of its work will be completed by October 1976.

The *Committee on Liaison with American Hospital Association*, Francis de Marneffe, M.D., chairperson, reported that there has been continuing active exchange of information between APA and AHA regarding pending legislation, federal regulations, national health insurance, and confidentiality of records.

The committee asked for support of House Rule 3658 and Senate Bill 1678 as well as House Rule 8231 and Senate Bill 2258. If passed, they would require that regulations be reviewed by Congress to ensure that they actually conform with the intent of Congress.

On mutual agreement with the AHA and for reasons of economy, the number of members will be reduced from six to four persons on both the APA committee and its AHA counterpart.

The *Committee on Psychiatric Nursing*, William B. Stone, M.D., chairperson, in collaboration with their American Nurses' Association counterpart, participated in APA's 1975 Institute on Hospital & Community Psychiatry. A session on "Predicting Interprofessional Alliances in the 80's" was held.

The committee has completed a first draft on the nurse psychotherapist, which is now being studied by the American Nurses' Association. It is hoped that this statement will be approved by both ANA and APA.

The committee has been performing a very important but informal liaison activity with ANA, particularly through its chairperson, and recommends that this be formalized by the APA President appointing an individual member (who is not the chair of the committee) as APA's representative to ANA. The Council referred this recommendation to the Task Force on Liaison Activities for inclusion in their overall conclusions.

The *Task Force on Liaison Activities*, Walter E. Barton, M.D., chairperson, in its first report identified and categorized liaison activities as either extra-APA, both substantive and ceremonial, or intra-APA, between councils and components with interface with staff at all levels. The task force is mainly concerned with developing guidelines for liaison activities, criteria for their establishment, and mechanisms for accountability.

The *Task Force to Study Standards and Guidelines for Psychiatrists Working with Paraprofessionals*, Donald H. Naftulin, M.D., chairperson, is composed of three persons in geographical proximity who will formulate standards and guidelines for psychiatrists working with paraprofessional personnel as supervisors, consultants, trainers, administrators, etc. Their report is expected by October 1976.

The Council on Internal Organization

MILTON GREENBLATT, M.D., CHAIRPERSON

THE COUNCIL MET in mid-October 1975 and heard from its many components. The actions that took place at that meeting and further actions by the Reference Committee and Board of Trustees at subsequent meetings will be described in this report.

The Council, together with business consultants, reviewed the work of the advertising agencies that handle APA's account for the *American Journal of Psychiatry*, *Psychiatric News*, *Hospital & Community Psychiatry*, and APA's exhibit programs. The agency for the *Journal* and *Psychiatric News* reported that revenues were satisfactory, with a general increase since the major dip around the time of the Hawaii meeting. The circulation of *Hospital & Community Psychiatry* has grown to about 17,000. One consultant stressed the importance of ethical standards in advertising and indicated that advertisers could probably be held easily to the ethical standards prescribed by APA. Another consultant remarked that APA's journals are holding their own despite increased competition and the fact that relatively few drugs are prescribed by psychiatrists. The commission that APA pays its advertising agencies may be slightly higher than the prevailing rate in general industry. Therefore, the Deputy Director for Business Administration was authorized by the Council to renegotiate contracts to achieve a saving.

Dr. Henry Laughlin chaired a study, undertaken with the aid of APA staff, of the history and purpose, costs and revenues, and current operations of the Hospital & Community Psychiatry Service and journal. Dr. Laughlin concluded that these interdependent operations merited high marks for both services rendered and cost efficiency. The APA contribution to these functions has increased in recent years for three main reasons: 1) a full overhead charge is now being made (for tax purposes), 2) subscription fees had not been raised commensurate with increasing costs, and 3) advertising revenues dipped last year. Fees have now been raised for the coming year, and vigorous circulation promotion efforts will be made. Advertising revenue shows signs of improvement, and rates will be raised.

The Council approved of the measures being taken to increase revenue and will continue to monitor these operations for the purpose of stimulating progress toward greater self-sufficiency and continuing program improvement. The Board of Trustees also requested that the Council review these operations again in one year.

The *Institute on Hospital & Community Psychiatry Program Committee*, Alyce C. Gullattee, M.D., chairperson, reported that the contributions at the recent institute were of a high quality and that the institute is regarded as a major continuing education effort, especially for ancillary personnel; Dr. Henry Work reported that 916 persons attended and that one-third of them received the Hospital & Community Psychiatry Service.

The Council recommended that the service be kept under continued monitoring and that further thought be given to possible modification of journal policy, leading to higher quality articles that might command a larger audience without sacrificing their appeal to the wide sectors of ancillary and allied personnel.

The *Task Force on Interface Between Industry and Psychiatry*, Harvey J. Tompkins, M.D., chairperson, recommended (and the Council, Reference Committee, and Board of Trustees later approved) that a Committee on Advertising Policy reporting to the Council be established. This committee would review advertising policy, monitor ads, resolve or finally adjudicate any differences that might occur among the editors of various APA journals, and be the final arbiter regarding the acceptance of ads that the editors believe may violate our advertising guidelines. The task force also reviewed and approved "Principles and Guidelines of Advertising Acceptance."

The Council discussed the ethical issues raised in the task force report and recommended, with subsequent concurrence by the Reference Committee and Board of Trustees, formation of a Task Force to Study the Impact of Potential Loss of Pharmaceutical Support. Such loss might result if pharmaceutical advertising and other direct and indirect support of APA by drug firms were terminated. Task force members, some of whom will be drawn from the Assembly, will also consider the related ethical issues that exist whenever APA receives funds from pharmaceutical companies for any purpose.

The Task Force to Study the Impact of Potential Loss of Pharmaceutical Support was granted $3,000 funding for fiscal year 1976–1977.

Peter A. Martin, M.D., chairperson of the *Committee on Program*, noted that the committee has been moving gradually from a "receptive" position to a more "active" position in stimulating submission of special sessions at the annual meeting to cover areas deemed important to a well-balanced program of continuing education. He stressed the importance of setting a central theme for the meeting very early in the year in conjunction with the President. The committee recommended the continuation of the open policy on panels as an excellent safeguard for participation in the program by the entire membership.

Twenty courses with an enrollment fee of $15 will be offered at the 1976 annual meeting; this continuing education program will be self-sustaining. Teaching tapes will run continuously through the day at the meeting and will provide AMA Category I credit for participants. There will also be a program of current films of interest to the membership.

There are numerous issues connected with recording, editing, and selling tapes of presentations at APA's various meetings. A task force may be established to consider these factors, but in the interim Mr. Robert L. Robinson, Director of Public Affairs, has been authorized by this Council to explore development of teaching tapes from the 1976 annual meeting and to report his findings to an early meeting of the Reference Committee.

The *Committee on Conventions*, W. Edward McGough, M.D., chairperson, recommended, with endorsement or approval by the Council. Reference Committee, and Board, that 1) a brief description of the placement service program be published in *Psychiatric News*, that employers and applicants who wish to use the service be encouraged to apply through the Membership Services Department, and that completed forms be reproduced and made available at the Placement Service booth during the annual meeting; 2) the Task Force on Arrangements assume responsibility for ensuring that adequate facilities be available for day-care services for children at the annual meeting, such program to be self-sustaining through charges to cover expenses; 3) the Board of Trustees endorse appointment of an ad hoc committee on spouses' scientific program; 4) certain categories of registration fees be increased to help defray the escalating costs of the annual meeting; 5) the Subcommittees on Technical and Scientific Exhibits be merged; and 6) exhibitors of sex therapy and related films rent an exhibit booth for placement of orders, exchange of technical data, etc., but that films be viewed in a separate sound-controlled room, with viewing limited to professionals.

The *Committee on Public Information*, Eric Plaut, M.D., chairperson, recommended hiring an assistant to the Director of Public Affairs, and the Board of Trustees approved implementation by the Medical Director, within available funds for program expansion. The committee also recommended a survey of district branches to identify their public information components and activities and a number of long-range plans, including development of a Joint Commission on Public Affairs, holding a conference relating APA to the media, creation of a Media Advisory Board, and a new popular publication to help build a better image for psychiatry.

The Board disapproved the committee's recommendation that a Commission on Public Affairs be formed to report to the Board.

The *Committee on Member Insurance and Retirement Plans*, Chester L. Trent, M.D., chairperson, brought several matters before the Council.

1. Professional Liability Insurance Program. The Council, the Reference Committee, and the Board approved a change in rates for professional liability insurance and recommended that filing of rate schedules for the states of California, Florida, New York, and Ohio be delayed until after Jan. 1, 1976, and that a study be undertaken of actuarial problems where serious rate differences exist between different companies in the same state.

2. Retirement Plan Trust. Results of a reevaluation of the APA member retirement plan, together with concerns relative to a recent audit of the plan, point to the need to make new arrangements for offering a retirement plan to APA members. The Retirement Plan Trustees recommended that the Retirement Plan Trust be dissolved and appropriate functions of the trust be transferred to a corporate trustee or other appropriate investment custodian. The Council, the Reference Committee, and the Board approved this recommendation with the understanding that the Retirement Plan Trustees will present a detailed proposal for final approval at a later date. These actions will in no way jeopardize existing member pension benefits.

The Board also approved funding a one-year task force study to consider problems (and their possible solutions) faced by persons with a psychiatric history in obtaining insurance.

Joan Eccleston, M.D., chairperson of the *Committee of Residents*, reported that the committee has markedly increased its liaison activities during the past year, both within and outside APA and its district branches. The committee has developed a recruitment packet for residents and has planned a series of editorials on issues of interest to residents for publication in *Psychiatric News*.

Upon recommendation of the committee and the Council, the Reference Committee will refer to the Assembly the request that its Membership Committee consider the appointment of an "ombudsor" for resolving disputes between psychiatric residents and residency programs, and will ask the American Association of Directors of Psychiatric Residency Training Programs to review this proposal and provide suggestions for its development and implementation.

The Council and Reference Committee recommended that the Committee of Residents be commended for its extensive activities of the past year on public information, liaison activities, recruitment, problem solving, and ongoing program review; this was endorsed by the Board.

The Council and Reference Committee voted to recommend the following:

1. APA determine and inform the Committee on Women what information exists in its files concerning the participation of women and other minority members at the local and national level as compared with male participation; this would include the number of such members, identification of their representation on district branch and national components, information on full- and part-time practices, and the age when they became active members (approved by the Board).

2. Mechanisms to acquire this information should be established when it is not currently available; such mechanisms would include requesting the Assembly, through its district branches, to provide data about its members (approved by the Board).

3. The President of APA was requested to provide information as to how he handled his appointments to include women and minority members and to make suggestions for the future.

The *Task Force on Manpower Problems*, Richard Steinhilber, M.D., chairperson, worked with Dr. Lee Gurel of the APA Central Office to define two manpower studies that were subsequently approved by the Board for funding by outside sources. The first study would identify nonmember psychiatrists and would assist in more accurately planning for meeting the nation's needs, e.g., in national health insurance, and make possible a complete census of all psychiatrists.

The goal of the second study would be to complete information on nonmembers and to add to existing information on members in a number of critical areas not previously collected, such as primary work site, work status, work activities, type of practice, areas of perceived educational need, and interests in continuing education. All data would be stored in the computer and be available for retrieval. These data would provide a basis for new member recruitment and make possible publication of a biographical directory.

Based on recommendations of the Committee of Residents, Drs. Gurel and White of the APA staff, the Council on Internal Organization, and the Reference Committee, the Board of Trustees requested that staff explore revision of the APA's membership brochure, as suggested at the Key Conference. Development of the brochure would be undertaken by the Committees on Membership of APA and the Assembly and by the Committee of Residents. Although this is considered a high priority, funds are not currently available to complete the project.

The Board accepted with thanks a review of APA campaign procedures for national elective office and for the mailing of related political literature. The Council designed guidelines in both areas to ensure the highest level of ethical political conduct among those seeking office in APA.

A further analysis of APA's councils and committees was made by Dr. Jack White and Ms. Patricia Scheidemandel of the Central Office. Some highlights from their study of 63 components include:

1. Men are slightly overrepresented as compared with women in service in APA components.
2. Component members are about five to eight years older on the average than APA members; however, some of the younger members, such as Falk Fellows, were not included in the analysis.
3. A comparison of the geographic distribution of component members with the general distribution for APA members showed that Massachusetts had about twice as many component members as the general APA population, and New York had fewer than might be expected. When viewed by census divisions, New England had about twice as many component members as might be expected, the Mid-Atlantic area had fewer, the South Atlantic area slightly more, and the East-North Central area fewer than might be expected. There is little evidence of overlap in objectives among the numerous components.

The Reference Committee endorsed the following guidelines for distribution to all councils and chairpersons of components:

1. Components should participate in the development of the council budget.
2. Financial statements should be made available to components at intervals.
3. Each component would assign specific agenda items to component members, including a budget item.
4. Any unusual or extra expenses of a component would require approval of the chair of the council.
5. Conference calls should be used more often.
6. Some components could function effectively with fewer members.
7. Components could often work as well using members from regional locations rather than having representation from all over the country.

The *Committee on Grants and Awards*, Jack Weinberg, M.D., chairperson, reviewed APA's award program, giving particular attention to two that are running out of funds—the Hofheimer Prize Board and the Vestermark Prize Board.

The Board approved the following recommendations: 1) it become APA policy that all new awards established by APA be self-sustaining, 2) the Medical Director be authorized to seek additional funds to continue the Hofheimer Prize, and 3) the Deputy Director for Business Administration be authorized to develop an APA award fund using the unrestricted funds in the grants and awards account and the funds currently in the special purposes account to help fund existing awards and to establish new awards where appropriate.

The Council received the reports of the several award boards, including the *Hofheimer Prize Board*, the *Vestermark Prize Board*, the *McGavin Award Selection Board*, and the *Hospital & Community Psychiatry Service Achievement Awards Board*, whose selections will be publicly announced at the appropriate times.

Dr. Greenblatt would like to express his appreciation to Dr. Jack White, staff liaison, and to the Council members for their enthusiastic efforts.

The Council on Emerging Issues

JERRY M. LEWIS, M.D., CHAIRPERSON

THE COUNCIL HELD three meetings during the past year. Two task forces of the Council continue to work on reports that will soon be ready for the Council's review. These are the *Task Force on Psychosurgery*, John Donnelly, M.D., chairperson, and the *Task Force to Define Mental Illness and "What is a Psychiatrist?"*, Morton Reiser, M.D., chairperson.

The *Task Force on Psychohistory*, Charles K. Hofling, M.D., chairperson, completed its report in 1974; it was unanimously approved by the Council for publication and was also approved by the Reference Committee and sent to the Board of Trustees. On December 6, 1975, the Board declined to authorize its publication as an APA task force report, giving the task force permission to publish it elsewhere. The Council on Emerging Issues continues to believe that the report is a scholarly and thoughtful document that should be available to all who are concerned about the issues of psychohistory and psychoprofiling.

The Council received a request from the Assembly of District Branches to prepare a charge for a national commission on the chronic psychotic patient. This issue has been discussed by the Council and additional information is being gathered from other agencies concerned with the welfare of the chronic psychotic patient.

A wide variety of emerging issues has been studied by the Council. These include the psychiatric aspects of decision making in the area of the dying patient, the lack of standards for sex therapy clinics, the current status of antipsychiatry groups, the variables involved in the increased number of assassination attempts, the misuse of psychiatry for political purposes, and changing suicide and homicide rates. The Council has also discussed at length the relationship of increased unemployment to mental health. A panel on this subject has been arranged for the 1976 annual meeting and will be chaired by Perry Ottenberg, M.D., vice-chairperson of the Council.

The Council plans to monitor a small number of task forces in order to have adequate time available to discuss a wide variety of emerging issues. From time to time the Council will recommend the formation of a task force regarding such an issue but will frequently ask that such task forces be affiliated with other Councils.

The Assembly of District Branches

AS 1975 BEGAN, the Key Conference was being actively planned, and the Assembly worked hard to make sure that there would be a good geographic distribution and representation of its members at the March session. Since many of the events leading up to the Key Conference stemmed from a decade-long concern in the Assembly about the governance of the organization, it was felt that the Assembly should be well represented. When the Assembly *Task Force on the Key Conference Recommendations* was appointed at the annual meeting in May, it began actively discussing both the structure of the Assembly and its relationship to other components of the organization.

The Procedural Code was reorganized during the summer and approved by the Assembly at its fall meeting. The Task Force on the Key Conference Recommendations continued to work out procedural relationships with the Board of Trustees. A Joint Conference Committee, whose penultimate meeting was scheduled for December 1975, began to finalize proposals to be incorporated in the proposed amendments to the Constitution.

Other committees of the Assembly reflected changes. The *Nominating Committee* faced the prospect of greater responsibility and the need to become more sophisticated in its processes. This committee is currently discussing the requirements of future candidates to submit written position statements and written responses to questions for publication in the election issue of *Psychiatric News*. There are also new proposals from the Key Conference on elections for representatives, better organized slates for area trustees, etc.

The *Membership Committee* has also been increasingly active. Representation on the APA Membership Committee reflects the concerns and ideas of younger members of the Assembly in assisting district branches, revising the pamphlet for prospective members, etc.

The Assembly *Budget Committee* will face new tasks during the coming year. The budget of the Assembly has never been thoroughly studied, and it will have to be totally reorganized in keeping with the changed responsibilities of the Assembly.

The *Task Force on Delivery of Mental Health Services* has

continued to work closely with the APA Committee on Financing of Mental Health Care and participated in the final report of that committee. Also, the task force has continued to support a three-tiered model of psychiatric peer review as developed by the APA Committee on Peer Review. Financing has been provided for a member of the APA Committee on Peer Review to visit district branches to aid them in establishing or improving their peer review process.

The *Rules Committee* is an increasingly important committee of the Assembly. At the May 1975 annual meeting the committee was extremely valuable in organizing the presentation of action papers. The Rules Committee continued to operate at the fall meetings, but the pressure of business, primarily related to the Key Conference, was so great that the Assembly was not able to complete its task properly.

The *Task Force on an Insurance Coding Project* was appointed by the Assembly to implement in several district branches a field trial of a coding system for psychiatric illness. This has been developed by the APA Task Force on Confidentiality in Relation to Third Party Payments. The Assembly requested that there be a trial in three areas, Northern and Southern California, Connecticut, and Wisconsin. Since it will be essential to include participation of the "Blues," the Assembly suggested that the trial might also include the use of Procedural Terminology for Psychiatrists. The pilot study will be in the form of a research project removed from the actual claims-settling process. The task force will continue to confer with insurance representatives and maintain liaison with other Assembly and APA components engaged in parallel endeavors.

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

MAY BE AN APPROPRIATE CHOICE FOR YOUR NEXT PATIENT WITH CLINICAL DEPRESSION/ANXIETY

See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and
ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

Proved efficacy in the treatment of clinical depression/anxiety

In the five years since its introduction, Sinequan (doxepin HCl) has objectively demonstrated its value in the treatment of clinical depression/anxiety. Clinical studies have shown Sinequan to be unsurpassed in effectiveness when compared to other tricyclic antidepressants.

In addition, Sinequan may offer certain advantages not generally seen with some other drugs of its class:

Marked antianxiety activity

In addition to its significant antidepressant activity, which is usually seen two to three weeks after initiation of therapy, Sinequan possesses marked antianxiety activity.

In double-blind comparisons between doxepin and amitriptyline,[1-3] the antidepressant effects of both drugs were comparable; however, more notable antianxiety effects were seen with doxepin. Superior antianxiety activity was reported among depressed patients treated in a crisis intervention clinic.[4]

Prompt sedative activity

Of the tricyclic antidepressants, Sinequan has been found to have one of the most prominent sedative effects.[5] This sedative property can be taken advantage of by administering the major portion of the daily dose *h.s.* Since clinically depressed/anxious patients frequently have accompanying sleep disturbances, this may be a valuable benefit.

Generally does not affect the activity of guanethidine at usual clinical doses[6]

"A new tricyclic antidepressant, doxepin, was evaluated for its ability to block the amine uptake mechanisms of the peripheral adrenergic neuron and blood platelet in man. At low doses, there was no evidence of inhibition of the amine pump." At clinical dosages up to 150 mg. per day, Sinequan can be given to man concomitantly with guanethidine and related compounds without blocking the antihypertensive effect.[6]

Cardiovascular side effects reported infrequently

In a series of double-blind comparative studies, 1706 doxepin-treated patients were evaluated for side effects. Incidence of tachycardia was associated with only 45 (2.6%) clinically depressed/anxious patients.[3]

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See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

DIAGNOSTIC EVALUATION FORM

NAME_____ DATE_____

Date and Place of Birth_____ Age_____ Marital Status_____

Home Address_____ Telephone_____

Business Address_____ Telephone_____

Spouse or Nearest Relative_____ Relationship_____

Address_____ Telephone_____

Referred by_____

CHIEF COMPLAINT:

PRESENT ILLNESS:

PAST HISTORY:

FAMILY HISTORY:



MAY BE AN APPROPRIATE CHOICE FOR YOUR NEXT CLINICALLY DEPRESSED/ANXIOUS PATIENT

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and
ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, paresthesias, ataxia, and extrapyramidal symptoms.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, facial edema, photosensitization, pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, and anorexia have been reported. (See anticholinergic effects).

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice and alopecia have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg. t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg./day to 150 mg./day.

In more severely ill patients an initial dose of 50 mg. t.i.d. may be required with subsequent gradual increase to 300 mg./day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg./day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg./day.

As an alternative regimen, the total daily dosage, up to 150 mg., may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Supply. Available as capsules containing doxepin HCl equivalent to 10 mg., 25 mg., 50 mg., and 100 mg. of doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN (doxepin HCl) 25 mg. and 50 mg. also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg./ml.) is available in 120 ml. bottles with an accompanying dropper calibrated at 5 mg., 10 mg., 15 mg., 20 mg., and 25 mg. SINEQUAN Oral Concentrate should be diluted with approximately 120 ml. of water or orange juice prior to administration.

More detailed professional information available on request.

Pfizer **LABORATORIES DIVISION**
PFIZER INC.

From Lederle Laboratories

Loxitane®
LOXAPINE SUCCINATE
Lederle

Breaks the "silence" of schizophrenia



Remarkable performance in symptom control

See LOXITANE prescribing information on the last page of this advertisement for warnings and precautions and for more detailed information concerning side effects.

Loxitane®
LOXAPINE SUCCINATE
Lederle

Clearly exhibited efficacy in the reduction of symptoms and symptom patterns

EVALUATION OF RESPONSE TO LOXITANE BY THE BRIEF PSYCHIATRIC RATING SCALE (BPRS) IN 221 SCHIZOPHRENIC PATIENTS

(98 acute schizophrenic patients in 5 double-blind studies[1] treated for 3 to 6 weeks; 123 chronic schizophrenic patients in 6 double-blind studies[1] treated for 8 to 12 weeks)

SYMPTOM CLUSTERS (BPRS)		PERCENT MAXIMUM POSSIBLE IMPROVEMENT: Acute Schizophrenic Patients	PERCENT MAXIMUM POSSIBLE IMPROVEMENT: Chronic Schizophrenic Patients		Pretreatment Score	Posttreatment Score	Statistical significance of change from Base Line: p=
DEPRESSION Somatic Concern Anxiety Guilt Feelings Depressive Mood		56%	27%	ACUTE	3.07	1.92	0.001
				CHRONIC	2.02	1.74	0.01
THOUGHT DISORDER Conceptual Disorganization Grandiosity Suspiciousness Hallucinatory Behavior Unusual Thought Content		61%	23%	ACUTE	3.88	2.11	0.001
				CHRONIC	3.30	2.78	0.05
ANERGIA Emotional Withdrawal Mannerisms and Posturing Motor Retardation Blunted Affect		47%	17%	ACUTE	3.75	2.46	0.001
				CHRONIC	3.75	3.27	0.05
EXCITEMENT-DISORIENTATION Excitement Disorientation		61%	21%	ACUTE	2.54	1.60	0.01
				CHRONIC	2.50	2.19	—

1. Data on file. Clinical Research Department, Lederle Laboratories.

See LOXITANE prescribing information on the last page of this advertisement fo

Loxitane®
LOXAPINE SUCCINATE
Lederle

Exhibited certain favorable trends in side effects profile

IN 31 STUDIES INVOLVING 469 ACUTE AND CHRONIC SCHIZOPHRENIC PATIENTS, CERTAIN FAVORABLE TRENDS WERE EXHIBITED IN THE LOXITANE SIDE EFFECTS PROFILE; THESE REQUIRE FURTHER TESTS AND BROADER CLINICAL EXPERIENCE FOR CONFIRMATION.



While cardiac arrest, blood dyscrasias and renal toxicity have been reported with other antipsychotic agents, they have not been seen with LOXITANE. Hepatotoxicity manifested by jaundice or biliary stasis has not been observed. Transient liver enzyme changes, however, have been reported, but it has not been determined whether they are related to LOXITANE administration.

Although a few cases of changes in ECG have been reported, a causal relationship between this reaction and LOXITANE has not been established. Clinical experience with LOXITANE has not demonstrated ocular toxicity; however, the possibility of its occurrence cannot be ruled out at this time. Of the various endocrine abnormalities, only galactorrhea has been reported with LOXITANE and only in rare instances. Manifestations of adverse effects on the central nervous system other than extrapyramidal symptoms have been encountered infrequently, and drowsiness, when it occurs, is usually mild and subsides with continued therapy. Skin rashes of uncertain etiology have been observed in a few patients during the hot summer months; therefore the possibility of phototoxicity and/or photosensitivity cannot be excluded.

In general, LOXITANE presents a profile of extrapyramidal side effects similar to that of other agents used in the treatment of schizophrenia.

Cardiovascular effects such as hypotension, hypertension, lightheadedness and syncope have been reported. Anticholinergic effects seen with LOXITANE include dry mouth, nasal congestion, constipation and blurred vision.

arnings and precautions and for more detailed information concerning side effects.

FOR ORAL USE

PRODUCT INFORMATION

DESCRIPTION

LOXITANE *loxapine*, a dibenzoxazepine compound, represents a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines. Chemically, it is 2 - chloro - 11 - (4 - methyl - 1 - piperazinyl) dibenz [b,f] [1,4] oxazepine. It is present in capsules as the succinate salt. Each 1.36 mg of loxapine succinate is equivalent to 1 mg of loxapine.

ACTIONS

Pharmacologically, loxapine is a tranquilizer for which the exact mode of action has not been established. However, changes in the level of excitability of subcortical inhibitory areas have been observed in several animal species in association with such manifestations of tranquilization as calming effects and suppression of aggressive behavior.

In normal human volunteers, signs of sedation were seen within 20 to 30 minutes after administration, were most pronounced within 1½ to 3 hours, and lasted through 12 hours. Similar timing of primary pharmacologic effects was seen in animals.

Absorption of loxapine following oral or parenteral administration is virtually complete. The drug is removed rapidly from the plasma and distributed in tissues. Animal studies suggest an initial preferential distribution in lungs, brain, spleen, heart, and kidney. Loxapine is metabolized extensively and is excreted mainly in the first 24 hours. Metabolites are excreted in the urine in the form of conjugates and in the feces unconjugated.

INDICATIONS

LOXITANE *loxapine succinate* is indicated for the manifestations of schizophrenia.

CONTRAINDICATIONS

LOXITANE is contraindicated in comatose or severe drug-induced depressed states (alcohol, barbiturates, narcotics, etc.)

LOXITANE is contraindicated in individuals with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Safe use of LOXITANE *loxapine succinate* during pregnancy or lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the benefits of treatment be weighed against the possible risks to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from midpregnancy with doses of 0.6 and 1.8 mg/kg, doses which approximate the usual human dose but which are considerably below the maximum recommended human dose.

Usage in Children: Studies have not been performed in children; therefore this drug is not recommended for use in children below the age of 16.

LOXITANE, like other tranquilizers, may impair mental and/or physical abilities, especially during the first few days of therapy. Therefore, ambulatory patients should be warned about activities requiring alertness (eg, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

PRECAUTIONS

LOXITANE loxapine succinate should be used with extreme caution in patients with a history of convulsive disorders since it lowers the convulsive threshold. Seizures have been reported in epileptic patients receiving LOXITANE at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy.

Loxapine has an antiemetic effect in animals. Since this effect may also occur in man, loxapine may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

LOXITANE should be used with caution in patients with cardiovascular disease. Increased pulse rates have been reported in the majority of patients receiving antipsychotic doses; transient hypotension has been reported. In the presence of severe hypotension requiring vasopressor therapy, the preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine.

The possibility of ocular toxicity from loxapine cannot be excluded at this time. Therefore, careful observation should be made for pigmentary retinopathy and lenticular pigmentation since these have been observed in some patients receiving certain other antipsychotic drugs for prolonged periods.

Because of possible anticholinergic action, the drug should be used cautiously in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

ADVERSE REACTIONS

CNS Effects: Manifestations of adverse effects on the central nervous system, other than extrapyramidal effects, have been seen infrequently. Drowsiness, usually mild, may occur at the beginning of therapy or when dosage is increased.

It usually subsides with continued LOXITANE therapy. The incidence of sedation has been less than that of certain aliphatic phenothiazines and slightly more than the piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported.

Extrapyramidal Reactions—Neuromuscular (extrapyramidal) reactions during the administration of LOXITANE *loxapine succinate* have been reported frequently, often during the first few days of treatment. In most patients, these reactions involved Parkinson-like symptoms such as tremor, rigidity, excessive salivation, and masked facies. Akathisia (motor restlessness) also has been reported relatively frequently. These symptoms are usually not severe and can be controlled by reduction of LOXITANE dosage or by administration of antiparkinson drugs in usual dosage. Dystonic and dyskinetic reactions have occurred less frequently, but may be more severe. Dystonias include spasms of muscles of the neck and face, tongue protrusion, and oculogyric movement. Dyskinetic reaction has been described in the form of choreo-athetoid movements. These reactions sometimes require reduction or temporary withdrawal of LOXITANE dosage in addition to appropriate counteractive drugs.

Persistent Tardive Dyskinesia—As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movement of the tongue, face, mouth, or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia; antiparkinson agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Cardiovascular Effects: Tachycardia, hypotension, hypertension, light-headedness, and syncope have been reported.

A few cases of ECG changes similar to those seen with phenothiazines have been reported. It is not known whether these were related to loxapine administration.

Skin: Dermatitis, edema (puffiness of face), pruritus, and seborrhea have been reported with loxapine. The possibility of photosensitivity and/or phototoxicity occurring has not been excluded; skin rashes of uncertain etiology have been observed in a few patients during hot summer months.

Endocrine Effects: No endocrine abnormalities have been reported.

Anticholinergic Effects: Dry mouth, nasal congestion, constipation, and blurred vision have occurred; these are more likely to occur with concomitant use of antiparkinson agents.

Other Adverse Reactions: Nausea, vomiting, weight gain, weight loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, and polydipsia have been reported in some patients.

DOSAGE AND ADMINISTRATION

LOXITANE *loxapine succinate* is administered orally usually in divided doses two to four times a day. Daily dosage (in terms of base equivalents) should be adjusted to the individual patient's needs as assessed by the severity of symptoms and previous history of response to antipsychotic drugs. Initial dosage of 10 mg twice daily is recommended, although in severely disturbed patients initial dosage up to a total of 50 mg daily may be desirable. Dosage should then be increased fairly rapidly over the first seven to ten days until there is effective control of psychotic symptoms. The usual therapeutic and maintenance range is 60 mg to 100 mg daily. However, as with other antipsychotic drugs, some patients respond to lower dosage and others require higher dosage for optimal benefit. Daily dosage higher than 250 mg is not recommended. For maintenance therapy, dosage should be reduced to the lowest level compatible with symptom control; many patients have been maintained satisfactorily at dosages in the range of 20 mg to 60 mg daily.

HOW SUPPLIED

LOXITANE *loxapine succinate* is supplied in the following base equivalent strengths:

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Consultant in Health Economics, American Psychiatric Association

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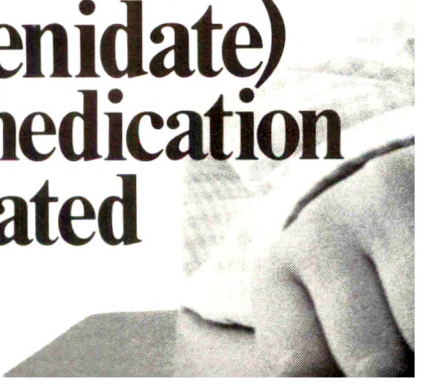
And side effects – insomnia and appetite loss – with Ritalin have occurred less frequently than with dextroamphetamine.[10,11]

Indeed, Ritalin is currently a drug of choice in many MBD situations,[10,12] and can prove to be an important element in many complete remedial programs for MBD.

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Drug treatment is not indicated for all children with MBD.

Dosage should be periodically interrupted. Often, these interruptions reveal some "stabilization" in the child's behavior even without medication, permitting a reduction in dosage and eventual discontinuance of drug therapy.

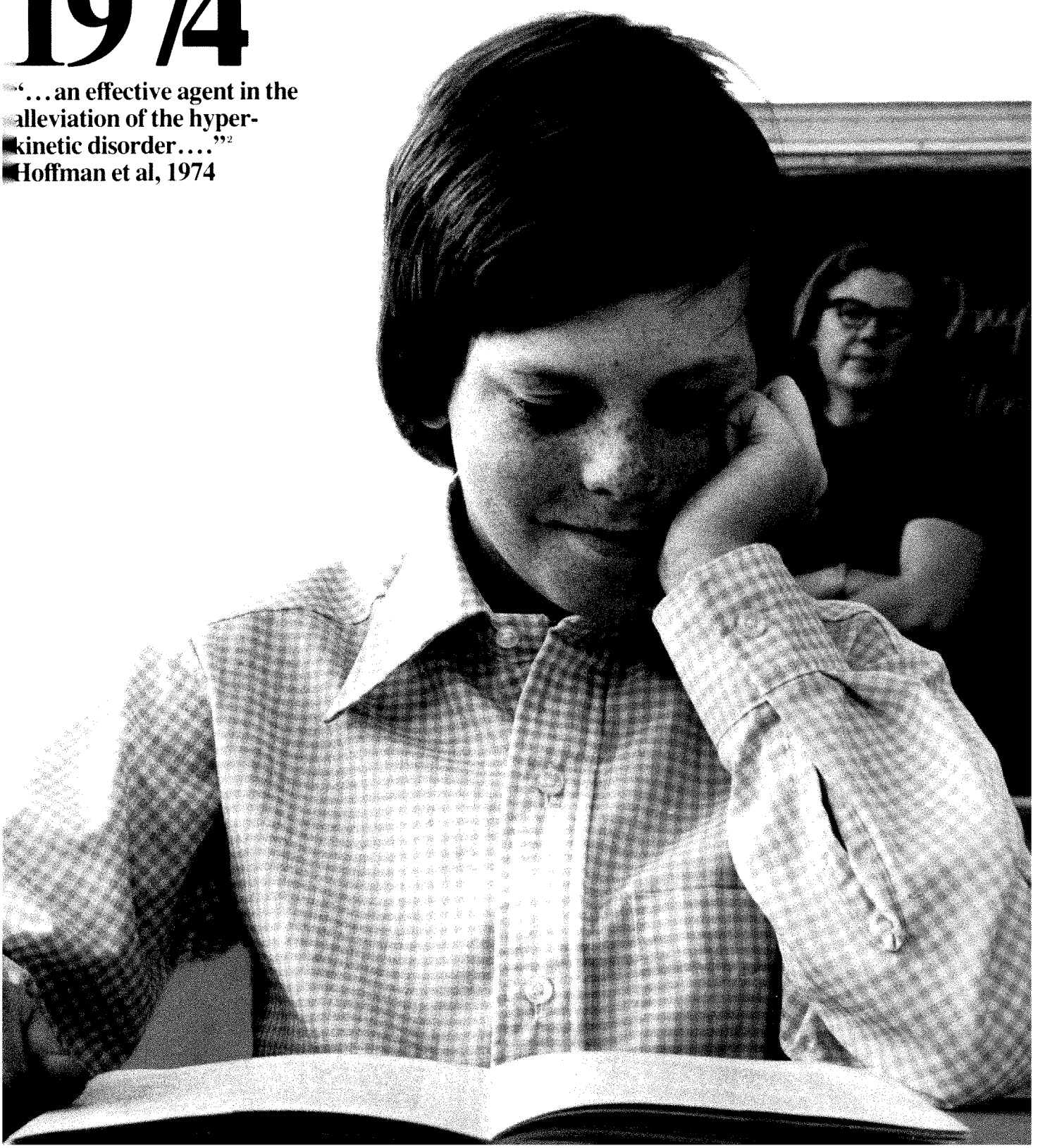
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the treatment of MBD

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"...an effective agent in the alleviation of the hyperkinetic disorder...."[2]
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TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over)

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-pak® blister units of 100.

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Consult complete product literature before prescribing.

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CONTROVERSIAL ISSUES IN HUMAN RELATIONS TRAINING GROUPS *edited by* **Kenneth T. Morris,** *Central Michigan Univ., Mount Pleasant, and* **Kenneth M. Cinnamon.** *Foreword by* **Mike Kanitz.** *(7 Contributors)* Controversial issues covered deal with such group dynamics variables as certification of group leaders, sex between members, necessary leader skills, necessary variables required for a successful group, the place of physical contact and structured experiences, screening of members, bias in group orientation and format, and whether these training groups are an art or a science. Each issue comprises one chapter with an editor's summary and synopsis section at the end. *'75, 168 pp., cloth-$9.75, paper-$5.95*

CLINICAL USE OF PSYCHOTHERAPEUTIC DRUGS (3rd Ptg.) *by* **Leo E. Hollister,** *Stanford Univ. School of Medicine, Palo Alto, California.* Clinical experience with the use of drugs in the treatment of patients is related to their pharmacological properties that bear most directly on their application to the treatment of emotional disorders in man. Each class of drugs is considered in relation to the chemical and pharmacological differences among its members, pharmacological properties and pharmacokinetics as they relate to their clinical use, specific clinical indications, general principles of use, use in combination with other drugs, and their side effects and toxicology. Antipsychotic, antimanic, antidepressant and antianxiety drugs are all closely examined. *'75, 192 pp., 11 il., 13 tables, $7.95*

LIFE STRESS AND ILLNESS *edited by* **E. K. Eric Gunderson,** *Univ. of California, San Diego, and* **Richard H. Rahe,** *Univ. of California, Los Angeles. (19 Contributors)* Relationships between stressful life events and various types of illness including mental illness and heart disease are examined in this book. Major emphasis is placed on the development of quantitative techniques for the measurement of life stress and the scaling of specific life events which have specific significance for the onset of disease. The physiological and psychological effects of severe stresses, such as prisoner of war confinement, are described, but the effects of milder stresses of everyday living are considered as well. The importance of cultural differences in determining the kinds of social stresses typically encountered in an individual's life are also illustrated. *'74, 276 pp., 25 il., 27 tables, $19.50*

CHILDREN'S EXPERIENCE WITH DEATH *by* **Rose Zeligs,** *Sherman Oaks, California.* This book shows how the child and adolescent is affected by his experience with death. Every chapter contains topical headings, a summary, and a list of references to make teaching and learning easier. Chapters cover developmental concepts of death, fear of death, the hospital experience of the dying child and adolescent, the handicapped child, suicide among children and adolescents, the death of a parent, and the influence of religion on his attitudes toward death. The author talks with children and listens to them, bringing a better understanding of children's ideas, attitudes, and feelings about death. *'74, 264 pp., $10.75*

SELF-DESTRUCTIVE BEHAVIOR *compiled and edited by* **Albert R. Roberts,** *Coppin State College, Baltimore, Maryland. (14 Contributors)* New insights concerning chronic risk-taking behavior and the heightening of coping mechanisms are provided in this book. Social, psychological, medical and psychiatric correlates of the most prevalent forms of self-destruction are all taken into account. Suicidal and subsuicidal behaviors such as the one-car accident, drug-related deaths, homicide followed by suicide, chronic alcoholism and cirrhosis of the liver, and cardiovascular diseases and diabetes mellitus exacerbated by compulsive overeating are described. In addition, the authors have made reference to the commonalities and causal links between many of these self-destructive acts. *'75, 232 pp., 9 il., 7 tables, $17.50*

INVOLUNTARY TREATMENT OF THE MENTALLY ILL: The Problem of Autonomy *by* **Michael Alfred Peszke,** *Univ. of Connecticut School of Medicine, Hartford.* This review puts the problem of involuntary treatment of mental illness into historical perspective and looks at it objectively, recognizing that mental illness cannot simply be legislated out of existence. The author suggests implementation for meeting the needs of the mentally ill based on the right of everyone to his own autonomy. Suggestions are proposed, leading to a situation in which no one in our society will be deprived of enlightened and appropriate treatment, while no one is deprived of freedom in a thoughtless or arbitrary fashion. *'75, 176 pp., 1 il., $10.75*

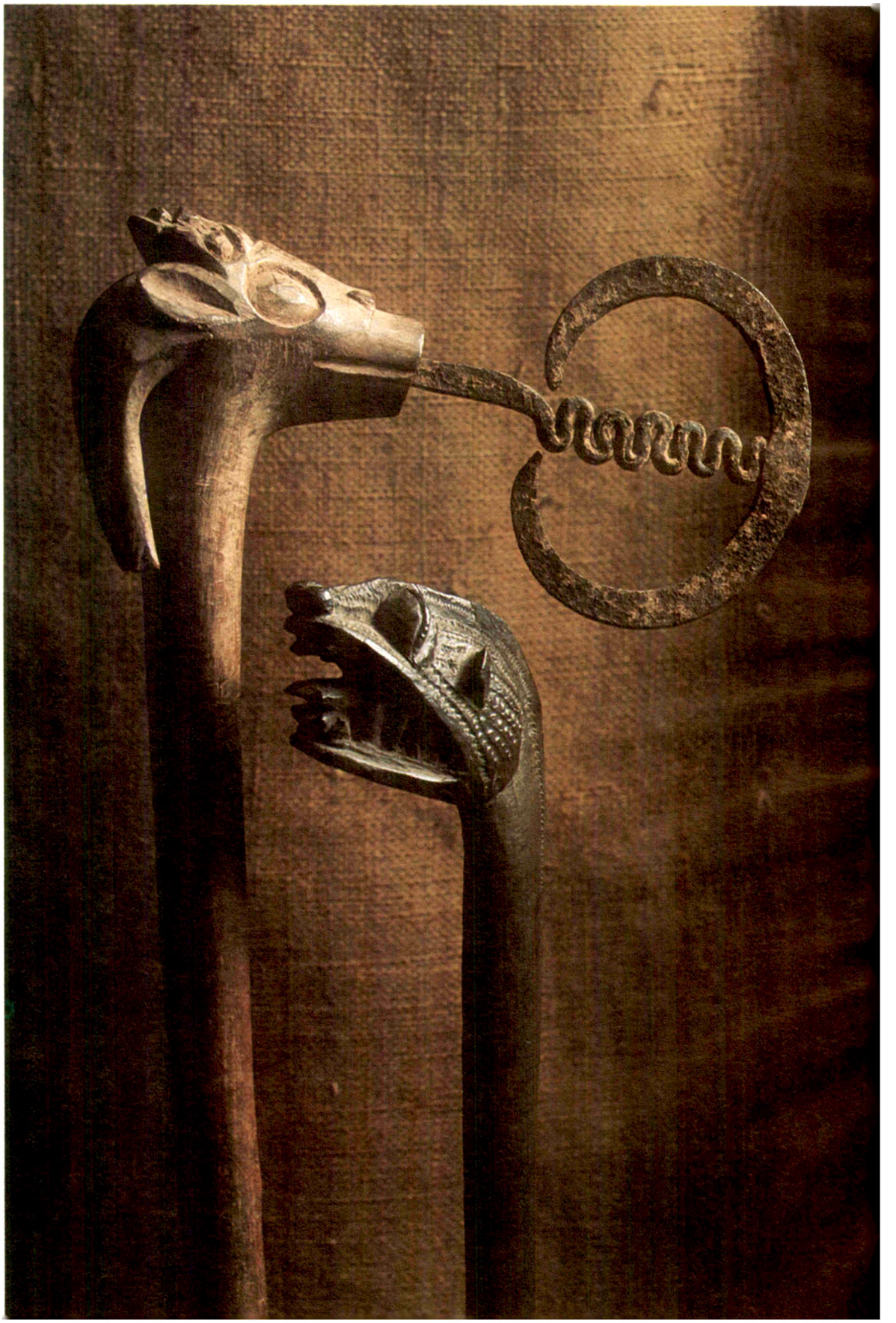
THE PROMISCUOUS TEENAGER *by* **Daniel T. Gianturco** *and* **Harmon L. Smith,** *both of Duke Univ., Durham, North Carolina.* Psychiatric guidelines are provided to understand why teenage sexual behavior is promiscuous or healthly. Teenage sexuality is viewed with the assumption that sexual activity involves important decision-making for a comprehensive cluster of interested and involved persons including teenagers, their parents and physicians. Ethical concepts are introduced to develop an integrated view of teenage sexual decision making. Sexual topics discussed include marriage, communal living, homosexuality, pregnancy, abortion, and contraception. Suggestions are included for management of the sexually disturbed teenager. *'74, 128 pp., 14 il., $8.50*

PSYCHIATRIC ASSESSMENT BY SPEECH AND HEARING BEHAVIOR *by* **Clyde L. Rousey,** *The Menninger Foundation, Topeka, Kansas. Foreword by* **J. Cotter Hirschberg.** *(18 Contributors)* Presentation of a theory of speech which has clinical relevance to the assessment of emotional health is the primary focus of this work. The studies cover such areas as diagnostic assessment of psychiatric patients, selection of trainees for residency programs in psychiatry, hearing problems in psychiatric patients, the phenomenon of speaking in tongues, early detection of educational problems resulting from emotional problems, factors affecting psychiatric diagnosis and treatment planning, and neurological indications in speech and hearing behavior. *'74, 392 pp., 12 tables, $18.75*

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Mankpo: Sceptres or staffs used by the Heviosso (lightning God) cult in medical ceremonies among the Ewe of Tongo and Fon of Dahomey. *Right,* staff with leopard's head, bared teeth and tongue (Ewe, Togo). *Left,* staff with ram's head—the sacred animal of the Heviosso—in the form of a serpent symbolizing the fire-spitting rainbow God of healing (Fon, Dahomey). From the collection of the Segy Gallery, New York City.

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Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudo-parkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

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It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9/74

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Help restore reality

- low dose, high potency phenothiazine useful for inpatient and outpatient treatment
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for management of disturbed behavior in schizophrenic and manic states

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Tablets	2,4,8,16 mg
REPETABS® Tablets 8 mg brand of repeat-action tablets	

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for maintenance or control of patients in hospital or home

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- average duration of effect is 6 hours

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CONTRAINDICATIONS TRILAFON is contraindicated in drug-associated central nervous system depression (barbiturates, alcohol, narcotics, analgesics, antihistamines). Perphenazine is contraindicated in the presence of existing blood dyscrasias, bone marrow depression and pre-existing liver damage, and in patients who are hypersensitive to perphenazine.

TRILAFON **Injection** should not be given to patients in coma or severely depressed states.

WARNINGS **Dosage in pregnancy:** Perphenazine should only be given to pregnant patients when, in the judgment of the physician, the potential benefits outweigh the possible risks.

Perphenazine can lower the convulsive threshold in susceptible individuals; it should be used with caution in patients with convulsive disorders. If the patient is being treated with an anticonvulsant agent, increased dosage of that agent may be required when perphenazine is used concomitantly.

Perphenazine should be used with caution in patients with psychic depression.

Perphenazine is not recommended for children under 12 years of age.

Perphenazine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery.

PRECAUTIONS As with any potent medication, patients receiving perphenazine should be under medical supervision, particularly if they are receiving high doses. Patients who have had any severe reaction to phenothiazines or to imipramine should be treated cautiously, under close medical supervision.

Although the following adverse reactions have not been reported in patients treated with perphenazine, the possibility that they might occur with TRILAFON should be considered: blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, eosinophilia); liver damage (biliary stasis); narrowing of the visual fields; pigmentation of the retina, cornea, or lens; cerebral edema; polyphagia; photophobia; hyperpyrexia.

If hypotension develops, levarterenol (norepinephrine) can be used, but not epinephrine, because epinephrine's action is blocked and partly reversed by perphenazine. Severe, acute hypotension has occurred with the use of phenothiazines and is of particular concern in patients with mitral insufficiency or pheochromocytoma.

A significant rise in body temperature may indicate an idiosyncratic reaction to perphenazine; treatment with perphenazine should be stopped if this occurs.

The antiemetic effect of perphenazine can obscure signs of toxicity due to overdosage of other drugs, or mask the symptoms of disease (eg, brain tumor or intestinal obstruction).

Contact dermatitis has been reported with a perphenazine solution; therefore, contact of hands or clothing by those handling perphenazine solutions should be avoided.

POTENTIATION Since phenothiazines can potentiate the central-nervous-system-depressant actions of opiates, antihistamines, barbiturates, and alcohol, less than the usual dosage of these agents is required when they are administered concomitantly with TRILAFON. Patients should be cautioned that their response to alcohol may be increased while they are being treated with TRILAFON.

Phenothiazines also potentiate the effects of atropine, heat, and phosphorus insecticides, and should be used with caution in persons exposed to these agents.

ADVERSE REACTIONS *Extrapyramidal reactions:* dystonia including protrusion, discoloration, aching and rounding of the tongue; tonic spasm of the masticatory muscles, tight feeling in the throat, slurred speech, dysphagia, oculogyric crisis, trismus, torticollis, retrocollis, muscle weakness, and aching and numbness of the limbs; akathisia; motor restlessness; dyskinesia, parkinsonism; hyperreflexia; and ataxia. The incidence and severity of these reactions usually increase with increased dosage, but have occurred in some patients receiving low dosage. Reduction in dosage or treatment with an antispasmodic agent will usually control extrapyramidal reactions. In some instances, however, these reactions may persist after discontinuation of treatment with perphenazine.

Persistent tardive dyskinesia: As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities. There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine, vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Allergic reactions: erythema, pruritus, urticaria, eczema, anaphylactoid reactions, and local and generalized edema. In extremely rare instances, individual idiosyncrasy or hypersensitivity to phenothiazines has resulted in cerebral edema, circulatory collapse, and death. Photosensitization, asthma, and exfoliative dermatitis have also occurred in patients treated with phenothiazines.

Autonomic reactions: blurred vision, dry mouth or salivation, nasal congestion, nausea, vomiting, hypertension, tachycardia, hypotension, anorexia, urinary frequency or incontinence, and constipation. Significant autonomic effects have been infrequent in patients receiving less than 24 mg. perphenazine daily.

Other reactions: endocrine disturbances (lactation, gynecomastia, galactorrhea, disturbances in the menstrual cycle), headaches, mild insomnia, altered cerebrospinal fluid proteins, ECG abnormalities, reactivation of psychosis, paradoxical excitement, paranoid-like reactions, catatonia, and systemic lupus erythematosus-like syndrome. Hypnotic effects appear to be minimal, particularly in patients who are permitted to remain active. The following adverse reactions, though rare, have also been reported to be associated with perphenazine treatment: agranulocytosis; jaundice; hyperpigmentation of the skin; grand mal convulsions; failure of ejaculation; hyperglycemia.

Side effects with intramuscular TRILAFON **Injection** have been infrequent and transient. Dizziness or significant hypotension after treatment with TRILAFON (perphenazine) **Injection** is a rare occurrence.

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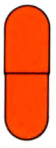
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Tofranil-**PM**®
imipramine **pamoate**

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In depression

Daily Dosage Chart



1 h.s.

A major reason for drug defaulting is multiple daily dosage schedules.

Ayd* reports that 70 percent of patients default on a q.i.d. regimen; 60 percent on t.i.d.; 30 percent on b.i.d., and only 7 percent on a single daily dose.

A single daily capsule that provides a full day's medication would logically assure better therapeutic results.

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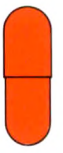
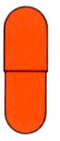
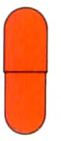
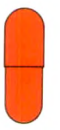
After therapy is initiated with the 75-mg. capsule, the 150-mg. capsule is the dose level at which optimum response is usually obtained.

Please review the prescribing information summarized on the back of this page.

*Ayd, F.J., Jr. (Ed.): Int. Drug Ther. Newslett. 7(9/10):35-50 (Nov./Dec.) 1972.

Tofranil-PM®
imipramine **pamoate**

One capsule lasts from bedtime to bedtime.

Initial Dose		For Maintenance Therapy			
Starting Dose	Usual Optimum Response Dose	A Full Range to Choose From*			
					
75 mg.	150 mg.	150 mg.	125 mg.	100 mg.	75 mg.

*Each capsule contains imipramine pamoate equivalent to 150, 125, 100 or 75 mg. imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;

—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;

—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;

—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;

—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response.If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks. Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
(B) 98-146-840-A(9/75) 667120

For complete details, including dosage and administration, please refer to the full prescribing information.

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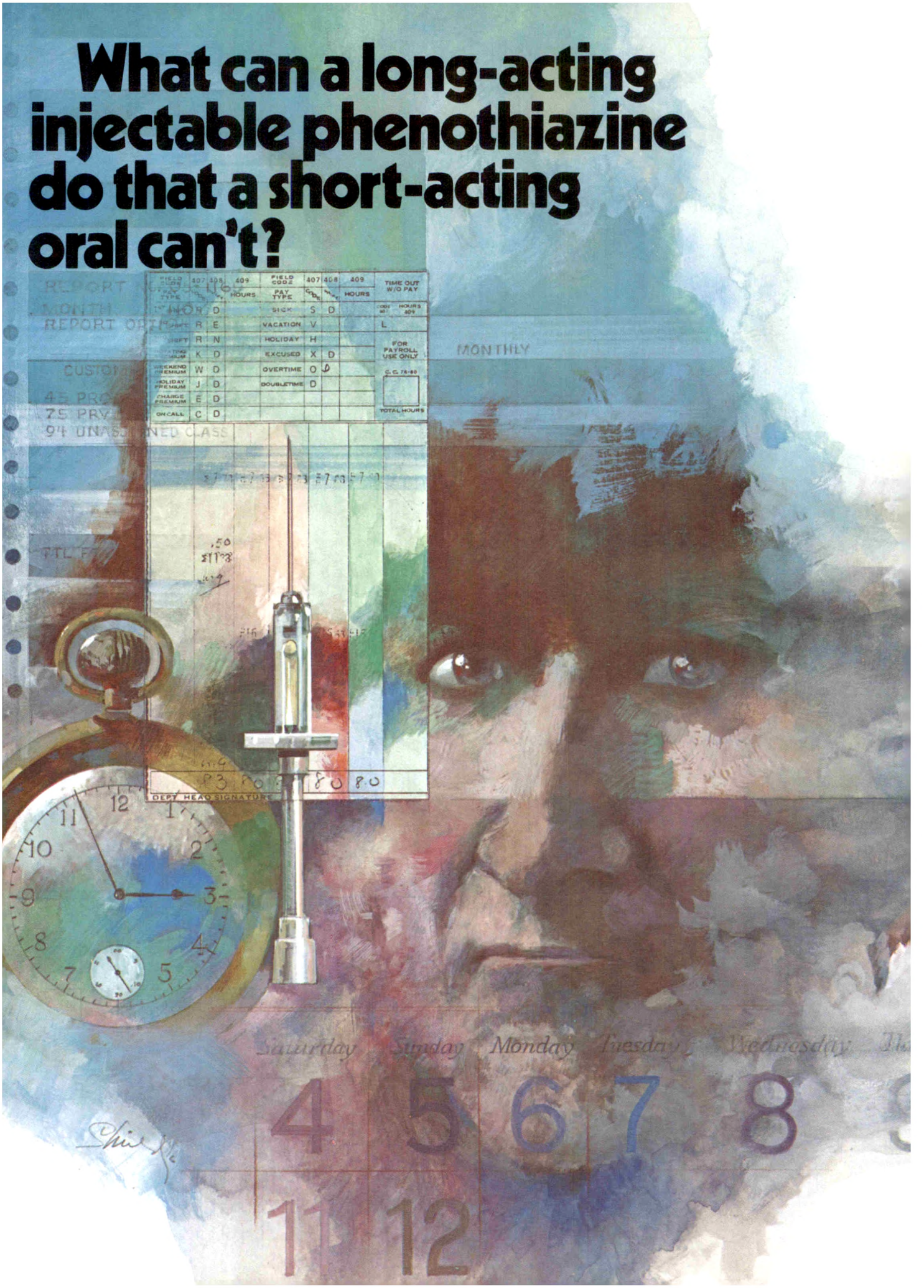
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It can save time, it can save money, and it can even save people – by reducing readmissions.

Time Prolixin Decanoate (Fluphenazine Decanoate Injection), with duration of action that may last up to 4 weeks or longer in patients on maintenance therapy, can effect important savings in nursing time.

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2¼ hrs. x 28 days = 63 hours of nursing time every 28 days	= 1 hr., 10 minutes nursing time in 28 days
NURSING TIME SAVED IN 28 DAYS	61 hours and 50 minutes or more than 7½ eight-hour working days

*Adapted from Platt R: Br J Social Psychiatry 2: 187-191, 1968.

Money Martin and Townend[1], who found from their study of Prolixin Decanoate that 15 of 39 patients could be maintained on 25 mg. every 4 weeks, also note that on this basis "a year's maintenance on chlorpromazine represents the order of 110 grams of phenothiazine as opposed to 0.33 gram of fluphenazine." Translating this into dollars shows the following potential hospital savings on the basis of patient population:

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Chlorpromazine (SKF brand) 110 grams	8 oz. concentrate 100 mg./ml.†	$48.75*	$24,375
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SAVINGS		$25.58 patient, year	$12,790 500 pts/yr.

* Calculated from prices published in 1975 Red Book.
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† With tablets, the annual cost is even greater.

People The controlled drug delivery system of Prolixin Decanoate helps get schizophrenic patients out of the hospital and helps them stay out. It promotes continuity of therapy—reducing outpatient drug defaulting from approximately 50% with oral medications according to one report[2] to about 16% according to another report[3].

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may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

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References:
1. Martin ICA and Townend RA: Brit J Psychiat 124: 173-6, 1974.
2. Goldberg HL, DiMascio A, Chaudhary B: Psychosomatics 11: 173-177, 1970.
3. Medical World News, February 11, 1972, p. 58H.
4. Denham J and Adamson L: Can Psychiat Assoc J 18: 235-7, 1973.

For product brief summary see following page.

H425-001R

PROLIXIN DECANOATE®

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may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia, liver damage or renal insufficiency, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

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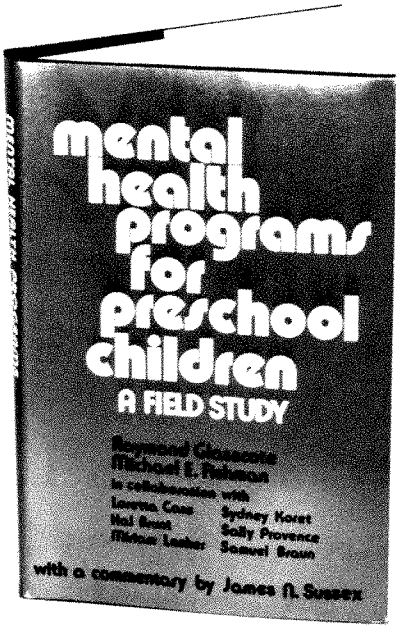
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This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



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The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline (primidone) for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards — used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate—As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail— A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

Ayerst®

Mysoline® (primidone)

Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

7538

Mysoline® (primidone)

may be the start of a better life for the epileptic

initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

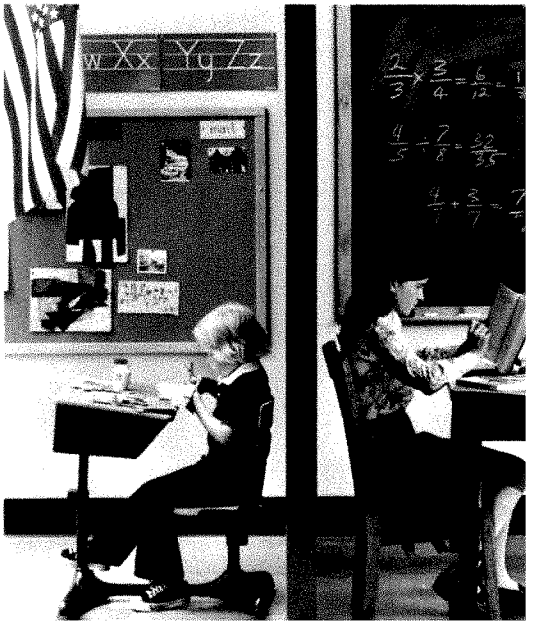
In patients already receiving other anticonvulsants MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets* — No. 430 — Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431 — Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension* — No. 3850 — Each 5 cc. (tea spoonful) contains 250 mg. of primidone, in bottles of 8 fluid ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. 37:49 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. 68:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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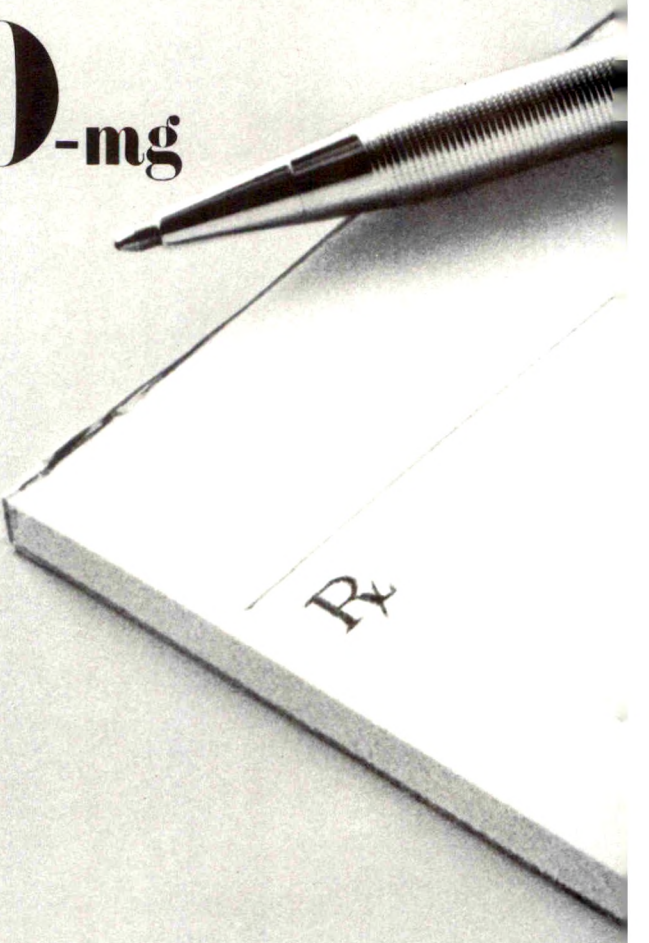
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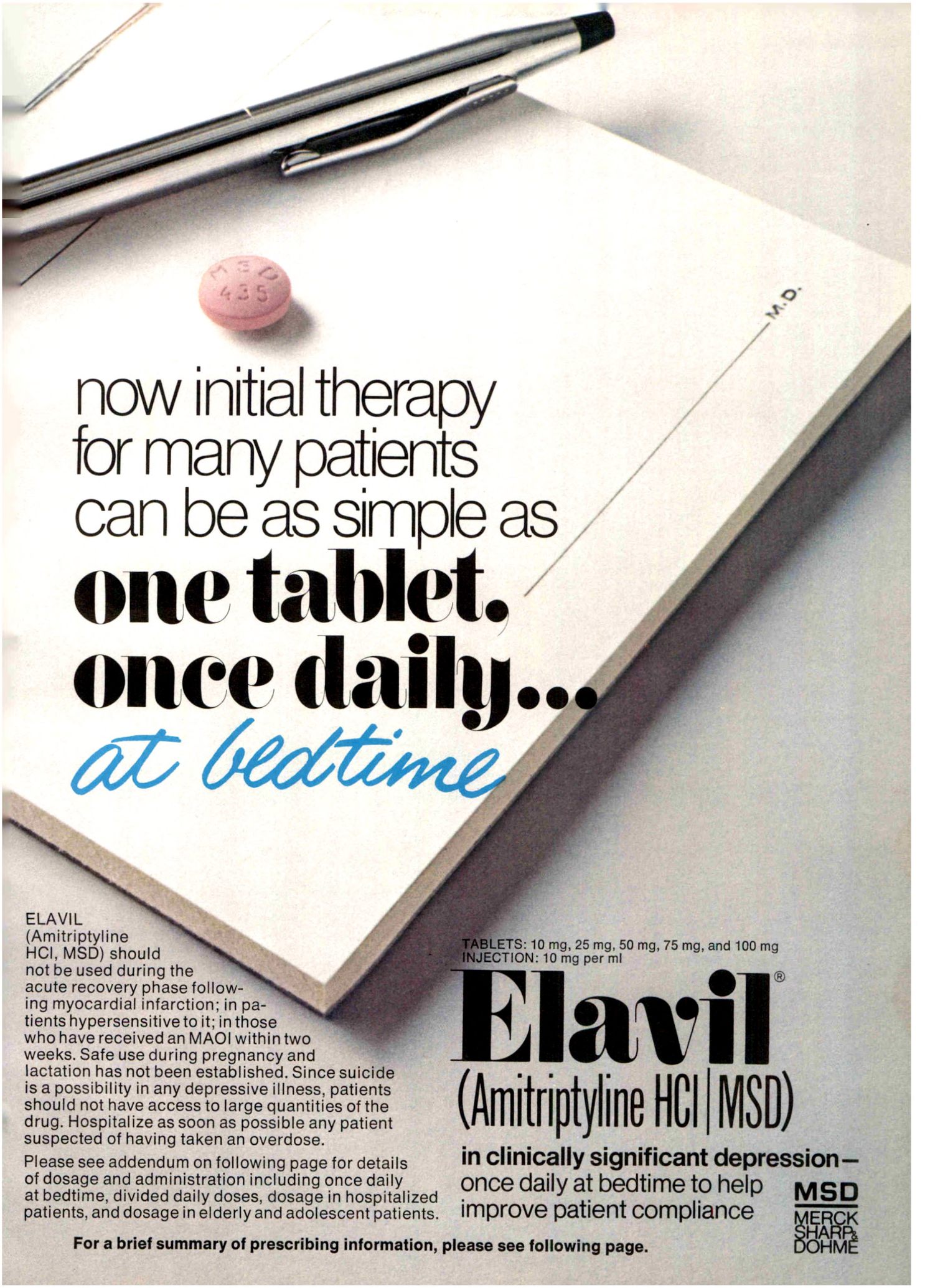
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to further simplify once-daily

h.s.

therapy





now initial therapy for many patients can be as simple as **one tablet, once daily...** *at bedtime*

ELAVIL (Amitriptyline HCl, MSD) should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks. Safe use during pregnancy and lactation has not been established. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

Please see addendum on following page for details of dosage and administration including once daily at bedtime, divided daily doses, dosage in hospitalized patients, and dosage in elderly and adolescent patients.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg
INJECTION: 10 mg per ml

Elavil®
(Amitriptyline HCl | MSD)

in clinically significant depression— once daily at bedtime to help improve patient compliance

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see following page.

NEW TABLETS

Elavil®

(Amitriptyline HCl | MSD)

75 mg and 100 mg

to further simplify once-daily

h.s.

therapy

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor or within at least 14 days following the discontinuance of a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: ***Note:*** Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. ***Cardiovascular:*** Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. ***CNS and Neuromuscular:*** Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. ***Anticholinergic:*** Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. ***Allergic:*** Skin rash, urticaria, photosensitization, edema of face and tongue. ***Hematologic:*** Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. ***Gastrointestinal:*** Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). ***Endocrine:*** Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. ***Other:*** Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. ***Withdrawal Symptoms:*** Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

addendum

MSD
MERCK SHARP & DOHME

Elavil®

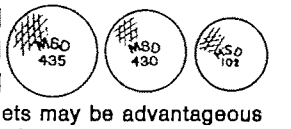
(Amitriptyline HCl | MSD)

Five tablet strengths for differing patient needs. The variety of dosage strengths available with ELAVIL makes possible great prescribing flexibility.

Once-a-day dosage at bedtime is an appropriate way to start—and maintain—many patients on therapy. The simplicity of this regimen helps improve patient compliance. ELAVIL may also be prescribed in divided daily doses.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

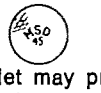
100 mg
75 mg
50 mg



These tablets may be advantageous when initiating therapy with once-daily bedtime dosage in adult outpatients. The usual starting dosage is 75 mg daily. Therapy may be initiated with 50 to 100 mg daily. This may be increased by 25 or 50 mg as necessary in the bedtime dose to a total of 150 mg per day.

These tablets may also be convenient for many hospitalized patients. These patients may need 100 mg a day initially which can be increased gradually to 200 mg a day if necessary. A small number of such patients may need as much as 300 mg a day.

25 mg



This tablet may prove useful when initiating therapy with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime doses.

10 mg



Because lower doses are generally recommended for adolescents and elderly patients, the 10-mg tablets may be most serviceable. Ten mg three times a day with 20 mg at bedtime may be satisfactory in adolescent and elderly patients who do not tolerate higher doses.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

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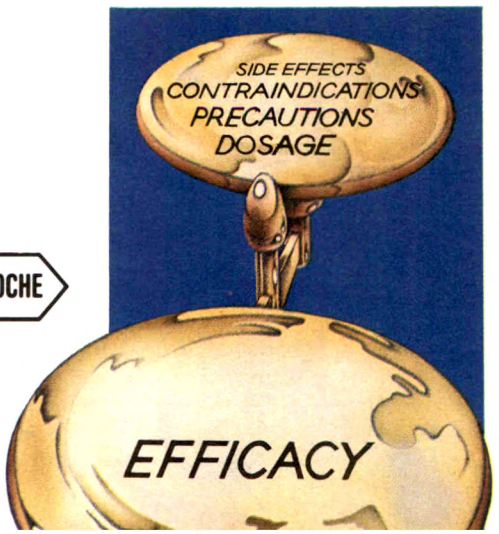
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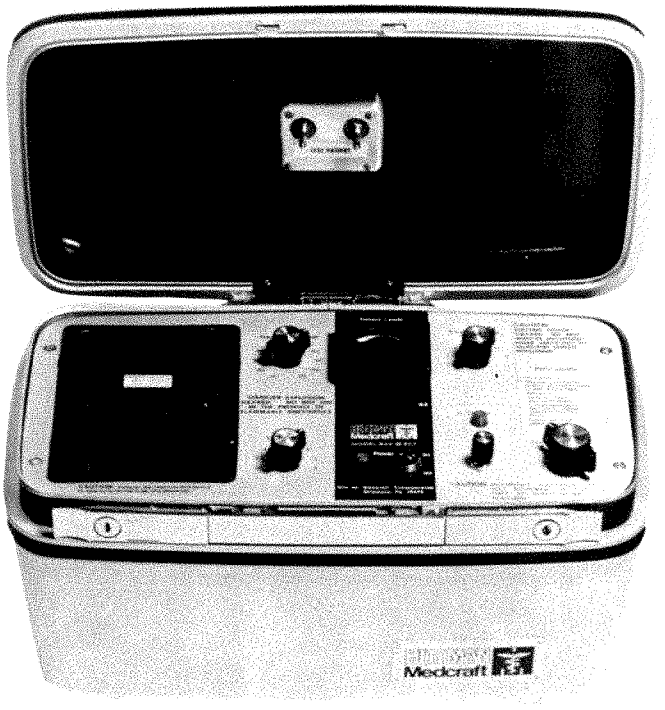
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In this issue

Bertram S. Brown on
The Life of Psychiatry

Also in this issue

Paul Chodoff on
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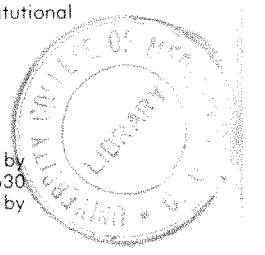
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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968, pp 1–12

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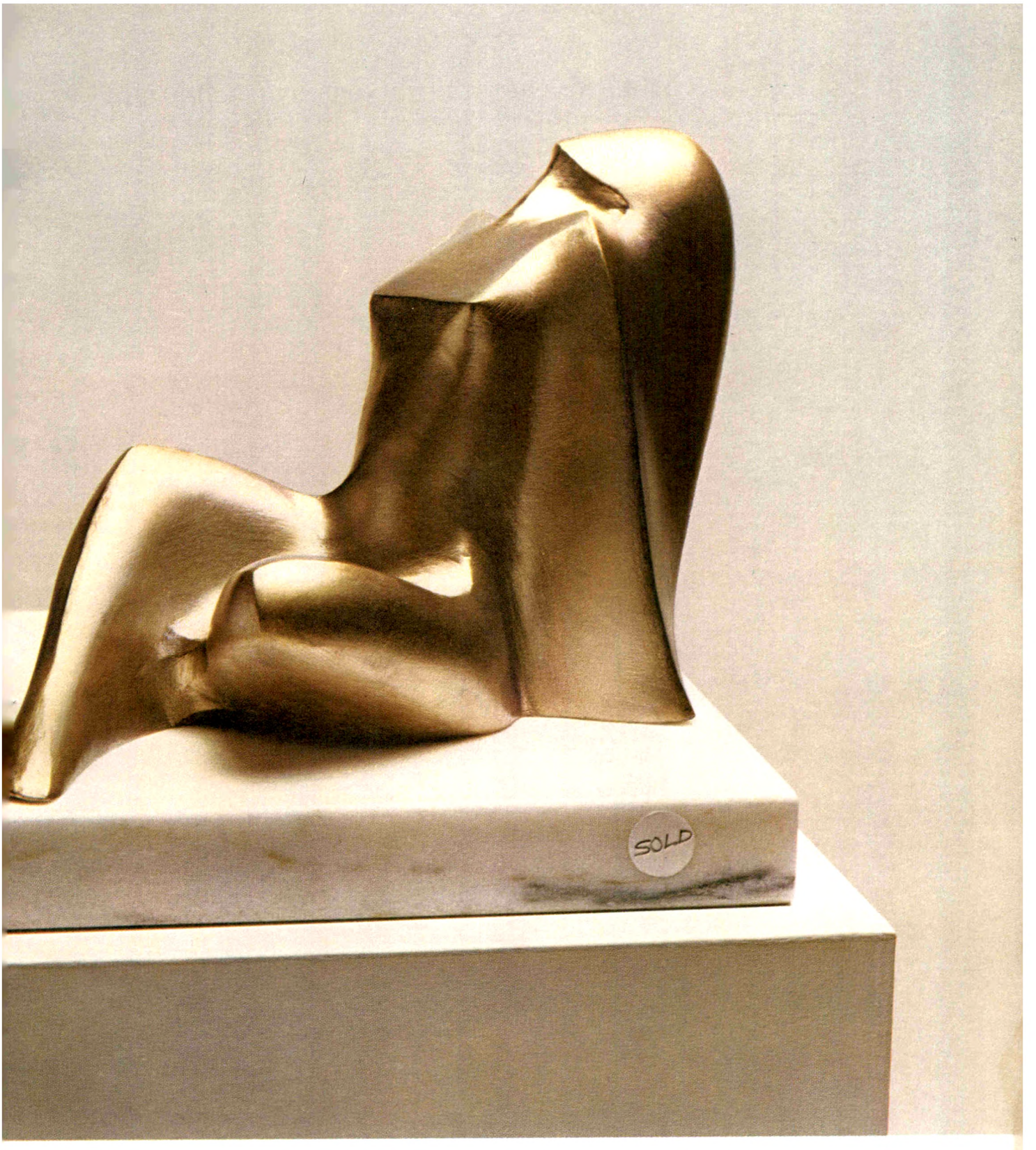
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Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy*—Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration—As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg. of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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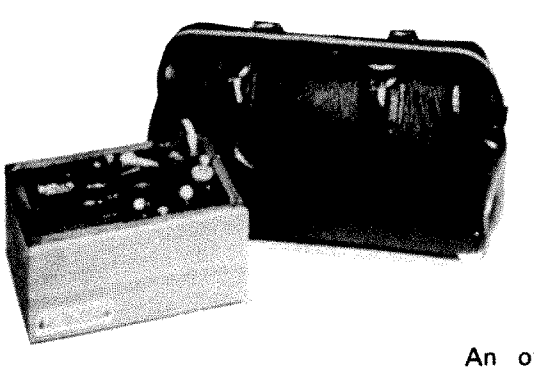
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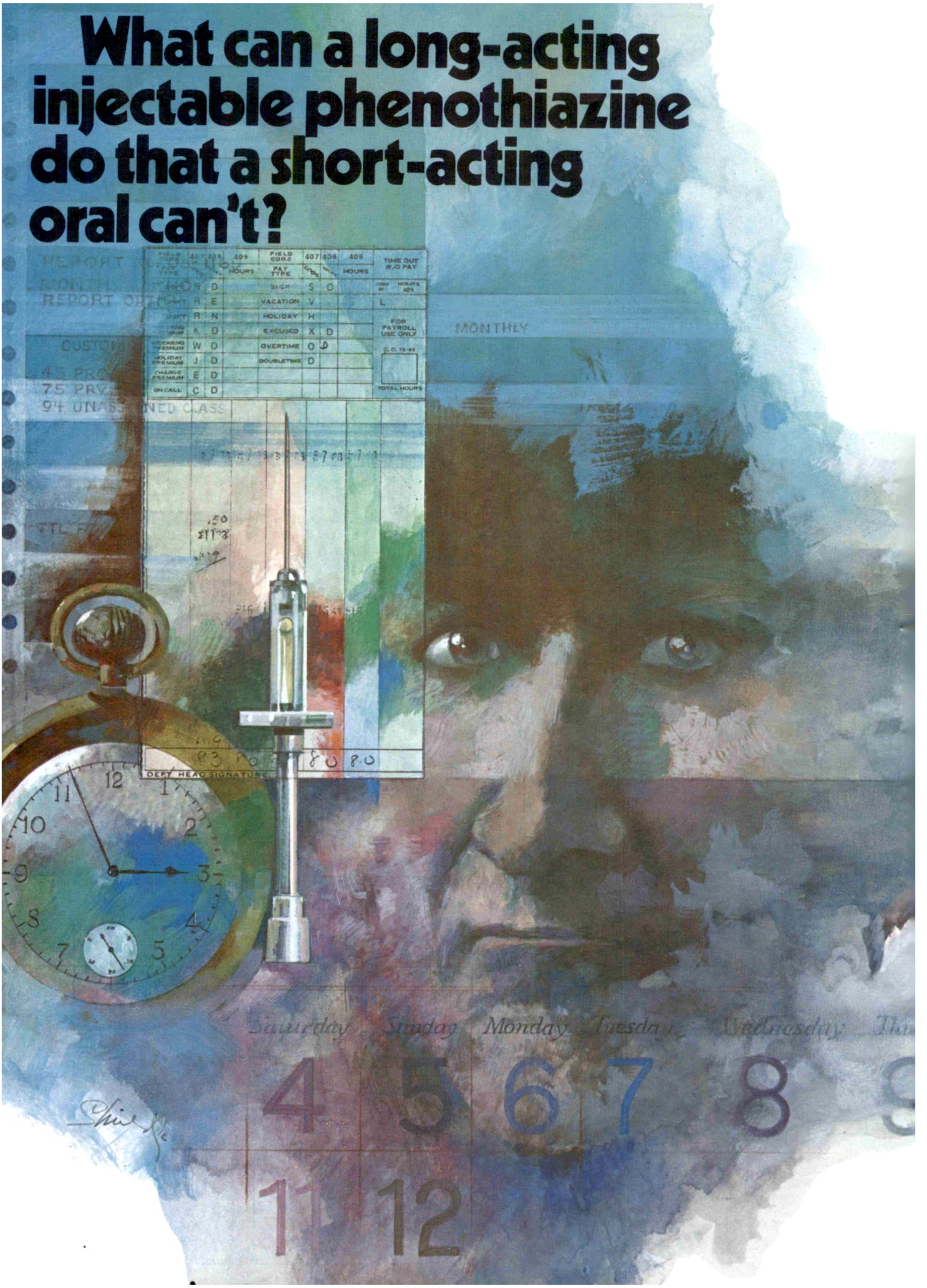
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What can a long-acting injectable phenothiazine do that a short-acting oral can't?



It can save time, it can save money, and it can even save people – by reducing readmissions.

Time Prolixin Decanoate (Fluphenazine Decanoate Injection), with duration of action that may last up to 4 weeks or longer in patients on maintenance therapy, can effect important savings in nursing time.

Approximate Staff Time Required to Medicate 18 Schizophrenic Patients*

8 a.m. ¾ hr. 1 p.m. ¾ hr. 5 p.m. ¾ hr.	1 injection every 28 days for most patients
= 2¼ hours nursing time	4 minutes required for each injection
2¼ hrs. x 28 days = 63 hours of nursing time every 28 days	= 1 hr., 10 minutes nursing time in 28 days
NURSING TIME SAVED IN 28 DAYS	61 hours and 50 minutes or more than 7½ eight-hour working days

*Adapted from Platt R. Br J Social Psychiatry 2: 187-191, 1968.

Money Martin and Townend[1], who found from their study of Prolixin Decanoate that 15 of 39 patients could be maintained on 25 mg. every 4 weeks, also note that on this basis " a year's maintenance on chlorpromazine represents the order of 110 grams of phenothiazine as opposed to 0.33 gram of fluphenazine." Translating this into dollars shows the following potential hospital savings on the basis of patient population:

Agent and amount/year	Form	Cost/patient/year	Cost/500 patients/year
Chlorpromazine (SKF brand) 110 grams	8 oz. concentrate 100 mg./ml.†	$48.75*	$24,375
Prolixin Decanoate .33 grams	5 ml. vials 25 mg./ml.	23.17**	11,585
SAVINGS		$25.58 patient, year	$12,790 500 pts/yr.

* Calculated from prices published in 1975 Red Book
** Calculated from Squibb 1975 price catalogue
† With tablets, the annual cost is even greater.



People The controlled drug delivery system of Prolixin Decanoate helps get schizophrenic patients out of the hospital and helps them stay out. It promotes continuity of therapy—reducing outpatient drug defaulting from approximately 50% with oral medications according to one report[2] to about 16% according to another report[3].

It reduces the number and length of rehospitalizations. In one study of 103 patients maintained on injections of long-acting fluphenazines, total hospital readmissions for a year were cut from 191 to 50 and inpatient time from 8,713 days to 1,335 days[4].

It facilitates return to a productive life. A 12-month followup of 103 discharged patients revealed 77% in full-time employment or household duties and only 23% unemployed[4].

PROLIXIN DECANOATE®

(Fluphenazine Decanoate Injection)

may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

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References:
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For product brief summary see following page.

H425-001R

PROLIXIN DECANOATE®

(Fluphenazine Decanoate Injection)

may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia, liver damage or renal insufficiency, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred, and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

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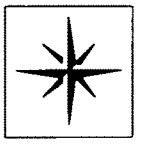
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LIBRIUM®
(chlordiazepoxide HCl)
5 mg, 10 mg, 25 mg capsules

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.
Contraindications: Patients with known hypersensitivity to the drug.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported. Use of any drug in pregnancy, lactation or in women of childbearing age requires that its potential benefits be weighed against its possible hazards.
Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.
Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.
Supplied: Librium® Capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritabs® Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

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SAFETY. IT'S A QUESTION OF BALANCE.

In choosing a tranquilizer for the patient whose excessive anxiety is disrupting psychotherapy there are a number of things to consider. One is effectiveness. A second is safety.

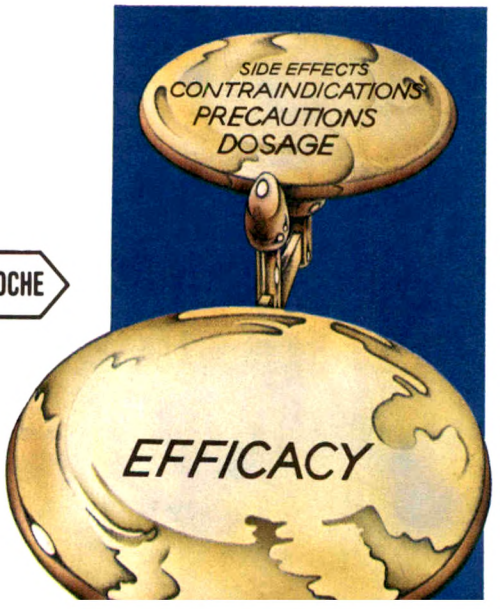
The first is more easily established. The second may be ascertained by comparison. In other words, with safety there is the question of balancing a drug's potential benefits against any potential risks.

When the antianxiety agent you are considering is Librium, you should know that it has a wide margin of safety.

The most common side effects of Librium are dose-related and thus largely avoidable. There appears to be a low incidence of drug dependence and tolerance. Librium rarely has any adverse effects on the cardiovascular or respiratory systems, and there are relatively few cases of known toxicity attributable to Librium.

You already know that Librium is effective. But you should also know that Librium, used in proper dosage, rarely interferes with mental acuity and thus is unlikely to interfere with the psychotherapeutic process. In addition, Librium is used concomitantly with many primary medications.

Librium. All in all, a very good balance.



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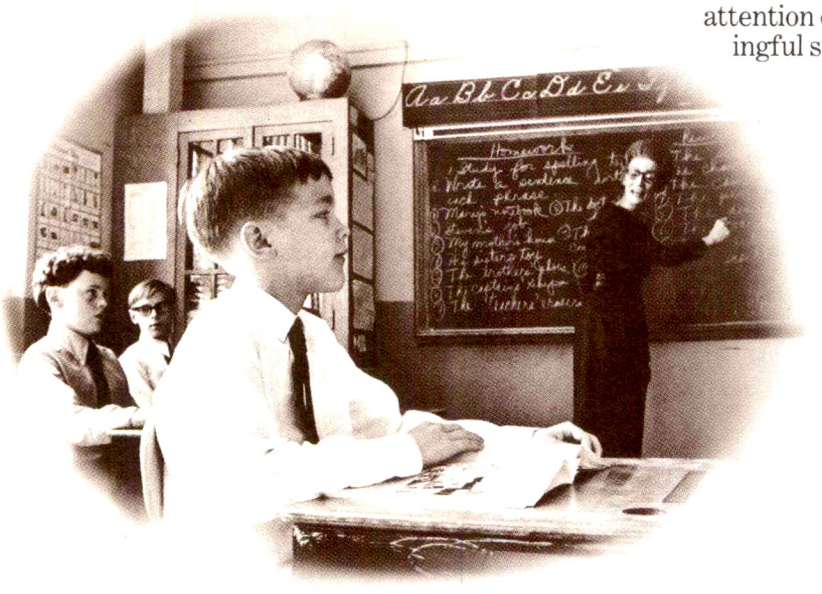
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Please see summary of product information on opposite page.

Tested by time and experience i

1962

"...a considerable decrease of hyperactivity...."[1]
Knobel, 1962



Over a decade of controlled studies and clinical experience has shown the effectiveness of Ritalin in reducing the hyperactivity,[1-3] distractibility,[1,4,5] and disorganized behavior[1-8] in the MBD child.

By lessening the effects of motor and attentional disorders, Ritalin can help the MBD child to better focus his attention on meaningful stimuli and thus can often improve cognition and promote learning.[6,9]

And side effects – insomnia ar appetite loss – with Ritalin have occurred less frequently than with dextroamphetamine.[10,11]

Indeed, Ritalin is currently a drug of choice in many MBD situations,[10,12] and can prove to be an important element in many complete remedial programs for MBD.

Therapy with Ritalin should b undertaken only after a medical diagnosis of MBD has been made. Drug treatment is not indicated for all children with MBD.

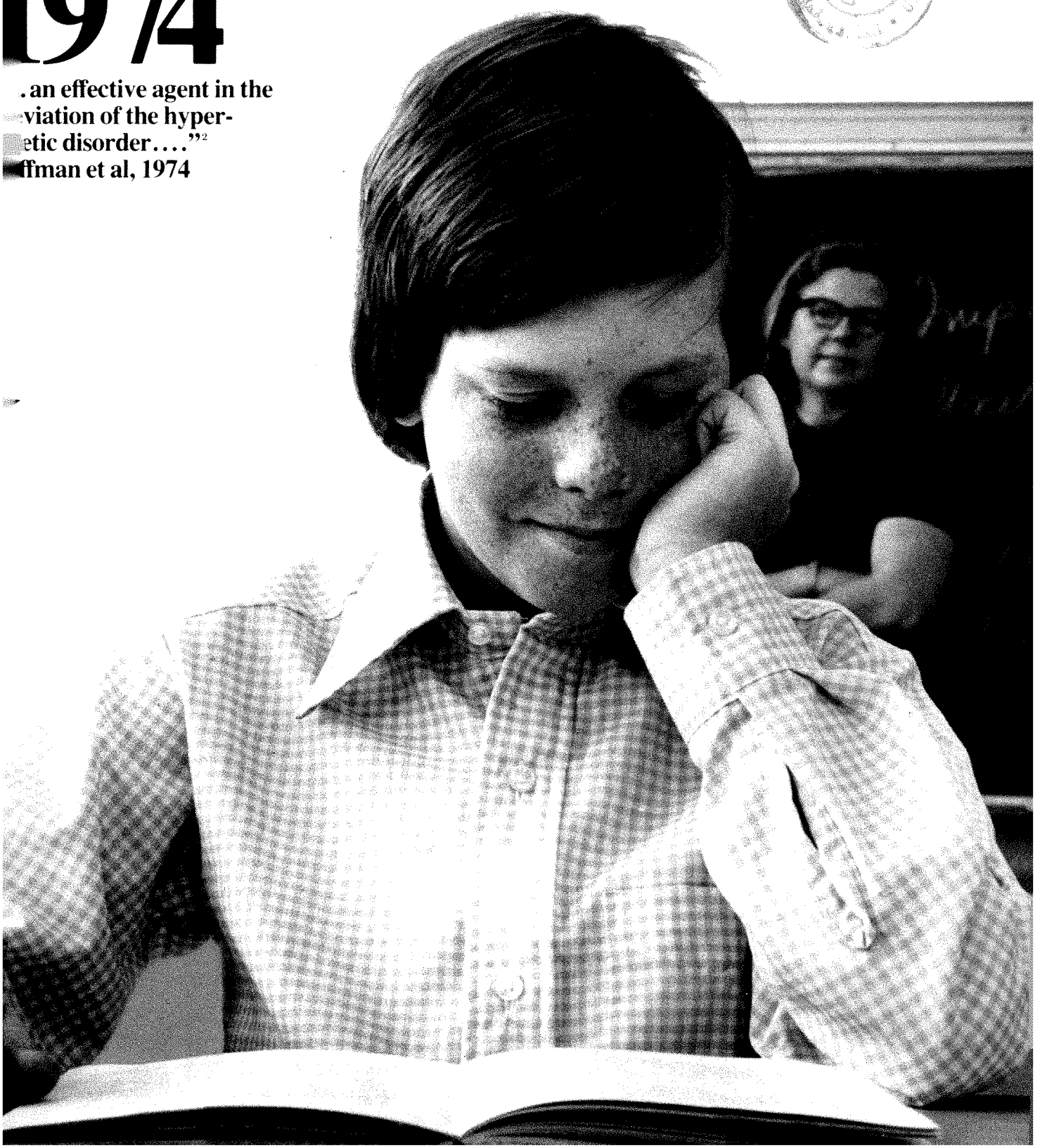
Dosage should be periodically interrupted. Often, these interruptions reveal some "stabilization" in the child's behavior even without medication, permitting a reduction in dosage and eventual discontinuance of drug therapy.

Ritalin®
(methylphenidate)
Only when medication is indicated

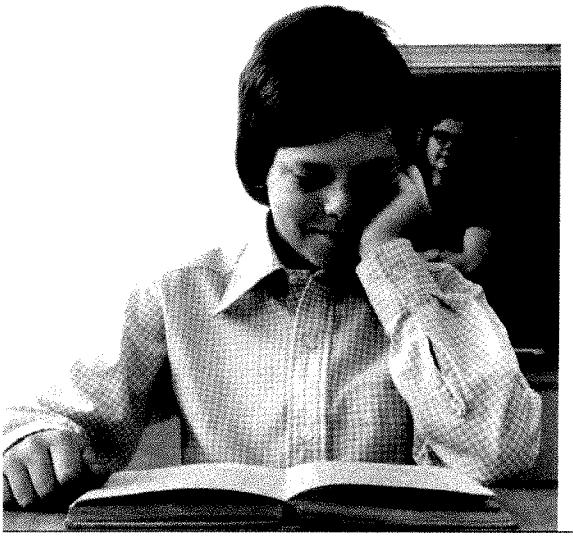
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ffman et al, 1974



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TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronic ly abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over)

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

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David K. Reynolds

Morita psychotherapy, a Buddhist-based Japanese therapy for the treatment of neuroses, is currently being practiced in its original in-patient form in several hospitals in Japan. For the first time, we have in this book, a full-length description in English of a fundamentally Eastern approach to man's neurotic tendencies, treatment, and cure.

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In depression

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A major reason for drug defaulting is multiple daily dosage schedules.

Ayd* reports that 70 percent of patients default on a q.i.d. regimen; 60 percent on t.i.d.; 30 percent on b.i.d., and only 7 percent on a single daily dose.

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Please review the prescribing information summarized on the back of this page.

*Ayd, F.J., Jr. (Ed.): Int. Drug Ther. Newslett. 7(9/10):35-50 (Nov./Dec.) 1972.

Tofranil-**PM**®
imipramine **pamoate**

One capsule
lasts from bedtime
to bedtime.

Initial Dose

For Maintenance Therapy

Starting Dose	Usual Optimum Response Dose	A Full Range to Choose From*			
					
75 mg.	150 mg.	150 mg.	125 mg.	100 mg.	75 mg.

*Each capsule contains imipramine pamoate equivalent to 150, 125, 100 or 75 mg. imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
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For complete details, including dosage and administration, please refer to the full prescribing information.

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Basic tools of primitive psychiatry



Mankpo: Sceptres or staffs used by the Heviosso (lightning God) cult in medical ceremonies among the Ewe of Tongo and Fon of Dahomey. *Right,* staff with leopard's head, bared teeth and tongue (Ewe, Togo). *Left,* staff with ram's head—the sacred animal of the Heviosso—in the form of a serpent symbolizing the fire-spitting rainbow God of healing (Fon, Dahomey). From the collection of the Segy Gallery, New York City.

Basic tool of Western psychiatry

Thorazine®
(chlorpromazine, SK&F)

Tablets: 25 and 50 mg of the HCl

- 'Thorazine' controls psychotic symptoms
- Especially useful in agitated, violent or anxious schizophrenic patients
- Unsurpassed clinical experience
- 18 convenient dosage forms and strengths

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudo-parkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only). Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg., in bottles of 50; in Single Unit Packages of 100 (intended for institutional use only). Injection, 25 mg./ml.; Syrup, 10 mg./5 ml.; Suppositories, 25 mg. and 100 mg.; Concentrate (intended for institutional use only), 30 mg./ml. and 100 mg./ml.

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Smith Kline & French Laboratories
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Philadelphia, Pa.

For effective management of schizophrenic symptoms.

for clinically significant depression. . .

Merck Sharp & Dohme introduces

75- and 100-mg

TABLETS

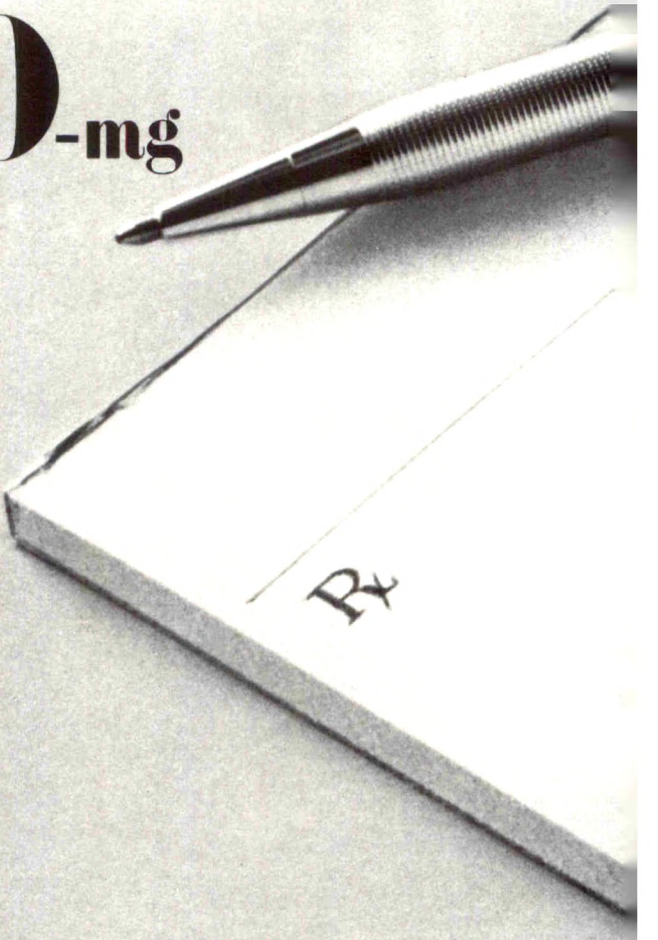
Elavil®

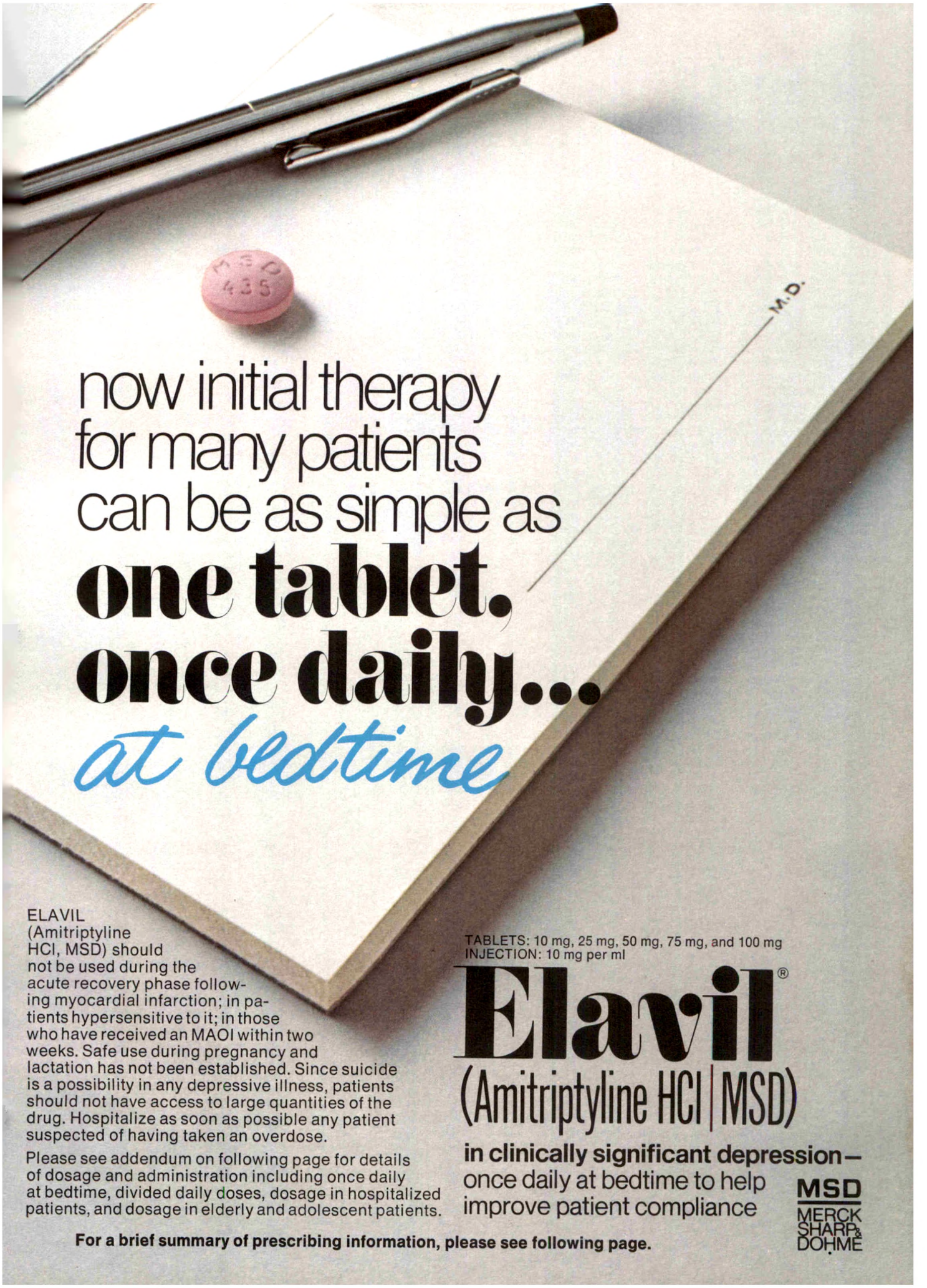
(Amitriptyline HCl | MSD)

to further simplify once-daily

h.s.

therapy





now initial therapy for many patients can be as simple as **one tablet, once daily...** *at bedtime*

ELAVIL (Amitriptyline HCl, MSD) should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks. Safe use during pregnancy and lactation has not been established. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

Please see addendum on following page for details of dosage and administration including once daily at bedtime, divided daily doses, dosage in hospitalized patients, and dosage in elderly and adolescent patients.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg
INJECTION: 10 mg per ml

Elavil®
(Amitriptyline HCl | MSD)

in clinically significant depression— once daily at bedtime to help improve patient compliance

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see following page.

NEW TABLETS

Elavil®

(Amitriptyline HCl | MSD)

75 mg and 100 mg

to further simplify once-daily

h.s.

therapy

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor or within at least 14 days following the discontinuance of a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramical symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

addendum

MSD
MERCK SHARP & DOHME

Elavil®

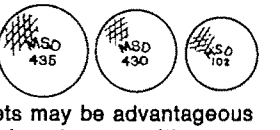
(Amitriptyline HCl | MSD)

Five tablet strengths for differing patient needs. The variety of dosage strengths available with ELAVIL makes possible great prescribing flexibility.

Once-a-day dosage at bedtime is an appropriate way to start—and maintain—many patients on therapy. The simplicity of this regimen helps improve patient compliance. ELAVIL may also be prescribed in divided daily doses.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

100 mg
75 mg
50 mg



These tablets may be advantageous when initiating therapy with once-daily bedtime dosage in adult outpatients. The usual starting dosage is 75 mg daily. Therapy may be initiated with 50 to 100 mg daily. This may be increased by 25 or 50 mg as necessary in the bedtime dose to a total of 150 mg per day.

These tablets may also be convenient for many hospitalized patients. These patients may need 100 mg a day initially which can be increased gradually to 200 mg a day if necessary. A small number of such patients may need as much as 300 mg a day.

25 mg



This tablet may prove useful when initiating therapy with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime doses.

10 mg



Because lower doses are generally recommended for adolescents and elderly patients, the 10-mg tablets may be most serviceable. Ten mg three times a day with 20 mg at bedtime may be satisfactory in adolescent and elderly patients who do not tolerate higher doses.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Schering

Out of touch



but not beyond reach...

Help restore reality

- low dose, high potency phenothiazine useful for inpatient and outpatient treatment
- helps orient psychiatric patients to their surroundings
- effective with minimal sedation, usually produces little lethargy or drowsiness

Trilafon®
brand of
perphenazine, NF
for management of disturbed behavior in schizophrenic and manic states

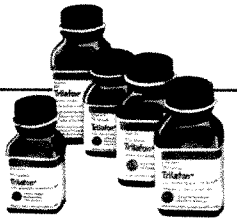
Versatile Dosage	
Injection	5 mg/ml, 1 ml ampul
Concentrate	16 mg/5 ml, 120 ml bottle
Tablets	2,4,8,16 mg
REPETABS® Tablets 8 mg brand of repeat-action tablets	

Please see next page for prescribing information.



Trilafon®
brand of
perphenazine, NF

For management of schizophrenic and manic disorders in psychiatric hospitals, mental health clinics, and with outpatient treatment



BENEFITS

TABLETS
for maintenance or control of patients in hospital or home

- generally improve cooperation and communication
- decrease need for custodial care, hasten discharge



CONCENTRATE
for maintenance or control of uncooperative in-hospital patients

- easily mixes with most liquids
- ensures easy, steady, certain administration



INJECTION
for emergency control of uncooperative in-hospital patients

- usually takes effect in 10 minutes
- average duration of effect is 6 hours

TRILAFON® Tablets
brand of perphenazine, NF

REPETABS® Tablets
Concentrate
Injection

CONTRAINDICATIONS TRILAFON is contraindicated in drug-associated central nervous system depression (barbiturates, alcohol, narcotics, analgesics, antihistamines). Perphenazine is contraindicated in the presence of existing blood dyscrasias, bone marrow depression and pre-existing liver damage, and in patients who are hypersensitive to perphenazine.

TRILAFON **Injection** should not be given to patients in coma or severely depressed states.

WARNINGS **Dosage in pregnancy:** Perphenazine should only be given to pregnant patients when, in the judgment of the physician, the potential benefits outweigh the possible risks.

Perphenazine can lower the convulsive threshold in susceptible individuals; it should be used with caution in patients with convulsive disorders. If the patient is being treated with an anticonvulsant agent, increased dosage of that agent may be required when perphenazine is used concomitantly.

Perphenazine should be used with caution in patients with psychic depression.

Perphenazine is not recommended for children under 12 years of age.

Perphenazine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery.

PRECAUTIONS As with any potent medication, patients receiving perphenazine should be under medical supervision, particularly if they are receiving high doses. Patients who have had any severe reaction to phenothiazines or to imipramine should be treated cautiously, under close medical supervision.

Although the following adverse reactions have not been reported in patients treated with perphenazine, the possibility that they might occur with TRILAFON should be considered: blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, eosinophilia); liver damage (biliary stasis); narrowing of the visual fields; pigmentation of the retina, cornea, or lens; cerebral edema; polyphagia; photophobia; hyperpyrexia.

If hypotension develops, levarterenol (norepinephrine) can be used, but not epinephrine, because epinephrine's action is blocked and partly reversed by perphenazine. Severe, acute hypotension has occurred with the use of phenothiazines and is of particular concern in patients with mitral insufficiency or pheochromocytoma.

A significant rise in body temperature may indicate an idiosyncratic reaction to perphenazine; treatment with perphenazine should be stopped if this occurs.

The antiemetic effect of perphenazine can obscure signs of toxicity due to overdosage of other drugs, or mask the symptoms of disease (eg, brain tumor or intestinal obstruction).

Contact dermatitis has been reported with a perphenazine solution; therefore, contact of hands or clothing by those handling perphenazine solutions should be avoided.

POTENTIATION Since phenothiazines can potentiate the central-nervous-system-depressant actions of opiates, antihistamines, barbiturates, and alcohol, less than the usual dosage of these agents is required when they are administered concomitantly with TRILAFON. Patients should be cautioned that their response to alcohol may be increased while they are being treated with TRILAFON.

Phenothiazines also potentiate the effects of atropine, heat, and phosphorus insecticides, and should be used with caution in persons exposed to these agents.

ADVERSE REACTIONS *Extrapyramidal reactions:* dystonia including protrusion, discoloration, aching and rounding of the tongue; tonic spasm of the masticatory muscles, tight feeling in the throat, slurred speech, dysphagia, oculogyric crisis, trismus, torticollis, retrocollis, muscle weakness, and aching and numbness of the limbs; akathisia; motor restlessness; dyskinesia, parkinsonism; hyperreflexia; and ataxia. The incidence and severity of these reactions usually increase with increased dosage, but have occurred in some patients receiving low dosage. Reduction in dosage or treatment with an antispasmodic agent will usually control extrapyramidal reactions. In some instances, however, these reactions may persist after discontinuation of treatment with perphenazine.

Persistent tardive dyskinesia: As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities. There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine, vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Allergic reactions: erythema, pruritus, urticaria, eczema, anaphylactoid reactions, and local and generalized edema. In extremely rare instances, individual idiosyncrasy or hypersensitivity to phenothiazines has resulted in cerebral edema, circulatory collapse, and death. Photosensitization, asthma, and exfoliative dermatitis have also occurred in patients treated with phenothiazines.

Autonomic reactions: blurred vision, dry mouth or salivation, nasal congestion, nausea, vomiting, hypertension, tachycardia, hypotension, anorexia, urinary frequency or incontinence, and constipation. Significant autonomic effects have been infrequent in patients receiving less than 24 mg. perphenazine daily.

Other reactions: endocrine disturbances (lactation, gynecomastia, galactorrhea, disturbances in the menstrual cycle), headaches, mild insomnia, altered cerebrospinal fluid proteins, ECG abnormalities, reactivation of psychosis, paradoxical excitement, paranoid-like reactions, catatonia, and systemic lupus erythematosus-like syndrome. Hypnotic effects appear to be minimal, particularly in patients who are permitted to remain active. The following adverse reactions, though rare, have also been reported to be associated with perphenazine treatment: agranulocytosis; jaundice; hyperpigmentation of the skin; grand mal convulsions; failure of ejaculation; hyperglycemia.

Side effects with intramuscular TRILAFON **Injection** have been infrequent and transient. Dizziness or significant hypotension after treatment with TRILAFON (perphenazine) **Injection** is a rare occurrence.

NOVEMBER 1972
Schering Corporation
Kenilworth, N.J. 07033

Schering

SWW-5320

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

MAY BE AN APPROPRIATE CHOICE FOR YOUR NEXT PATIENT WITH CLINICAL DEPRESSION/ANXIETY

See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and
ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

Proved efficacy in the treatment of clinical depression/anxiety

In the five years since its introduction, Sinequan (doxepin HCl) has objectively demonstrated its value in the treatment of clinical depression/anxiety. Clinical studies have shown Sinequan to be unsurpassed in effectiveness when compared to other tricyclic antidepressants.

In addition, Sinequan may offer certain advantages not generally seen with some other drugs of its class:

Marked antianxiety activity

In addition to its significant antidepressant activity, which is usually seen two to three weeks after initiation of therapy, Sinequan possesses marked antianxiety activity.

In double-blind comparisons between doxepin and amitriptyline,[1-3] the antidepressant effects of both drugs were comparable; however, more notable antianxiety effects were seen with doxepin. Superior antianxiety activity was reported among depressed patients treated in a crisis intervention clinic.[4]

Prompt sedative activity

Of the tricyclic antidepressants, Sinequan has been found to have one of the most prominent sedative effects.[5] This sedative property can be taken advantage of by administering the major portion of the daily dose *h.s.* Since clinically depressed/anxious patients frequently have accompanying sleep disturbances, this may be a valuable benefit.

Generally does not affect the activity of guanethidine at usual clinical doses[6]

"A new tricyclic antidepressant, doxepin, was evaluated for its ability to block the amine uptake mechanisms of the peripheral adrenergic neuron and blood platelet in man. At low doses, there was no evidence of inhibition of the amine pump." At clinical dosages up to 150 mg. per day, Sinequan can be given to man concomitantly with guanethidine and related compounds without blocking the antihypertensive effect.[6]

Cardiovascular side effects reported infrequently

In a series of double-blind comparative studies, 1706 doxepin-treated patients were evaluated for side effects. Incidence of tachycardia was associated with only 45 (2.6%) clinically depressed/anxious patients.[3]

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1. Grof, P. et al.: Curr. Ther. Res. 16:470, May 1974.
2. Hackett, E. and Kline, N. S.: Psychosomatics 10:21, May-June (Section II) 1969.
3. Pitts, N. E.: Psychosomatics 10:164, May-June 1969.
4. Kiev, A. and Slavin, J. R.: Scientific Exhibit, Presented at the 126th Annual Meeting of the American Psychiatric Association, Honolulu, Hawaii, May 7-11, 1973.
5. Claghorn, J. L., in Claghorn, J. L. et al.: Patient Care 5:108, Oct. 15, 1971.
6. Fann, W. E. et al.: Psychopharmacologia 22:111, 1971.

See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

DIAGNOSTIC EVALUATION FORM

NAME_____ DATE_____

Date and Place of Birth_____ Age_____ Marital Status_____

Home Address_____ Telephone_____

Business Address_____ Telephone_____

Spouse or Nearest Relative_____ Relationship_____

Address_____ Telephone_____

Referred by_____

CHIEF COMPLAINT:

PRESENT ILLNESS:

PAST HISTORY:

FAMILY HISTORY:



MAY BE AN APPROPRIATE CHOICE FOR YOUR NEXT CLINICALLY DEPRESSED/ANXIOUS PATIENT

SINEQUAN®
DOXEPIN HCl

100-mg., 50-mg., 25-mg. Capsules and
ORAL CONCENTRATE 10 mg./ml., 120-ml. (4-oz.) bottles

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate
Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.
Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.
Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.
Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, paresthesias, ataxia, and extrapyramidal symptoms.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, facial edema, photosensitization, pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, and anorexia have been reported. (See anticholinergic effects).

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice and alopecia have been occasionally observed as adverse effects.
Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg. t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg./day to 150 mg./day.

In more severely ill patients an initial dose of 50 mg. t.i.d. may be required with subsequent gradual increase to 300 mg./day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg./day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg./day.

As an alternative regimen, the total daily dosage, up to 150 mg., may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.
Supply. Available as capsules containing doxepin HCl equivalent to 10 mg., 25 mg., 50 mg., and 100 mg. of doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN (doxepin HCl) 25 mg. and 50 mg. also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg./ml.) is available in 120 ml. bottles with an accompanying dropper calibrated at 5 mg., 10 mg., 15 mg., 20 mg., and 25 mg. SINEQUAN Oral Concentrate should be diluted with approximately 120 ml. of water or orange juice prior to administration.

More detailed professional information available on request.

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Clinical Geropsychiatry
by Adrian Verwoerdt, M.D.

Providing mental health services for the aged is rapidly becoming an urgent concern for psychiatrists working within the gerontological specialties.

CLINICAL GEROPSYCHIATRY is an insightful and ultimately indispensable guide to the problems attributed to the aging personality. Working within a medical-psychological framework, Dr. Verwoerdt has based his consideration on the biological, psychological, and social levels of human activity. His presentation of theoretical concepts serves to facilitate a better understanding of the psychopathology of growing old as well as an approach to treatment focusing on the origins of those symptoms associated with aging.

The material is organized into 22 chapters, each covering a separate and distinct facet of aging, supplemented with a list of references and suggestions for further study.

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The Sexual Experience
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The editors have called on 47 of the most knowledgeable and experienced authorities on sexuality to present this subject in all its richness and contradiction. No perspective has been denied in this unprecedented contribution to the literature of sexuality. In addition to perceptive discussions of sexual development, behavior, and identity, the text includes clinical reviews of the latest research on sex and medicine, marriage, and mental illness. Of special interest are several articles unique in their expanse of detail on such relevant topics as sex and the arts, sex legislation, pornography, and prostitution. A brilliant essay on the women's movement enumerates the effects of the movement on the social and economic circumstances of female life and thus on the changing nature of female sexual behavior.

1976/about 700 pages/117 illustrations, 14 tables/$23.50

Clinical Management of Sexual Disorders
edited by Jon Meyer, M.D.

In preparing this text, Dr. Meyer has met the need for a single volume containing the best of recent thought on remedial therapy for patients with sexual disorders including the determination of significant variables in sexual conditions, and the application of selected treatment techniques.

Each phase of the treatment process is adequately presented from the perspective of one or more well-qualified authorities on their particular subject. The nineteen contributors have provided thorough documentation so that anyone interested in further pursuit of the subject may easily know where to begin the search.

Addressed to no particular specialty, this volume will provide a valuable supplement to the skills of any clinician or practitioner who must regularly confront the problems associated with sexual dysfunction.

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Human Sexuality
A Health Practitioner's Text
by Richard Green, M.D.

An unusual sourcebook with strong clinical flavor, this book is aimed at the physician, medical student, or other health service professional needing a quick reference for use in counseling patients with sex oriented questions or complaints.

Green provides factual information on birth control, homosexuality, sex assignment, heterosexual relationships, and sexual behavior during pregnancy and in cardiac or mentally retarded patients. Throughout the text, he emphasizes the importance of attitudes and techniques that will serve the patient's need for dignity and the clinician's need for gathering information.

The increased sensitivity and adequacy of response that can be achieved through an understanding of human sexuality as revealed in these pages will permit the physician to offer honest sexual advice, free of the distortion of moral judgement.

1975/312 pages/illustrated/$14.50 (hardbound), $9.50 (paperbound)

Comprehensive Textbook of Psychiatry II (CTP-II)
edited by Alfred M. Freedman, M.D., Harold T. Kaplan, M.D., and Benjamin J. Sadock, M.D.

In this two volume, second edition of the highly regarded COMPREHENSIVE TEXTBOOK OF PSYCHIATRY, the editors have successfully performed the prodigious task of molding the knowledge and experience of more than 200 distinguished practicing psychiatric scholars into a new standard for comprehensive texts. This is one of those rare works that actually lives up to the promise of its title in that it presents the full scope of its subject from a theoretical as well as a clinical perspective.

This truly encyclopedic reference has already been recognized as the one title just as indispensable to the student as it is to the established practitioner or teacher. The achievement of CTP-II will be of interest to everyone concerned with the foundations, progress, and future of contemporary psychiatry.

1975/3000 pages/471 illustrations/$65.00

Medical Aspects of Human Sexuality
750 Questions Answered By 500 Authorities
compiled by Harold I. Lief, M.D.

MEDICAL ASPECTS OF HUMAN SEXUALITY was written on the premise that sex related problems are the proper concern of everyone caring for the psychological or physiological well being of their patients. Neither didactic nor casual in approach, it provides authoritative responses to the questions most frequently encountered in daily practice. The question and answer format permits the inclusion of several views on those problems for which empirical studies have provided inconclusive results.

In this thorough and sensitive collection of informed dialogue from the journal of the same name, every detail of the sexual experience is considered. The topics range from basic questions on physiology and anatomy to the complex issues involved with sexual dysfunction, deviancy, marital relationships, venereal disease, and sex roles in modern society.

For the concerned practitioner, student, or researcher it will prove to be an excellent source of straightforward, factual assistance.

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Loxitane®
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Breaks the "silence" of schizophrenia



Remarkable performance in symptom control

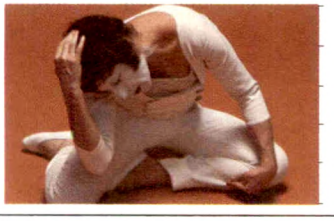
See LOXITANE prescribing information on the last page of this advertisement for warnings and precautions and for more detailed information concerning side effects.

Loxitane®
LOXAPINE SUCCINATE
Lederle

Clearly exhibited efficacy in the reduction of symptoms and symptom patterns

EVALUATION OF RESPONSE TO LOXITANE BY THE BRIEF PSYCHIATRIC RATING SCALE (BPRS) IN 221 SCHIZOPHRENIC PATIENTS

(98 acute schizophrenic patients in 5 double-blind studies[1] treated for 3 to 6 weeks; 123 chronic schizophrenic patients in 6 double-blind studies[1] treated for 8 to 12 weeks)

SYMPTOM CLUSTERS (BPRS)		PERCENT MAXIMUM POSSIBLE IMPROVEMENT: Acute Schizophrenic Patients	PERCENT MAXIMUM POSSIBLE IMPROVEMENT: Chronic Schizophrenic Patients		Pretreatment Score	Posttreatment Score	Statistical significance of change from Base Line $p=$
DEPRESSION Somatic Concern Anxiety Guilt Feelings Depressive Mood		56%	27%	ACUTE	3.07	1.92	0.001
				CHRONIC	2.02	1.74	0.01
THOUGHT DISORDER Conceptual Disorganization Grandiosity Suspiciousness Hallucinatory Behavior Unusual Thought Content		61%	23%	ACUTE	3.88	2.11	0.001
				CHRONIC	3.30	2.78	0.05
ANERGIA Emotional Withdrawal Mannerisms and Posturing Motor Retardation Blunted Affect		47%	17%	ACUTE	3.75	2.46	0.001
				CHRONIC	3.75	3.27	0.05
EXCITEMENT-DISORIENTATION Excitement Disorientation		61%	21%	ACUTE	2.54	1.60	0.01
				CHRONIC	2.50	2.19	—

1. Data on file, Clinical Research Department, Lederle Laboratories

See LOXITANE prescribing information on the last page of this advertisement

Loxitane®
LOXAPINE SUCCINATE
Lederle

Exhibited certain favorable trends in side effects profile

IN 31 STUDIES INVOLVING 469 ACUTE AND CHRONIC CHIZOPHRENIC PATIENTS, CERTAIN FAVORABLE TRENDS WERE EXHIBITED IN THE OXITANE SIDE EFFECTS PROFILE; THESE REQUIRE FURTHER TESTS AND BROADER LINICAL EXPERIENCE FOR CONFIRMATION.



While cardiac arrest, blood dyscrasias and renal toxicity have been reported with other antipsychotic agents, they have not been seen with LOXITANE. Hepatotoxicity manifested by jaundice or biliary stasis has not been observed. Transient liver enzyme changes, however, have been reported, but it has not been determined whether they are related to LOXITANE administration.

Although a few cases of changes in ECG have been reported, a causal relationship between this reaction and LOXITANE has not been established. Clinical experience with LOXITANE has not demonstrated ocular toxicity; however, the possibility of its occurrence cannot be ruled out at this time. Of the various endocrine abnormalities, only galactorrhea has been reported with LOXITANE and only in rare instances. Manifestations of adverse effects on the central nervous system other than extrapyramidal symptoms have been encountered infrequently, and drowsiness, when it occurs, is usually mild and subsides with continued therapy. Skin rashes of uncertain etiology have been observed in a few patients during the hot summer months; therefore the possibility of phototoxicity and/or photosensitivity cannot be excluded.

In general, LOXITANE presents a profile of extrapyramidal side effects similar to that of other agents used in the treatment of schizophrenia.

Cardiovascular effects such as hypotension, hypertension, lightheadedness and syncope have been reported. Anticholinergic effects seen with LOXITANE include dry mouth, nasal congestion, constipation and blurred vision.

nings and precautions and for more detailed information concerning side effects.

FOR ORAL USE

PRODUCT INFORMATION

DESCRIPTION

LOXITANE *loxapine*, a dibenzoxazepine compound, represents a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines. Chemically, it is 2 - chloro - 11 - (4 - methyl - 1 - piperazinyl) dibenz [b,f] [1,4] oxazepine. It is present in capsules as the succinate salt. Each 1.36 mg of loxapine succinate is equivalent to 1 mg of loxapine.

ACTIONS

Pharmacologically, loxapine is a tranquilizer for which the exact mode of action has not been established. However, changes in the level of excitability of subcortical inhibitory areas have been observed in several animal species in association with such manifestations of tranquilization as calming effects and suppression of aggressive behavior.

In normal human volunteers, signs of sedation were seen within 20 to 30 minutes after administration, were most pronounced within 1½ to 3 hours, and lasted through 12 hours. Similar timing of primary pharmacologic effects was seen in animals.

Absorption of loxapine following oral or parenteral administration is virtually complete. The drug is removed rapidly from the plasma and distributed in tissues. Animal studies suggest an initial preferential distribution in lungs, brain, spleen, heart, and kidney. Loxapine is metabolized extensively and is excreted mainly in the first 24 hours. Metabolites are excreted in the urine in the form of conjugates and in the feces unconjugated.

INDICATIONS

LOXITANE *loxapine succinate* is indicated for the manifestations of schizophrenia.

CONTRAINDICATIONS

LOXITANE is contraindicated in comatose or severe drug-induced depressed states (alcohol, barbiturates, narcotics, etc.)

LOXITANE is contraindicated in individuals with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Safe use of LOXITANE *loxapine succinate* during pregnancy or lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the benefits of treatment be weighed against the possible risks to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from midpregnancy with doses of 0.6 and 1.8 mg/kg, doses which approximate the usual human dose but which are considerably below the maximum recommended human dose.

Usage in Children: Studies have not been performed in children; therefore this drug is not recommended for use in children below the age of 16.

LOXITANE, like other tranquilizers, may impair mental and/or physical abilities, especially during the first few days of therapy. Therefore, ambulatory patients should be warned about activities requiring alertness (eg, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

PRECAUTIONS

LOXITANE loxapine succinate should be used with extreme caution in patients with a history of convulsive disorders since it lowers the convulsive threshold. Seizures have been reported in epileptic patients receiving LOXITANE at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy.

Loxapine has an antiemetic effect in animals. Since this effect may also occur in man, loxapine may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

LOXITANE should be used with caution in patients with cardiovascular disease. Increased pulse rates have been reported in the majority of patients receiving antipsychotic doses; transient hypotension has been reported. In the presence of severe hypotension requiring vasopressor therapy, the preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine.

The possibility of ocular toxicity from loxapine cannot be excluded at this time. Therefore, careful observation should be made for pigmentary retinopathy and lenticular pigmentation since these have been observed in some patients receiving certain other antipsychotic drugs for prolonged periods.

Because of possible anticholinergic action, the drug should be used cautiously in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

ADVERSE REACTIONS

CNS Effects: Manifestations of adverse effects on the central nervous system, other than extrapyramidal effects, have been seen infrequently. Drowsiness, usually mild, may occur at the beginning of therapy or when dosage is increased.

It usually subsides with continued LOXITANE therapy. The incidence of sedation has been less than that of certain aliphatic phenothiazines and slightly more than the piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported.

Extrapyramidal Reactions—Neuromuscular (extrapyramidal) reactions during the administration of LOXITANE *loxapine succinate* have been reported frequently, often during the first few days of treatment. In most patients, these reactions involved Parkinson-like symptoms such as tremor, rigidity, excessive salivation, and masked facies. Akathisia (motor restlessness) also has been reported relatively frequently. These symptoms are usually not severe and can be controlled by reduction of LOXITANE dosage or by administration of antiparkinson drugs in usual dosage. Dystonic and dyskinetic reactions have occurred less frequently, but may be more severe. Dystonias include spasms of muscles of the neck and face, tongue protrusion, and oculogyric movement. Dyskinetic reaction has been described in the form of choreo-athetoid movements. These reactions sometimes require reduction or temporary withdrawal of LOXITANE dosage in addition to appropriate counteractive drugs.

Persistent Tardive Dyskinesia—As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movement of the tongue, face, mouth, or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia; antiparkinson agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Cardiovascular Effects: Tachycardia, hypotension, hypertension, light-headedness, and syncope have been reported.

A few cases of ECG changes similar to those seen with phenothiazines have been reported. It is not known whether these were related to loxapine administration.

Skin: Dermatitis, edema (puffiness of face), pruritus, and seborrhea have been reported with loxapine. The possibility of photosensitivity and/or phototoxicity occurring has not been excluded; skin rashes of uncertain etiology have been observed in a few patients during hot summer months.

Endocrine Effects: No endocrine abnormalities have been reported.

Anticholinergic Effects: Dry mouth, nasal congestion, constipation, and blurred vision have occurred; these are more likely to occur with concomitant use of antiparkinson agents.

Other Adverse Reactions: Nausea, vomiting, weight gain, weight loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, and polydipsia have been reported in some patients.

DOSAGE AND ADMINISTRATION

LOXITANE *loxapine succinate* is administered orally usually in divided doses two to four times a day. Daily dosage (in terms of base equivalents) should be adjusted to the individual patient's needs as assessed by the severity of symptoms and previous history of response to antipsychotic drugs. Initial dosage of 10 mg twice daily is recommended, although in severely disturbed patients initial dosage up to a total of 50 mg daily may be desirable. Dosage should then be increased fairly rapidly over the first seven to ten days until there is effective control of psychotic symptoms. The usual therapeutic and maintenance range is 60 mg to 100 mg daily. However, as with other antipsychotic drugs, some patients respond to lower dosage and others require higher dosage for optimal benefit. Daily dosage higher than 250 mg is not recommended. For maintenance therapy, dosage should be reduced to the lowest level compatible with symptom control; many patients have been maintained satisfactorily at dosages in the range of 20 mg to 60 mg daily.

HOW SUPPLIED

LOXITANE *loxapine succinate* is supplied in the following base equivalent strengths:

CAPSULES Hard Shell Printed "Lederle"

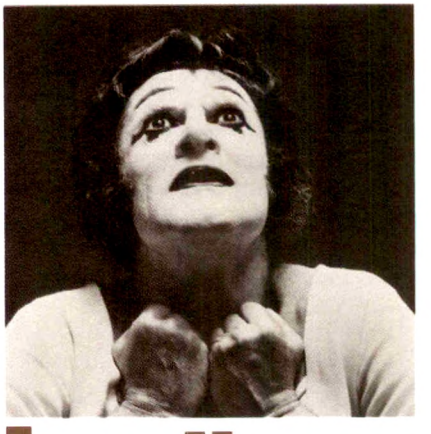
10 mg—Green and Yellow; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5360.

25 mg—Green Two Tone; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5361.

50 mg—Green and Blue; Bottles of 100, 1000, and unit dose 10 x 10's—Product Number 5362.

REV. 3/75

For the manifestations of schizophrenia



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Because schizophrenia is symptoms

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For Brief Summary, please see following page.

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75-379

RESULTS OFTEN SEEN IN A WEEK



Because the somatic and psychic complaints that accompany depressive neurosis can interfere with the treatment of the underlying psychopathology—rapid relief of these symptoms is often crucial to therapy.

In 14 double-blind studies of four weeks duration, 339 patients with depressive neurosis received Mellaril. In these studies, 55% of the overall improvement was observed by the end of the first week, and a total of 293 patients (86%) improved during the four weeks.*

Mellaril usually does not cause euphoria or undue sedation and is nonaddicting. Patients, therefore, generally remain alert and better able to respond to therapy. (The physician should, however, caution patients against participating in activities which require complete mental alertness, e.g., driving.)

*Data on file at Sandoz Pharmaceuticals.

MELLARIL®
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TABLETS: 25 mg and 50 mg thioridazine HCl, U.S.P.

for the short-term treatment of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling. The following reactions have occurred with phenothiazines and should be considered: *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign. and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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THE AMERICAN JOURNAL OF PSYCHIATRY

The Life of Psychiatry

BY BERTRAM S. BROWN, M.D.

Focusing on the definition, scope, and role of psychiatry today, the author discusses whether psychiatry is primarily medical, whether it is overstepping its boundaries in attempting to treat problems of living, and whether it is too involved with social questions. On the basis of an examination of the current scientific base of psychiatry, he predicts that psychiatry will continue to grow in size and diversity, that it will refocus substantially on biological issues, that it will become more humble about what it can do with regard to social problems, and that it will continue to yield new therapeutic measures and techniques.

THIS PERSPECTIVE on the life of psychiatry is both personal and professional, drawing on both my evolving self-concept as a psychiatrist and my role as director of the National Institute of Mental Health. Although I will focus on psychiatry, many of my comments apply to the broad field of mental health. To write a truly comprehensive and current paper on this field is difficult. Therefore, I will discuss briefly or omit several important areas, such as the potential impact of national health insurance, the role of Professional Standards Review Organizations (PSROs), the new health planning legislation (Public Law 93-641), and other critical areas. Rather, I will attempt to focus on some of the potentials and limits of psychiatry at a time when the resiliency of the profession is being tested by calls for both expansion and contraction. The simple fact that many of these calls originate within the profession will make the proof of the test all the more crucial.

Revised version of a paper presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Brown is Director, National Institute of Mental Health, Alcohol, Drug Abuse, and Mental Health Administration, 5600 Fishers Lane, Rockville, Md. 20852.

I would like to highlight a particular evolution that has taken place. Twenty-five years ago, when our predecessors spoke of psychiatry they were also speaking of the broader field of mental health. What they and their contemporaries termed "mental health" some leaders today term "human services." Many of these leaders are psychiatrists, but the issue of how much psychiatry represents mental health and how much mental health represents human services is at the heart of the great debate concerning the role, definition, scope, and boundaries of psychiatry.

The same issue applies to NIMH. There is a "strict constructionist" view of the mission of NIMH that emphasizes health sciences; there is also a broad view that emphasizes human services and other social missions.

What is the role of psychiatry? Does it even have a role, or is it, as some would have it, dead or dying? Is it perhaps gathering strength? What is the nature of its identity crisis? What are its prospects?

COMPARISONS WITH GENERAL MEDICINE

Measured by the sheer number of practitioners,[1] psychiatry has had an astonishing growth since 1939. In that year there were about 3,000 psychiatrists (2.3 per 100,000 of the population of the United States). Most of them were called "alienists." In 1948, when NIMH was founded, there were 4,700. In 1950 there were 7,100 (4.7 per 100,000). By 1960 this number had almost doubled to 14,000 (7.7 per 100,000). During the decade of the 1960s the number of psychiatrists increased by 65% to 23,200 in 1970 (11.3 per 100,000). Today there are 27,000 psychiatrists in the United

[1]Data on the number of psychiatrists before 1960 were compiled from records on APA membership and filled psychiatric residencies. Data on psychiatrists and other physicians for 1960 to 1973 are based on *Distribution of Physicians in the United States* (1). I have estimated the projection for 1980, taking into consideration the number, production, and attrition of practicing psychiatrists today.

States.[2] Estimating according to the number of potential psychiatrists in the educational system now, there will be 30,000 by 1980 (17.8 per 100,000).

Thus the number of psychiatrists has increased by 16% in the past 5 years and 45% in the past 10 years. Since the founding of NIMH it has increased more than fivefold.

How does this compare with general medicine? In the decade from 1963 to 1973 the total number of nonpsychiatrist physicians increased by 81,422, from 259,894 in 1963 to 341,316 in 1973, an increase of 31.3%. During the same decade the number of psychiatrists increased by 8,482, from 16,581 to 25,063, an increase of 51.2%. Additionally, the proportion of psychiatrists in the total number of physicians increased by 0.8%, from 6.0% to 6.8%.

A comparison with other specialty groups shows that psychiatry's growth during 1963 through 1973 was the third largest. It was surpassed only by internal medicine, which increased by 2.7%, and by radiology, which increased by 1%.

However, the number of psychiatric residents in the United States today has reached a plateau. According to data from APA and the American Medical Association (AMA), the number of residents in training in 1972 was 4,699, in 1973 it was 4,739, and in 1974 it was 4,802. Approximately 1,000 American medical school graduates were in the first year of psychiatric residency in September 1972, representing 10.5% of the graduating class. In September 1974, there were again about 1,000 graduates who were in the first year of psychiatric residency. However, these residents represented only about 8.9% of a larger graduating class. Although the number remained approximately the same, the proportion of medical school graduates entering psychiatric residency dropped by approximately 15% between 1972 and 1974.

One feature of medicine in general that applies dramatically to the statistics on the number of psychiatrists is the role of foreign medical graduates. According to data from APA and AMA, foreign medical graduates constituted 32% of all psychiatric residents in 1974. They now comprise more than 50% of the psychiatrists in practice in many state institutions; in a few states their proportion in these hospitals nears 100%.

Activities in Congress, in the states, and in professional organizations that may limit the immigration or the ability to practice of foreign medical graduates will have an immediate and severe impact on patient care. The issue of the willingness of other psychiatrists to work in state institutions will stand out clearly if foreign medical graduates are excluded from these jobs.

About 1,500 psychiatrists in the United States complete their residency training each year, and about 850 leave active practice. Thus about 750 psychiatrists are annually added to the national total.

It is generally estimated (2) that about half of all psychiatrists active today are in general psychiatry, 13% deal with adults, 10.5% specialize in psychoanalysis, and just over 9% are in child psychiatry. Somewhat more than 4% work in community or social psychiatry, and only 0.5% report specializing in geriatric psychiatry.

Another aspect of these numbers and proportions is extremely interesting. A 1972 NIMH survey of psychiatrists (3) showed that the percentages of psychiatrists from minority groups were relatively small—1.4% were black, 1.5% were Spanish-American, and 1.3% were from other minority groups. Although earlier figures are not readily available for comparison, the proportion of minority psychiatrists is apparently growing, albeit slowly.

For yet another perspective on the issue of numbers, consider the distribution of psychiatrists per 100,000 population in this country (2). Eleven states, including South Dakota, Wyoming, and Alabama, have less than 5 psychiatrists for every 100,000 people. Twenty states, including Alaska, Maine, Minnesota, and Virginia, have more than 5 but less than 8 psychiatrists per 100,000. In 8 states the ratio is at least 12 psychiatrists per 100,000. New York State has 25, Massachusetts has 22, Connecticut has 20, Maryland has 19, Vermont and California have 17 each, Colorado has 15, and Hawaii has 13. The average number of psychiatrists per 100,000 population for the entire United States is 11.9.

The District of Columbia stands head and shoulders above the other states in terms of number of psychiatrists. Its ratio is 58 psychiatrists per 100,000 people—more than double that of New York State and more than quadruple the average. The numbers do not include the more than 2,200 psychiatrists who work for the federal government in the District of Columbia.

The wide variation in number of psychiatrists per 100,000 people among the states is important. The figures range from 5 to more than 50; i.e., one state has more than 10 times as many psychiatrists per 100,000 people as another. When rural areas are distinguished from metropolitan areas and those in between in each state, one finds that some areas have 20 to 30 times as many psychiatrists per 100,000 people as other areas. These ranges underlie the issues pressing at health and mental health manpower legislation. The term now most commonly used to describe this situation is "maldistribution."

PSYCHIATRIC TRAINING

The recent policy shifts in psychiatric training have been among the most dramatic in the history of all professional training. Several years ago the various professional associations engaged in vigorous debate over the requirement of a medical internship for medical

[2]Although there are no hard data on the number of psychiatrists in the world today, information from the World Health Organization and the World Psychiatric Association indicate that the figure is between 75,000 and 100,000. Thus the United States has between one-quarter and one-third of the world's psychiatrists.

school graduates planning to enter psychiatry. Two polarities of opinion emerged. One group held that dropping the internship would be a dreadful mistake, and the other felt that most of a medical internship was irrelevant to psychiatric practice and should be dropped. The latter position ultimately won, and the American Board of Psychiatry and Neurology dropped the internship requirement.

During the next year or so we will undoubtedly see a reversal of this policy and a reinstitution of the internship or an internship-like experience as either a prerequisite to or the initial part of psychiatric residency training. It is important to note that the experience of caring for a variety of patients with serious physical illnesses rather than the loss of a year called an "internship" has been the missing ingredient.

In a practical sense, the movement of psychiatry back into the health arena is more than a conceptual issue. It is a descriptive issue on the current educational scene.

Along with the increase in the number of psychiatrists there has been an increase in the number of residency programs (4). There were 270 residency programs in the United States in 1973, representing positions for 4,992 residents; 4,131 of these positions (86%) were filled. The residency programs ranged from those which were highly clinically oriented to those which worked with chronic patients in state institutions to those which were psychodynamically oriented university programs. Moreover, these programs of psychiatric training and education were not limited to psychiatrists. Training in psychiatry and behavioral sciences has been provided to nonpsychiatric physicians, particularly those providing primary care, and to other mental health professionals.

Of even greater importance is the provision of psychiatric, behavioral, and social science training for medical students. Every medical school now has a department of psychiatry. In the future, programs in psychiatry and behavioral and social sciences will be provided and emphasized in all of the stages in the education of medical students.

THE PATIENT POPULATION

The heart and the end point of the issues of scope and definition are in psychiatry's interaction with patients and other people. The context of the following patient population statistics is traditional, including the services of state and Veterans Administration hospitals, outpatient clinics, and community mental health centers. The numbers of patients are somewhat understated because they do not include those seen by psychiatrists in private practice and by other mental health professionals. Also not included are family service agencies or what I call "fringe groups," the plethora of sensitivity training groups, experimental programs, and other encounter sessions and seminars.

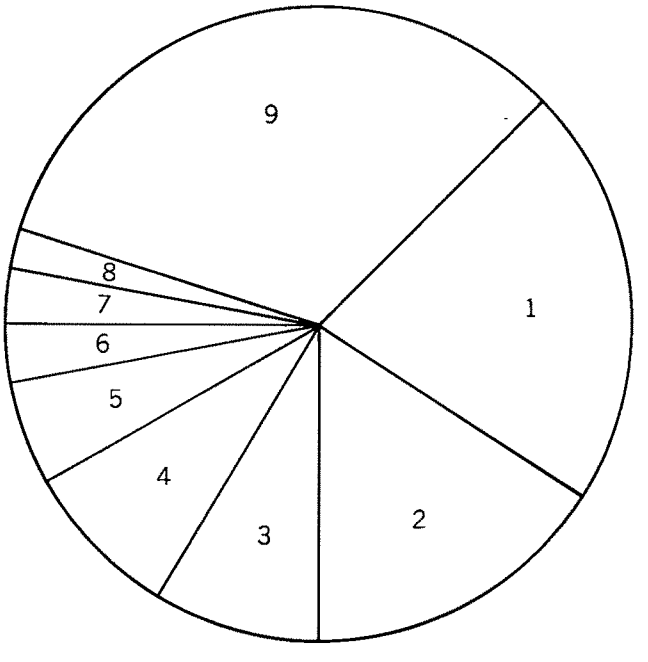
In 1955, about 1.7 million people (1% of the total U.S. population) had some contact with the mental health care system (5). By the mid-1960s this number had increased to 2.6 million (1.4% of the population). Our most recent figure (for 1971) is more than 4 million (2.4% of the population). These figures represent all patient care episodes and include some duplication.

A conservative estimate for 1975 would be 5 million to 6 million people in contact with the mental health care system. The diagnostic categories of those who were receiving some kind of treatment in 1971 (6) included schizophrenia (22.5%), depressive disorder (15.4%), alcohol disorder (8.8%), organic brain syndrome (5.4%), mental retardation (3.1%), drug disorder (2.9%), and psychosis other than schizophrenia and depression (1.5%). All other diagnoses totaled 32.4%, and 8% of the patients were undiagnosed (see figure 1).

The national utilization rate, the percentage of the population that in a given year comes to the attention of the formal mental health care system, was about 2% or 2.5% in 1971 (5). These contacts may be as brief as 10 minutes in the emergency room of a hospital or as long as the entire year in an inpatient unit.

The range of utilization rates in different parts of the United States is somewhat similar to the distribution of psychiatrists. The utilization rate in rural parts of

FIGURE 1
Distribution of Diagnoses of More Than 4 Million Patients Who Had Contact with the Mental Health Care System in 1971 (6)



1: Schizophrenia (22.5%)
2: Depressive disorder (15.4%)
3: Alcohol disorder (8.8%)
4: Undiagnosed disorder (8.0%)
5: Organic brain syndrome (5.4%)
6: Mental retardation (3.1%)
7: Drug disorder (2.9%)
8: Psychosis other than schizophrenia or depression (1.5%)
9: All other disorders (32.4%)

the country, which have about 5 psychiatrists per 100,000 people, is 0.5% to 0.75%. The utilization rate of such urban areas as San Mateo County, midtown Manhattan, and the suburbs of Washington, D.C., is about 7% or 8%. This disparity is behind the demands of both liberals and conservatives to improve access to mental health care and to meet the criterion of equity.

Looking at this situation most conservatively, i.e., projecting a 1% increase in the national utilization rate, continued support for training at no less than the present level, and no fewer entrants into the profession, we can predict a shortage of nearly 10,000 psychiatrists by 1981.

The forces behind the increased utilization rates are related to the purchasing power that is now available through third-party financing. The rates will increase even more with the passage of some form of national health insurance in the near future. They are also related to the fact that seeking psychiatric assistance has less of a stigma attached to it today.

A LOOK AT THE PROFESSION

I want to emphasize that there are different types of psychiatrists, that their training experiences are diverse, and that the types of patients they see are also diverse. This is another reflection of the issues of scope, definition, and role.

I believe that psychiatry faces less danger from adversity than it does from diversity. Although diversity is a danger, it is much more a sign of growth and experimentation, a developmental process that relates clearly to the history of our evolving specialty and the state of our scientific knowledge. A healthy and sound aspect of diversity is that it results in a focusing and narrowing of our skills. Each type of psychiatry may treat the very persons who need and benefit from that special focus of experience and approach.

The following seven types of psychiatrists comprise a loose, non-mutually-exclusive typology:

Type 1, the neuropsychiatrist, is rapidly becoming a vanishing breed. This is the pragmatic clinical practitioner who is as comfortable with neurology as with psychiatry. His patients range from the simply troubled to the severely ill, and he may see patients for brief counseling or hospitalize them. He may use convulsive therapy; he may prescribe drugs.

I recently checked on the number of people who took Board examinations in both neurology and psychiatry in 1973 and 1974. According to data from APA, several hundred took one or the other and passed; the number who took both in 1974 can be counted on one hand. Two or three decades ago, however, the majority of psychiatrists took both examinations.

My type 2 psychiatrist—the psychodynamically oriented psychotherapist—seemed on the path to dominance only a short time ago. This psychiatrist may see patients one to three times a week over a period of 6 months to 2 years or more. Steeped in psychoanalytic theory, he concentrates on an office practice; although he may hospitalize his patients occasionally, he usually transfers patients needing hospitalization to a colleague. The frequency of this type of psychiatrist is leveling off.

The psychoanalyst, type 3, has taken full psychoanalytic training and practices psychoanalysis predominantly. He may have 5 to 15 patients and may allocate a small proportion of his time to consultation or other outside activities. Because this type of psychiatrist was dominant, from 1945 to 1955 it was nearly impossible for a nonpsychoanalyst to become chairman of a department or professor of psychiatry. Today the number of psychoanalysts is dwindling, and an exclusively psychoanalytic psychiatrist is rarely found at the head of a department of psychiatry.

Type 4, the biological psychiatrist, includes some of the brightest young people in the country. Skilled in the psychobiological sciences, this cadre is changing the profession as a whole and influencing society.

My type 5 is the child psychiatrist. Many child psychiatrists were pediatricians before turning to psychiatry. The growth of child psychiatry as a specialty has been phenomenal. According to data from AMA, there were 751 child psychiatrists in the United States in 1963; in 1973 there were 2,362. This represents a growth rate of 200%. General psychiatry's growth rate was 46% during the same period.

I would classify type 6 as the social psychiatrist—the program administrator or the director of a mental health center or a new alcoholism or drug abuse endeavor. His work is at the interface of troubled behavior and social systems.

Finally, there is type 7, which I call the "experimental" or "fringe" group. This psychiatrist uses a variety of techniques and is often found in unusual places doing unusual things. This is a critically important group because when the fads filter out and the charlatans and phonies take their leave, type 7 promises to be the wave of the future. If we look carefully at the history of psychiatry we find that there have always been elements in the fringe and experimental groups that have had more influence ultimately than was generally recognized at the time.

KEY ISSUES

The major issues I have focused on have to do with the definition, scope, and role of psychiatry. The critical subissues are whether psychiatry is primarily medical and therefore concerned with taking care of the seriously clinically sick, whether psychiatry is overdoing its task in attempting to treat those with transient or less severe clinical problems, and whether psychiatry is too involved in social questions.

Thus the issues center around three categories of people—the very sick and psychotic patients, those who are greatly handicapped by emotional and psychological disturbances, and those who are experiencing

problems of living, for whom the desired quality of life has proved elusive.

It is around these groups that the tensions in our field are being generated; two points of view seem to predominate. There are those who believe that psychiatry has clearly bitten off more than it can chew and ought to get back to treating serious mental illness, and there are those who feel that unless we work to change social systems we shall inevitably have a continuing flow of sick and troubled people.

I realize that people at both ends of this spectrum are keenly aware that the intermediate zone is one of great diagnostic difficulty. It takes skill and knowledge to ascertain what might be a life problem and what might be the earliest signs of an oncoming depression or other mental illness. Such diagnostic requirements demand that we take an extensive look at our clinical and scientific base and what we do or do not know about prevention.

THE SCIENTIFIC BASE

It is one thing to have a value base for what we think we should do, but it is quite another to have a scientific base—an objective set of measures about whether what we do is effectively achieving what we set out to do. Psychiatry has been a major service-providing instrument of that vital and nourishing enterprise we call mental health research. Psychiatry has played a significant but smaller role in the actual conduct of research. Only a few psychiatrists—numbering in the hundreds—are full-time researchers. This is a vital area of debate as we deliberate over the nature of financial support policies in research in general and psychiatry in particular. In their roles as conceptual, clinical, and program leaders, psychiatrists have played a leading role in stimulating and supporting research endeavors by other mental health professionals and behavioral and social scientists.

At NIMH we have just finished a 2-year effort by a research task force that involved 300 people and $1 million to review $1 billion worth of research in the United States supported by NIMH and others.

The report (7) shows that the number of research projects supported annually by NIMH has grown from 38 in 1948, when the first appropriations were made under the National Mental Health Act of 1946, to nearly 1,500 in 1975. About 30% of the total NIMH research budget has been devoted to research in the biomedical and behavioral sciences, including studies designed to identify physiological, psychological, social, and cultural factors that shape human behavior. Out of this effort has emerged work of enormous relevance, not only for enlarging our understanding of human behavior but for productive clinical research as well.

The task force report will vitalize debates now under way as to the nature and scope of the psychiatric research enterprise, including such issues as balance and priorities. To some degree, these are but other ways of looking at the scope and definition of psychiatry itself. A brief example will suffice: over the last 20 years we have seen the ratio of the amount of NIMH-supported biomedical research to psychosocial research reversed; 20 years ago the ratio of biomedical research to psychosocial research was 2 to 1; in 1975 it was 1 to 2.

This dramatic shift reflects the broadening of NIMH's mission in the late 1960s into social problem areas such as crime and delinquency. Within the mental illness sphere of the NIMH program, biomedically oriented research continues to be predominant but with an increasingly fine line of distinction between the two approches.

The Subjective Approach

One of the ways I have approached the issue of psychiatry's scientific base is to return to the subjective. For example, when I really wonder about the depth and wisdom of my opinion or judgment on a given situation, I ask what I want for myself, my wife, my children, my parents. Some of the answers that emerged for me follow:

1. I would much rather have a mid-life depression today than 20 years ago (assuming my present age both now and then) because of the current advances in the treatment of depression and acute depression, the understanding of its biology, and the availability of drugs.

2. I think I would rather one of my children have an acute schizophrenic break now than 20 years ago. I will not go farther because I know the complexities surrounding schizophrenia, and I do not say this with quite the certainty that I use in discussing mid-life depression. However, I think we have made enough advances in the treatment of acute schizophrenia that the scientific base is ahead of that of 20 years ago.

3. Suppose my marriage were in serious trouble, with agony, misery, sleeplessness, concern, trouble with the children, and job trouble. Would I be any better off being treated by a psychiatrist today than I would have 20 years ago? I think not. The best possible source for help for me in this situation would be a mature, wise psychiatrist. There has been an increment of knowledge in the area of marital and family therapy, but that increment is small compared with what is known or needs to be known. The critical variable today is the same as it was 20 years ago—the maturity and wisdom of the helping person.

The research task force (7) found that research investments in the area of helping the practitioner deal with troubled people have not yielded a cornucopia of techniques or programs. To realize our hopes and expectations in this area we must reassess what we have been doing, decide what we have been doing incorrectly, and ascertain what we can do better.

The relatively low yield in social research may be due to many factors. It may be that we are just now

building the base in this complex field so that workers in the next decade or so may provide useful answers. It may be that the methodologies necessary to get answers in the area of social problems are so complex that we have yet to develop effective tools. There are those who would have us abandon this area altogether. This I cannot support. To do it better—perhaps even to make use of some of the resources from more promising areas—makes sense, but to do nothing seems both unprofessional and unwise.

A Look at Possibilities

Let me now move from my subjective position to a more general frame of reference:

1. My answer to the question of whether psychiatry is up to the task of dealing with serious mental illness and whether it is doing the job has to be yes. Emphatically, however, I must add that we are not doing it perfectly, nor are we doing enough of it, given our knowledge base. In spite of our advances in knowledge and the addition of chemotherapy to our armamentarium, we must still wait for research to uncover some basic unknowns. Even so, we could do it better if we made the best use of what we know now, particularly about community treatment and care.

2. My answer to whether psychiatry is dealing effectively with that 5% or 10% of the population who are not psychotic but who are very troubled, whose functioning is disturbed, or who are on the verge of becoming disturbed also has to be yes. However, I must add that the gains in this area have been less significant than the gains in the biological area in the struggle against schizophrenia and psychotic depression.

3. My best answer to whether psychiatry's scientific base regarding the treatment of the unhappy, the alienated, and the troubled has increased in the past 20 years is that it has increased very little.

For example, are people who have been laid off from work better off today than those who lost their jobs in the 1930s? In one sense, of course, with unemployment insurance and other social benefits, the outlook for economic recovery is considerably better than it was then, but are these people better off in terms of what psychiatry has to offer?

Many more people who develop troubling symptoms ask for help today than asked for it in the 1930s, perhaps for no other reason than that it is so much more available. However, only a minimal amount of knowledge has accumulated—beyond our increased ability to care for the mentally or emotionally disabled—to strengthen our capacity for dealing with the unemployment problem as it affects the emotional well-being of the individual.

When it comes to the question of whether psychiatry *should* treat people with problems of living, one of the most powerful dynamics is the great influence of the dollar. If the troubled are willing to pay, and the psychiatrist is willing to accept, the question becomes moot.

What Lies Ahead?

On the basis of the research task force report (7) and the knowledge my colleagues and others in the field have gained, I predict in complete confidence that psychiatry will continue to grow in size and diversity.

There is no sign at present that there is a lack of growth in the size of psychiatry or that the demand for its services is decreasing. Any profession that is growing in size, that is having more and more demands put on it, and that has such a diversity in training programs, types of practitioners, and therapeutic techniques is not a dying profession.

By reason of the nature of psychiatry's scientific base and its cultural connection to medicine, I also predict that the profession will refocus to a significant extent on clinical—and particularly biological—psychiatry, that it will become more humble about what it can do in regard to social problems and issues, and that it will continue to yield new experimental therapeutic measures and techniques. Most of these will fall by the wayside, but a few should lead to more effective treatment.

For the rest of this century and probably well into the next, treating the hard core of mental illness—the schizophrenias and the depressions—and dealing with troubled behavior that is or threatens to become incapacitating will continue to be vital activities in the thriving profession of psychiatry.

The psychiatrist will continue to be called on to help with problems of living that are less serious but more widespread. However, so will the psychologist, the social worker, the psychiatric nurse, the educator, the clinical sociologist, the clergyman, and others who for years have been working in this area.

In other words, psychiatry can expect to find itself in collegial competition with other professions and, perhaps, with paraprofessionals in the treatment of people whose problems in living are not associated with serious clinical illness.

From the standpoint of the patient, or the client, there may be two strong reasons for going to a skilled problems-of-living psychiatrist rather than to another mental health professional. One is to enjoy whatever protection can be assumed to lie in the M.D. degree—the ethics and the traditions associated with medicine for thousands of years. The other is to benefit from the greater diagnostic acumen that the psychiatrist has, or should have, in case of an underlying biological condition requiring attention. When there is no such condition, the wise pastoral counselor, the humanistic psychologist, and the warmhearted psychiatrist may be equally effective; the results depend more on the quality and less on the type of training of the human beings who serve as therapists or counselors. In the final analysis, the patient, the consumer, will make the final judgment.

In 1949 I had the privilege of hearing David Wechsler, who developed the Wechsler Adult Intelligence Scale, give a lecture on the future of psychology. He

discussed the then red-hot issue of whether psychologists could do psychotherapy. He predicted the resistance of psychiatrists. He also predicted that the issue would not be settled at an American Psychiatric Association meeting or at an American Psychological Association meeting but "in the American marketplace."

I believe that the question of who will deal with the most pervasive, although relatively minor, of our mental health problems will be decided in the marketplace. If the nonmedical worker proves as acceptable and efficacious as the physician as well as less expensive, psychiatry can be expected to play less of a role in treating the so-called problems of living. In the treatment of serious mental illness, however, psychiatry is likely to remain dominant.

CONCLUSIONS

Psychiatry is growing in size, absolutely as well as in relation to general medicine. Although from the production point of view it has passed its peak growth, I expect psychiatry will continue to grow into the mid-1980s.

Psychiatry is growing in diversity. Parts of the profession are returning to basic biological and medical foundations; parts are undistinguishable in practice from other mental health professions; parts are under attack, and the outcome is unclear—particularly in social psychiatry. A critical area is the future of child psychiatry—or general psychiatry's role in dealing with families and children.

Psychiatry is undergoing severe criticism from within and without, but the demand for psychiatric services has shown no concomitant diminution and none is in sight.

In my opinion the test of the marketplace, both in the quality of the product and people's willingness to pay, is such that it would be unwise to limit psychiatry's services only to the mentally ill, although its responsibility and strength in this area are clear. It would be unwise and grandiose for psychiatry to claim a monopoly on treating the unhappinesses of the human condition, but it will be a challenge to see if the profession is worth its higher fees in this area.

The interface between psychiatry and social systems is an area in which psychiatry has in some ways overpromised. This interface is the site of hope for integrating prevention efforts with social concerns, for integrating scientific knowledge with humanistic concerns, and, in my opinion, it is a proper domain of psychiatry.

Finally, let me describe a more personal conclusion. I have been practicing something that has been loosely called "political psychiatry." Without bombast, this concerns my having been an administrator dealing with executive, legislative, judicial, and other affairs—serving as deputy director or director of NIMH for a decade. I think the issue of my accountability within these affairs should be brought out.

I have pondered over the fact that two of the groups now under the most severe attack from the public are politicians and psychiatrists. I think what these two groups have in common is the breakdown in trust on the part of their constituents and patients. The issue of trust is basic not only to psychiatry and developmental psychology, but also to the nature of government and governance. Thus it has been my purpose in writing this paper to be as accountable as I can, to state my view, and to continue to welcome dissent and criticism. There are battles to be won or lost in the marketplace, but we must realize we are not battling each other; the fight is on behalf of the mentally ill and in the pursuit of happiness for all.

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The Case for Involuntary Hospitalization of the Mentally Ill

BY PAUL CHODOFF, M.D.

The author examines three points of view on the question of society's right to involuntarily hospitalize a mentally ill individual. The "abolitionists" oppose involuntary hospitalization entirely; the medical model psychiatrists support the need for commitment under certain circumstances and so do the civil liberties lawyers, but by different standards. The author believes that with the current overreliance on the dangerousness standard, we are witnessing a pendular swing in which the rights of the mentally ill to be treated and protected are being set aside in the rush to give them their freedom. He favors a return to the use of medical criteria by psychiatrists, albeit with constructive legal safeguards.

I WILL BEGIN this paper with a series of vignettes designed to illustrate graphically the question that is my focus: under what conditions, if any, does society have the right to apply coercion to an individual to hospitalize him against his will, by reason of mental illness?

Case 1. A woman in her mid 50s, with no previous overt behavioral difficulties, comes to believe that she is worthless and insignificant. She is completely preoccupied with her guilt and is increasingly unavailable for the ordinary demands of life. She eats very little because of her conviction that the food should go to others whose need is greater than hers, and her physical condition progressively deteriorates. Although she will talk to others about herself, she insists that she is not sick, only bad. She refuses medication, and when hospitalization is suggested she also refuses that on the grounds that she would be taking up space that otherwise could be occupied by those who merit treatment more than she.

Case 2. For the past 6 years the behavior of a 42-year-old woman has been disturbed for periods of 3 months or longer. After recovery from her most recent episode she has been at home, functioning at a borderline level. A month ago she again started to withdraw from her environment. She pays increasingly less attention to her bodily needs, talks very little, and does not respond to questions or attention from those about her. She lapses into a mute state and lies in her bed in a totally passive fashion. She does not respond to other people, does not eat, and does not void. When her arm is raised from the bed it remains for several minutes in the position in which it is left. Her medical history and a physical examination reveal no evidence of primary physical illness.

Case 3. A man with a history of alcoholism has been on a binge for several weeks. He remains at home doing little else than drinking. He eats very little. He becomes tremulous and misinterprets spots on the wall as animals about to attack him, and he complains of "creeping" sensations in his body, which he attributes to infestation by insects. He does not seek help voluntarily, insists there is nothing wrong with him, and despite his wife's entreaties he continues to drink.

Case 4. Passersby and station personnel observe that a young woman has been spending several days at Union Station in Washington, D.C. Her behavior appears strange to others. She is finally befriended by a newspaper reporter who becomes aware that her perception of her situation is profoundly unrealistic and that she is, in fact, delusional. He persuades her to accompany him to St. Elizabeths Hospital, where she is examined by a psychiatrist who recommends admission. She refuses hospitalization and the psychiatrist allows her to leave. She returns to Union Station. A few days later she is found dead, murdered, on one of the surrounding streets.

Case 5. A government attorney in his late 30s begins to display pressured speech and hyperactivity. He is too busy to sleep and eats very little. He talks rapidly, becomes irritable when interrupted, and makes phone calls all over the country in furtherance of his political ambitions, which are to begin a campaign for the Presidency of the United States. He makes many purchases, some very expensive, thus running through a great deal of money. He is rude and tactless to his friends, who are offended by his behavior, and his job is in jeopardy. In spite of his wife's pleas he insists that he does not have the time to seek or accept treatment, and he refuses hospitalization. This is not the first such disturbance for this individual; in fact, very similar episodes have been occurring at roughly 2-year intervals since he was 18 years old.

Case 6. Passersby in a campus area observe two young women standing together, staring at each other, for over an hour. Their behavior attracts attention, and eventually the police take the pair to a nearby precinct station for questioning. They refuse to answer questions and sit mutely, staring into space. The police request some type of psychiatric examination but are informed by the city attorney's office that state law (Michigan) allows persons to be held for observation only if they appear obviously dangerous to themselves or others. In this case, since the women do not seem homicidal or suicidal, they do not qualify for observation and are released.

Less than 30 hours later the two women are found on the floor of their campus apartment, screaming and writhing in pain with their clothes ablaze from a self-made pyre. One

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Dr. Chodoff is Clinical Professor of Psychiatry, George Washington University School of Medicine. He is also in private practice at 1904 R St., N.W., Washington, D.C. 20009.

woman recovers; the other dies. There is no conclusive evidence that drugs were involved (1).

Most, if not all, people would agree that the behavior described in these vignettes deviates significantly from even elastic definitions of normality. However, it is clear that there would not be a similar consensus on how to react to this kind of behavior and that there is a considerable and increasing ferment about what attitude the organized elements of our society should take toward such individuals. Everyone has a stake in this important issue, but the debate about it takes place principally among psychiatrists, lawyers, the courts, and law enforcement agencies.

Points of view about the question of involuntary hospitalization fall into the following three principal groups: the "abolitionists," medical model psychiatrists, and civil liberties lawyers.

THE ABOLITIONISTS

Those holding this position would assert that in none of the cases I have described should involuntary hospitalization be a viable option because, quite simply, it should never be resorted to under any circumstances. As Szasz (2) has put it, "we should value liberty more highly than mental health no matter how defined" and "no one should be deprived of his freedom for the sake of his mental health." Ennis (3) has said that the goal "is nothing less than the abolition of involuntary hospitalization."

Prominent among the abolitionists are the "anti-psychiatrists," who, somewhat surprisingly, count in their ranks a number of well-known psychiatrists. For them mental illness simply does not exist in the field of psychiatry (4). They reject entirely the medical model of mental illness and insist that acceptance of it relies on a fiction accepted jointly by the state and by psychiatrists as a device for exerting social control over annoying or unconventional people. The anti-psychiatrists hold that these people ought to be afforded the dignity of being held responsible for their behavior and required to accept its consequences. In addition, some members of this group believe that the phenomena of "mental illness" often represent essentially a tortured protest against the insanities of an irrational society (5). They maintain that society should not be encouraged in its oppressive course by affixing a pejorative label to its victims.

Among the abolitionists are some civil liberties lawyers who both assert their passionate support of the magisterial importance of individual liberty and react with repugnance and impatience to what they see as the abuses of psychiatric practice in this field—the commitment of some individuals for flimsy and possibly self-serving reasons and their inhuman warehousing in penal institutions wrongly called "hospitals."

The abolitionists do not oppose psychiatric treatment when it is conducted with the agreement of those being treated. I have no doubt that they would try to gain the consent of the individuals described earlier to undergo treatment, including hospitalization. The psychiatrists in this group would be very likely to confine their treatment methods to psychotherapeutic efforts to influence the aberrant behavior. They would be unlikely to use drugs and would certainly eschew such somatic therapies as ECT. If efforts to enlist voluntary compliance with treatment failed, the abolitionists would not employ any means of coercion. Instead, they would step aside and allow social, legal, and community sanctions to take their course. If a human being should be jailed or a human life lost as a result of this attitude, they would accept it as a necessary evil to be tolerated in order to avoid the greater evil of unjustified loss of liberty for others (6).

THE MEDICAL MODEL PSYCHIATRISTS

I use this admittedly awkward and not entirely accurate label to designate the position of a substantial number of psychiatrists. They believe that mental illness is a meaningful concept and that under certain conditions its existence justifies the state's exercise, under the doctrine of parens patriae, of its right and obligation to arrange for the hospitalization of the sick individual even though coercion is involved and he is deprived of his liberty. I believe that these psychiatrists would recommend involuntary hospitalization for all six of the patients described earlier.

The Medical Model

There was a time, before they were considered to be ill, when individuals who displayed the kind of behavior I described earlier were put in "ships of fools" to wander the seas or were left to the mercies, sometimes tender but often savage, of uncomprehending communities that regarded them as either possessed or bad. During the Enlightenment and the early nineteenth century, however, these individuals gradually came to be regarded as sick people to be included under the humane and caring umbrella of the Judeo-Christian attitude toward illness. This attitude, which may have reached its height during the era of moral treatment in the early nineteenth century, has had unexpected and ambiguous consequences. It became overextended and partially perverted, and these excesses led to the reaction that is so strong a current in today's attitude toward mental illness.

However, reaction itself can go too far, and I believe that this is already happening. Witness the disastrous consequences of the precipitate dehospitalization that is occurring all over the country. To remove the protective mantle of illness from these disturbed people is to expose them, their families, and their communities to consequences that are certainly maladaptive and possibly irreparable. Are we really acting in accordance with their best interests when we allow them to "die with their rights on" (1) or when we condemn

them to a "preservation of liberty which is actually so destructive as to constitute another form of imprisonment" (7)? Will they not suffer "if [a] liberty they cannot enjoy is made superior to a health that must sometimes be forced on them" (8)?

Many of those who reject the medical model out of hand as inapplicable to so-called "mental illness" have tended to oversimplify its meaning and have, in fact, equated it almost entirely with organic disease. It is necessary to recognize that it is a complex concept and that there is a lack of agreement about its meaning. Sophisticated definitions of the medical model do not require only the demonstration of unequivocal organic pathology. A broader formulation, put forward by sociologists and deriving largely from Talcott Parsons' description of the sick role (9), extends the domain of illness to encompass certain forms of social deviance as well as biological disorders. According to this definition, the medical model is characterized not only by organicity but also by being negatively valued by society, by "nonvoluntariness," thus exempting its exemplars from blame, and by the understanding that physicians are the technically competent experts to deal with its effects (10).

Except for the question of organic disease, the patients I described earlier conform well to this broader conception of the medical model. They are all suffering both emotionally and physically, they are incapable by an effort of will of stopping or changing their destructive behavior, and those around them consider them to be in an undesirable sick state and to require medical attention.

Categorizing the behavior of these patients as involuntary may be criticized as evidence of an intolerably paternalistic and antitherapeutic attitude that fosters the very failure to take responsibility for their lives and behavior that the therapist should uncover rather than encourage. However, it must also be acknowledged that these severely ill people are not capable at a conscious level of deciding what is best for themselves and that in order to help them examine their behavior and motivation, it is necessary that they be alive and available for treatment. Their verbal message that they will not accept treatment may at the same time be conveying other more covert messages—that they are desperate and want help even though they cannot ask for it (11).

Although organic pathology may not be the only determinant of the medical model, it is of course an important one and it should not be avoided in any discussion of mental illness. There would be no question that the previously described patient with delirium tremens is suffering from a toxic form of brain disease. There are a significant number of other patients who require involuntary hospitalization because of organic brain syndrome due to various causes. Among those who are not overtly organically ill, most of the candidates for involuntary hospitalization suffer from schizophrenia or one of the major affective disorders. A growing and increasingly impressive body of evidence points to the presence of an important genetic-biological factor in these conditions; thus, many of them qualify on these grounds as illnesses.

Despite the revisionist efforts of the anti-psychiatrists, mental illness *does* exist. It does not by any means include all of the people being treated by psychiatrists (or by nonpsychiatrist physicians), but it does encompass those few desperately sick people for whom involuntary commitment must be considered. In the words of a recent article, "The problem is that mental illness is not a myth. It is not some palpable falsehood propagated among the populace by power-mad psychiatrists, but a cruel and bitter reality that has been with the human race since antiquity" (12, p.1483).

Criteria for Involuntary Hospitalization

Procedures for involuntary hospitalization should be instituted for individuals who require care and treatment because of diagnosable mental illness that produces symptoms, including marked impairment in judgment, that disrupt their intrapsychic and interpersonal functioning. All three of these criteria must be met before involuntary hospitalization can be instituted.

1. *Mental illness*. This concept has already been discussed, but it should be repeated that only a belief in the existence of illness justifies involuntary commitment. It is a fundamental assumption that makes aberrant behavior a medical matter and its care the concern of physicians.

2. *Disruption of functioning*. This involves combinations of serious and often obvious disturbances that are both intrapsychic (for example, the suffering of severe depression) and interpersonal (for example, withdrawal from others because of depression). It does not include minor peccadilloes or eccentricities. Furthermore, the behavior in question must represent symptoms of the mental illness from which the patient is suffering. Among these symptoms are actions that are imminently or potentially dangerous in a physical sense to self or others, as well as other manifestations of mental illness such as those in the cases I have described. This is not to ignore dangerousness as a criterion for commitment but rather to put it in its proper place as one of a number of symptoms of the illness. A further manifestation of the illness, and indeed, the one that makes involuntary rather than voluntary hospitalization necessary, is impairment of the patient's judgment to such a degree that he is unable to consider his condition and make decisions about it in his own interests.

3. *Need for care and treatment*. The goal of physicians is to treat and cure their patients; however, sometimes they can only ameliorate the suffering of their patients and sometimes all they can offer is care. It is not possible to predict whether someone will respond to treatment; nevertheless, the need for treatment and the availability of facilities to carry it out constitute essential preconditions that must be met to justify requir-

ing anyone to give up his freedom. If mental hospital patients have a right to treatment, then psychiatrists have a right to ask for treatability as a front-door as well as a back-door criterion for commitment (7). All of the six individuals I described earlier could have been treated with a reasonable expectation of returning to a more normal state of functioning.

I believe that the objections to this formulation can be summarized as follows.

1. The whole structure founders for those who maintain that mental illness is a fiction.

2. These criteria are also untenable to those who hold liberty to be such a supreme value that the presence of mental illness per se does not constitute justification for depriving an individual of his freedom; only when such illness is manifested by clearly dangerous behavior may commitment be considered. For reasons to be discussed later, I agree with those psychiatrists (13, 14) who do not believe that dangerousness should be elevated to primacy above other manifestations of mental illness as a sine qua non for involuntary hospitalization.

3. The medical model criteria are "soft" and subjective and depend on the fallible judgment of psychiatrists. This is a valid objection. There is no reliable blood test for schizophrenia and no method for injecting grey cells into psychiatrists. A relatively small number of cases will always fall within a grey area that will be difficult to judge. In those extreme cases in which the question of commitment arises, competent and ethical psychiatrists should be able to use these criteria without doing violence to individual liberties and with the expectation of good results. Furthermore, the possible "fuzziness" of some aspects of the medical model approach is certainly no greater than that of the supposedly "objective" criteria for dangerousness, and there is little reason to believe that lawyers and judges are any less fallible than psychiatrists.

4. Commitment procedures in the hands of psychiatrists are subject to intolerable abuses. Here, as Peszke said, "It is imperative that we differentiate between the principle of the process of civil commitment and the practice itself" (13, p. 825). Abuses can contaminate both the medical and the dangerousness approaches, and I believe that the abuses stemming from the abolitionist view of no commitment at all are even greater. Measures to abate abuses of the medical approach include judicial review and the abandonment of indeterminate commitment. In the course of commitment proceedings and thereafter, patients should have access to competent and compassionate legal counsel. However, this latter safeguard may itself be subject to abuse if the legal counsel acts solely in the adversary tradition and undertakes to carry out the patient's wishes even when they may be destructive.

Comment

The criteria and procedures outlined will apply most appropriately to initial episodes and recurrent attacks of mental illness. To put it simply, it is necessary to find a way to satisfy legal and humanitarian considerations and yet allow psychiatrists access to initially or acutely ill patients in order to do the best they can for them. However, there are some involuntary patients who have received adequate and active treatment but have not responded satisfactorily. An irreducible minimum of such cases, principally among those with brain disorders and process schizophrenia, will not improve sufficiently to be able to adapt to even a tolerant society.

The decision of what to do at this point is not an easy one, and it should certainly not be in the hands of psychiatrists alone. With some justification they can state that they have been given the thankless job of caring, often with inadequate facilities, for badly damaged people and that they are now being subjected to criticism for keeping these patients locked up. No one really knows what to do with these patients. It may be that when treatment has failed they exchange their sick role for what has been called the impaired role (15), which implies a permanent negative evaluation of them coupled with a somewhat less benign societal attitude. At this point, perhaps a case can be made for giving greater importance to the criteria for dangerousness and releasing such patients if they do not pose a threat to others. However, I do not believe that the release into the community of these severely malfunctioning individuals will serve their interests even though it may satisfy formal notions of right and wrong.

It should be emphasized that the number of individuals for whom involuntary commitment must be considered is small (although, under the influence of current pressures, it may be smaller than it should be). Even severe mental illness can often be handled by securing the cooperation of the patient, and certainly one of the favorable effects of the current ferment has been to encourage such efforts. However, the distinction between voluntary and involuntary hospitalization is sometimes more formal than meaningful. How "voluntary" are the actions of an individual who is being buffeted by the threats, entreaties, and tears of his family?

I believe, however, that we are at a point (at least in some jurisdictions) where, having rebounded from an era in which involuntary commitment was too easy and employed too often, we are now entering one in which it is becoming very difficult to commit anyone, even in urgent cases. Faced with the moral obloquy that has come to pervade the atmosphere in which the decision to involuntarily hospitalize is considered, some psychiatrists, especially younger ones, have become, as Stone (16) put it, "soft as grapes" when faced with the prospect of committing anyone under any circumstances.

THE CIVIL LIBERTIES LAWYERS

I use this admittedly inexact label to designate those members of the legal profession who do not in prin-

ciple reject the necessity for involuntary hospitalization but who do reject or wish to diminish the importance of medical model criteria in the hands of psychiatrists. Accordingly, the civil liberties lawyers, in dealing with the problem of involuntary hospitalization, have enlisted themselves under the standard of dangerousness, which they hold to be more objective and capable of being dealt with in a sounder evidentiary manner than the medical model criteria. For them the question is not whether mental illness, even of disabling degree, is present, but only whether it has resulted in the probability of behavior dangerous to others or to self. Thus they would scrutinize the cases previously described for evidence of such dangerousness and would make the decision about involuntary hospitalization accordingly. They would probably feel that commitment is not indicated in most of these cases, since they were selected as illustrative of severe mental illness in which outstanding evidence of physical dangerousness was not present.

The dangerousness standard is being used increasingly not only to supplement criteria for mental illness but, in fact, to replace them entirely. The recent Supreme Court decision in *O'Connor v. Donaldson* (17) is certainly a long step in this direction. In addition, "dangerousness" is increasingly being understood to refer to the probability that the individual will inflict harm on himself or others in a specific physical manner rather than in other ways. This tendency has perhaps been carried to its ultimate in the *Lessard v. Schmidt* case (18) in Wisconsin, which restricted suitability for commitment to the "extreme likelihood that if the person is not confined, he will do immediate harm to himself or others." (This decision was set aside by the U.S. Supreme Court in 1974.) In a recent Washington, D.C., Superior Court case (19) the instructions to the jury stated that the government must prove that the defendant was likely to cause "substantial physical harm to himself or others in the reasonably foreseeable future."

For the following reasons, the dangerousness standard is an inappropriate and dangerous indicator to use in judging the conditions under which someone should be involuntarily hospitalized. Dangerousness is being taken out of its proper context as one among other symptoms of the presence of severe mental illness that should be the determining factor.

1. To concentrate on dangerousness (especially to others) as the sole criterion for involuntary hospitalization deprives many mentally ill persons of the protection and treatment that they urgently require. A psychiatrist under the constraints of the dangerousness rule, faced with an out-of-control manic individual whose frantic behavior the psychiatrist truly believes to be a disguised call for help, would have to say, "Sorry, I would like to help you but I can't because you haven't threatened anybody and you are not suicidal." Since psychiatrists are admittedly not very good at accurately predicting dangerousness to others, the evidentiary standards for commitment will be very stringent. This will result in mental hospitals becoming prisons for a small population of volatile, highly assaultive, and untreatable patients (14).

2. The attempt to differentiate rigidly (especially in regard to danger to self) between physical and other kinds of self-destructive behavior is artificial, unrealistic, and unworkable. It will tend to confront psychiatrists who want to help their patients with the same kind of dilemma they were faced with when justification for therapeutic abortion on psychiatric grounds depended on evidence of suicidal intent. The advocates of the dangerousness standard seem to be more comfortable with and pay more attention to the factor of dangerousness to others even though it is a much less frequent and much less significant consequence of mental illness than is danger to self.

3. The emphasis on dangerousness (again, especially to others) is a real obstacle to the right-to-treatment movement since it prevents the hospitalization and therefore the treatment of the population most amenable to various kinds of therapy.

4. Emphasis on the criterion of dangerousness to others moves involuntary commitment from a civil to a criminal procedure, thus, as Stone (14) put it, imposing the procedures of one terrible system on another. Involuntary commitment on these grounds becomes a form of preventive detention and makes the psychiatrist a kind of glorified policeman.

5. Emphasis on dangerousness rather than mental disability and helplessness will hasten the process of deinstitutionalization. Recent reports (20, 21) have shown that these patients are not being rehabilitated and reintegrated into the community, but rather, that the burden of custodialism has been shifted from the hospital to the community.

6. As previously mentioned, emphasis on the dangerousness criterion may be a tactic of some of the abolitionists among the civil liberties lawyers (22) to end involuntary hospitalization by reducing it to an unworkable absurdity.

DISCUSSION

It is obvious that it is good to be at liberty and that it is good to be free from the consequences of disabling and dehumanizing illness. Sometimes these two values are incompatible, and in the heat of the passions that are often aroused by opposing views of right and wrong, the partisans of each view may tend to minimize the importance of the other. Both sides can present their horror stories—the psychiatrists, their dead victims of the failure of the involuntary hospitalization process, and the lawyers, their Donaldsons. There is a real danger that instead of acknowledging the difficulty of the problem, the two camps will become polarized, with a consequent rush toward extreme and untenable solutions rather than working toward reasonable ones.

The path taken by those whom I have labeled the

abolitionists is an example of the barren results that ensue when an absolute solution is imposed on a complex problem. There are human beings who will suffer greatly if the abolitionists succeed in elevating an abstract principle into an unbreakable law with no exceptions. I find myself oppressed and repelled by their position, which seems to stem from an ideological rigidity which ignores that element of the contingent immanent in the structure of human existence. It is devoid of compassion.

The positions of those who espouse the medical model and the dangerousness approaches to commitment are, one hopes, not completely irreconcilable. To some extent these differences are a result of the vantage points from which lawyers and psychiatrists view mental illness and commitment. The lawyers see and are concerned with the failures and abuses of the process. Furthermore, as a result of their training, they tend to apply principles to classes of people rather than to take each instance as unique. The psychiatrists, on the other hand, are required to deal practically with the singular needs of individuals. They approach the problem from a clinical rather than a deductive stance. As physicians, they want to be in a position to take care of and to help suffering people whom they regard as sick patients. They sometimes become impatient with the rules that prevent them from doing this.

I believe we are now witnessing a pendular swing in which the rights of the mentally ill to be treated and protected are being set aside in the rush to give them their freedom at whatever cost. But is freedom defined only by the absence of external constraints? Internal physiological or psychological processes can contribute to a throttling of the spirit that is as painful as any applied from the outside. The "wild" manic individual without his lithium, the panicky hallucinator without his injection of fluphenazine hydrochloride and the understanding support of a concerned staff, the sodden alcoholic—are they free? Sometimes, as Woody Guthrie said, "Freedom means no place to go."

Today the civil liberties lawyers are in the ascendancy and the psychiatrists on the defensive to a degree that is harmful to individual needs and the public welfare. Redress and a more balanced position will not come from further extension of the dangerousness doctrine. I favor a return to the use of medical criteria by psychiatrists—psychiatrists, however, who have been chastened by the buffeting they have received and are quite willing to go along with even strict legal safeguards as long as they are constructive and not tyrannical.

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Drug and Psychotherapy Interactions in Depression

BY LINO COVI, M.D., RONALD S. LIPMAN, PH.D., RENATO D. ALARCON, M.D., AND VIRGINIA K. SMITH

The authors conducted a series of multiple regression analyses of data from depressed patients. They found that 8 factors consistently predicted treatment response: a lower initial level of distress, imipramine treatment, a positive attitude toward group psychotherapy, and a good employment history predicted lower posttreatment distress levels; estrogen maintenance treatment was related to better response to diazepam, and a low level of intelligence predicted better response to both diazepam and imipramine; and a low initial level of interpersonal sensitivity and a significant other's having an unfavorable attitude toward psychiatric treatment were associated with better response to group psychotherapy.

ONE OF THE MOST VALUABLE contributions that clinical research can make to the practice of psychiatry is to add to the repertoire of possible predictors of response to treatment.

The prevalence of depressive illness is quite high. Klerman (1) maintained that 10% of the population of the United States will have an episode of clinical depression at some time in their lives. This high incidence of depression will be reflected in the proportion of depressive patients to patients with other illnesses being treated by clinicians. An increased ability to choose the right treatment for the right patient is therefore a particularly important contribution to public health. Current clinical psychiatric practice tends to prefer the combination of pharmacotherapy and psychotherapy in the treatment of depression in outpatient settings. The differential indications for psychotherapy and pharmacotherapy and their combinations have been insufficiently studied (2). This report presents some experimental data bearing on the prediction of response to six types of treatment of depression in a homogeneous patient population.

The following combinations were studied: 1) group psychotherapy with placebo, 2) minimal supportive psychotherapy with placebo, 3) imipramine in combination with group therapy, 4) imipramine in combination with minimal supportive psychotherapy, 5) diazepam in combination with group psychotherapy, and 6) diazepam in combination with minimal supportive psychotherapy. The study sample consisted of women who ranged in age from 20 to 50, who were nonpsychotic, and who had a depression severity score of at least 7 on the Raskin Depression Screen (3).

The study was conducted in the outpatient departments of the Phipps Psychiatric Clinic of Johns Hopkins Hospital in East Baltimore and the Gundry Hospital in West Baltimore. Two experienced psychiatrists (Joseph H. Pattison, M.D., and James E. Smith III, M.D.) treated all of the psychiatric patients at both clinics. Written informed consent was obtained from the patients included in the study. After a 2-week placebo washout period, the patients were randomly assigned to one of the six types of treatment described above for a 16-week active treatment period. Further details of the design and techniques used in the study have been described elsewhere (4, 5).

Three groups of individuals provided improvement ratings: 1) the patients completed two self-rating scales, the Hopkins Symptom Check List (HSCL) (5) and the Profiles of Mood State (POMS) (5); 2) the treating psychiatrist rated improvement using global mood scales derived from the POMS; and 3) a person significant to the patient provided global improvement ratings and ratings of the quality of his relationship with the patient on the Barrett-Lennard Relationship Inventory (RI) (6). The implications of using a significant other person's ratings in research have been reported elsewhere (7).

Other data obtained to provide possible predictors of improvement included such demographic information about the patient as age, marital status, and education; physician ratings, such as the Brief Psychiatric Rating Scale (BPRS) (8); chronicity of the patient's illness; and the patient's medication history. The patient also rated the treating physician on the RI, and a social worker who interviewed the significant other person rated that person's views toward psychiatric treatment.

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Dr. Covi is Associate Professor of Psychiatry and Ms. Smith is Biostatistician, Johns Hopkins University School of Medicine, Baltimore, Md. Dr. Lipman is Chief, Clinical Studies Section, Psychopharmacology Research Branch, National Institute of Mental Health, Rockville, Md., and Dr. Alarcon is Assistant Professor of Psychiatry, Universidad Peruana Cayetano Heredia, Lima, Peru. Address reprint requests to Dr. Covi at the Henry Phipps Psychiatric Clinic, 601 North Broadway, Baltimore, Md. 21205.

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METHOD

Three hundred forty-six patients were screened for the study; 279 of these were accepted for treatment, and 218 completed the placebo washout period and entered the active phase. Two hundred twelve of these 218 patients returned for at least two visits after beginning active medication. One hundred forty-nine patients completed the entire 16-week active treatment phase with reasonable compliance to study procedures.

The typical patient was married (71% of the 149 patients who completed the study), was Protestant (50%), worked as a housewife (60%), was in Hollingshead's social class IV (9) (39%), and had had at least one previous depressive episode (60%). Most of the patients were chronically ill. The mean length of their present illness was 41 weeks; the median was 25 weeks. Covariance analyses, using a 2×3×2 design (therapy×medication×clinic), revealed a general and strong superiority of imipramine over diazepam[1] and placebo according to doctors' and patients' ratings of symptoms and mood. These findings have been reported in detail elsewhere (4, 5).

For the present analyses we used 22 potential predictors of improvement selected on the basis of previous research findings and/or from hypotheses developed on the basis of clinical experience. This approach provided a further test of the consistency of the results of the previous covariance analyses (4, 5).[2]

The 22 predictors are as follows:

1. The initial level of distress indicated on each of the measures of improvement. This predictor has often proven to be the single most powerful predictor of outcome (10).
2. Type of psychotherapy, i.e., group psychotherapy versus minimal supportive psychotherapy.
3. Drugs, i.e., imipramine, diazepam, or placebo.
4. Age as a continuous variable (3).
5. Marital status, i.e., never married, currently married, or formerly married (3).
6. Occupation, grouped into the seven categories described by Hollingshead (9).
7. Social class, using the five classes of the Hollingshead classification (9). Variables 6 and 7 were found by Downing and associates (11) to be predictive of drug treatment response.
8. Chronicity in terms of duration of present illness in weeks, which in psychotherapy studies has predicted outcome (12).
9. Peak of illness according to a 7-point rating designed to reflect when the worst point of distress occurred, from less than 1 week to 1 year or more before entering the study.
10. The patient's attitude toward assignment to group psychotherapy, as judged by the physicians who interviewed the patient before the study began on a 5-point scale, from very eager to very reluctant.
11. Concurrent treatment with an estrogen type of medication, either as part of an oral contraceptive regimen or as part of a postmenopausal replacement treatment. (Surprisingly, estrogen treatment showed a high level of significance in interaction with medication, i.e., in enhancing the therapeutic effect of diazepam, in several multivariate covariance analyses in this study).
12. Clinical setting, whether at Phipps Psychiatric Clinic or Gundry Hospital.
13. The level of distress noted by the patient on the interpersonal sensitivity factor of the HSCL before beginning active treatment. The 7 items included in this factor are as follows: feeling critical of others, feeling easily annoyed or irritated, having temper outbursts you could not control, your feelings being easily hurt, feeling others do not understand you or are unsympathetic, feeling that people are unfriendly or dislike you, and feeling inferior to others. This factor was felt to describe personality characteristics common to individuals who tend to adjust poorly to group interactions.
14. The four types of depression described by Overall and Hollister (13), i.e., anxious, hostile, retarded, and agitated, as determined by computer assignment on the basis of the BPRS rating given by the intake physicians. Overall and Hollister reported interactions between tranquilizers and antidepressant medications and these types of depression.
15. The attitude toward psychiatric treatment expressed by the significant other person in the patient's life as rated by a social worker after an interview with this person. On the basis of clinical impression, it was felt that a favorable attitude of the family of the patient toward psychiatric treatment would facilitate the patient's response to treatment.
16. The attitude of the patient toward the treating physician as measured by the total score derived from the RI. A previous study (14), in which two of us participated (L.C. and R.S.L.), showed that a positive relationship between the patient and the physician predicted a favorable treatment outcome.
17. An endogenomorphic score developed to conform with Donald Klein's theory of differential drug response in endogenomorphic and nonendogenomorphic depression (15). It should be noted that Klein's theory of differential drug response also included neurotic and reactive subtypes that were not really measured by the endogenomorphic dimension.
18. An endogenomorphic score similar to the one used for predictor 17 but derived from the patient's self-rating.
19. The employment history of the patient categorized into the four classes of good, fair, poor, and none.
20. The patient's intelligence as judged by the treat-

[1]The dosage for imipramine ranged from 100 to 200 mg a day (average=125 mg a day). The dosage for diazepam ranged from 10 to 20 mg a day (average=12.5 mg a day).

[2]Multiple regression analysis permits a statistical test of the individual effect of each variable in the predictor equation by adjusting the score of each item in the equation for the effect of all other items in the equation.

ing physician on the first visit and classified on a 7-point scale, from much above average to much below average.

21. The attitude of the patient toward the study medications as determined by the treating physician on the first visit on a 7-point scale, ranging from markedly positive to markedly negative.

22. The patient's attitude toward group therapy as determined by the treating physician on the first visit on a 7-point scale, ranging from markedly positive to markedly negative. Variables 19, 20, and 22 were found to be predictive of treatment outcome by Downing and associates (11).

The predictor pool, consisting of these 22 predictors and their interactions with type of medication and with type of therapy, was searched, using a stepwise multiple regression approach (16), to determine the variables or combination of variables that most reliably predicted improvement for each of the improvement criteria in the three areas of depression, anxiety, and global improvement.

The statistical program used for the multiple regression analyses included only patients who had no missing data. In this regard, the patient sample of 212 comprised the 149 patients who completed the full 16 weeks of the study with reasonable adherence to protocol plus an additional 63 patients who completed at least 2 weeks of the study with adherence to protocol. These 212 patients represented the data base for potential analyses. In the case of the 63 patients who did not complete the full study (on average, these patients completed 11 weeks of the study), their scores on their last visits were used in the end-point analysis reported here. Their last valid visit scores were treated as equivalent to the 16th-week scores of the patients who completed the 16 weeks of treatment. An average of 179 patients entered into the multiple regression analysis.

The attrition in patients was entirely due to missing data, which included data missing on the following variables: 1) age (2 missing), 2) marital status (2 missing), 3) occupation (3 missing), 4) social class (2 missing), 5) attitude toward psychotherapy (5 missing), 6) HSCL interpersonal sensitivity factor score at week 3 (1 missing), and 7) attitude of the significant other toward psychiatric treatment (13 missing).

RESULTS

Eight measures were found to be highly consistent predictors of outcome, i.e., they were significantly related to outcome in at least one-third of the predictor analyses (see tables 1–3). These 8 predictors include 4 variables that predicted outcome regardless of treatment assignment, 2 that predicted outcome in com-

TABLE 1
Multiple Regression Analysis Data for Consistent Predictors of Change in Depressive Symptoms*

Item	Significance Levels of Consistent Predictors								Analysis
	1	3	11	13	15	19	20	22	
Patients' ratings									
58-item-HSCL depression factor (11 items)									
Main effects	n.s.	.0001	n.s.	.0002	n.s.	n.s.	n.s.	.01	—
Drug interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	.01	.01	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.39
72-item-HSCL depression factor (10 items)									
Main effects	.02	.0001	n.s.	n.s.	n.s.	n.s.	n.s.	.01	—
Drug interactions	n.s.	n.s.	.03	n.s.	n.s.	n.s.	n.s.	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	.01	.03	n.s.	n.s.	.05	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.50
52-item-POMS-derived depression factor									
Main effects	.01	.0001	n.s.	.01	n.s.	n.s.	n.s.	.01	—
Drug interactions	n.s.	n.s.	.01	n.s.	n.s.	n.s.	.02	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	.01	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.45
Doctors' rating									
POMS-derived depression factor									
Main effects	.01	.01	n.s.	n.s.	n.s.	.01	n.s.	.03	—
Drug interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	n.s.	.02	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.28

* The predictors were studied alone (main effects), in combination with type of medication (drug interactions), and in combination with type of therapy (therapy interactions).

TABLE 2
Multiple Regression Analysis Data for Consistent Predictors of Change in Anxiety Symptoms*

Item	Significance Levels of Consistent Predictors								Analysis
	1	3	11	13	15	19	20	22	
Patients' ratings									
58-item-HSCL anxiety factor (7 items)									
Main effects	.0001	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	.01	—
Drug interactions	n.s.	n.s.	.03	n.s.	n.s.	n.s.	.04	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.38
72-item-HSCL anxiety-somatic factor (5 items)									
Main effects	.0001	.01	n.s.	n.s.	n.s.	.04	n.s.	.03	—
Drug interactions	n.s.	n.s.	n.s.	.03	n.s.	n.s.	.05	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.42
72-item-HSCL anxiety-phobic-panic factor (3 items)									
Main effects	.0001	.01	n.s.	n.s.	n.s.	n.s.	n.s.	.05	—
Drug interactions	n.s.	n.s.	.04	n.s.	n.s.	n.s.	n.s.	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.43
52-item-POMS-derived anxiety factor									
Main effects	.0001	.0002	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
Drug interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	.02	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.38
Doctors' rating									
POMS-derived anxiety factor									
Main effects	.04	n.s.	n.s.	n.s.	n.s.	.0002	n.s.	.01	—
Drug interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
Therapy interactions	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.28

* The predictors were studied alone (main effects), in combination with type of medication (drug interactions), and in combination with type of therapy (therapy interactions).

bination with drug assignment, and 2 that predicted outcome in combination with therapy assignment.

The general predictors of outcome are as follows: predictor 1, initial level of distress (a low initial level of distress correlated with good outcome); predictor 3, drug assignment (patients who received imipramine improved substantially more than patients who received diazepam or placebo, and those who received diazepam or placebo did not reliably differ from each other); predictor 19, employment history (poor employment history before entering the study predicted less improvement than did good, fair, or no employment history); and predictor 22, the patient's attitude toward group psychotherapy (a positive attitude toward group psychotherapy predicted better outcome). A representative sample of means for these main effect predictors is presented in table 4.

The predictors that interacted with type of medication (see figures 1 and 2) were as follows: predictor 11, concurrent treatment with an estrogen type of medication, which was related to a better response to diazepam; and predictor 20, intelligence (a lower level of intelligence predicted a relatively better response to both imipramine and diazepam).

The predictors that interacted with type of therapy (see figures 3 and 4) were as follows: predictor 13, initial level of distress on interpersonal sensitivity (patients who had lower scores felt less critical of others, less inferior to others, etc., and did relatively better in group psychotherapy than in minimal supportive psychotherapy); predictor 15, attitude toward psychiatric treatment expressed by the significant other person in the patient's life as rated by a social worker (a relatively less positive attitude toward treatment predicted that the patient would do better in group psychotherapy than in minimal supportive therapy).

DISCUSSION

Given the present state of the art of psychiatric treatment, the clinician's decision to assign depressed patients to psychotherapy and/or pharmacotherapy is largely dependent on clinical intuition. Although covar-

TABLE 3
Multiple Regression Analysis Data for Consistent Predictors of Change in Global Improvement Ratings*

Item	Significance Levels of Consistent Predictors								Analysis
	1	3	11	13	15	19	20	22	
Patients' global improvement ratings (7-point scale**)									
Main effects	—	.001	n.s.	.01	n.s.	.04	n.s.	.01	—
Drug interactions	—	n.s.	n.s.	n.s.	n.s.	n.s.	.01	.04	—
Therapy interactions	—	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.35
Doctors' global improvement ratings (7-point scale**)									
Main effects	—	.0001	n.s.	n.s.	n.s.	.0001	n.s.	.02	—
Drug interactions	—	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	—
Therapy interactions	—	n.s.	n.s.	.01	.03	n.s.	n.s.	n.s.	—
R^2 (for 22 predictors)	—	—	—	—	—	—	—	—	.41

* The predictors were studied alone (main effects), in combination with type of medication (drug interactions), and in combination with type of therapy (therapy interactions).
** The 7-point scale ranged from very much worse to no change to very much better.

TABLE 4
Adjusted Means for Consistent Main Effects of 4 Predictors by Various Outcome Criteria

Item	Doctors' Rating	Patients' Ratings		
	POMS-Derived Depression Factor	72-Item-HSCL Anxiety-Somatic Factor (5 items)	72-Item-HSCL Depression Factor (10 items)	52-Item-POMS-Derived Depression Factor
Predictor 1				
High initial level of distress	2.654	1.915	1.445	1.601
Low initial level of distress	1.348	0.314	0.588	0.689
Predictor 3				
Placebo	2.392	1.149	1.091	1.324
Imipramine	1.730	0.834	0.580	0.712
Diazepam	2.314	1.115	1.200	1.415
Predictor 19				
Good employment history	1.727	0.961	—	—
Fair employment history	2.255	0.962	—	—
Poor employment history	2.276	1.301	—	—
No employment history	3.149	1.031	—	—
Predictor 22				
More positive attitude toward group psychotherapy	1.673	0.750	0.582	0.826
Less positive attitude toward group psychotherapy	2.678	1.261	1.376	1.511

iance analyses of controlled studies typically give important information concerning the relative efficacy of various drugs and psychotherapies, the more complex problem of sorting out the relative contributions of the many noncontrolled factors that can influence treatment outcome is better served by stepwise multiple regression analyses.

We have attempted to be conservative in interpreting the results of our study by stressing only the consistent predictors of response that showed a high level of significance and that were found reliably related to outcome in more than one-third of the analyses performed. Furthermore, these predictors were selected on the basis of findings from previous studies or on the basis of extensive clinical experience. We therefore feel that some of the more serious objections to

FIGURE 1
Representative Sample of Effects of Interactions of Drugs and Predictor 11 on Patients' Adjusted Mean Distress Levels

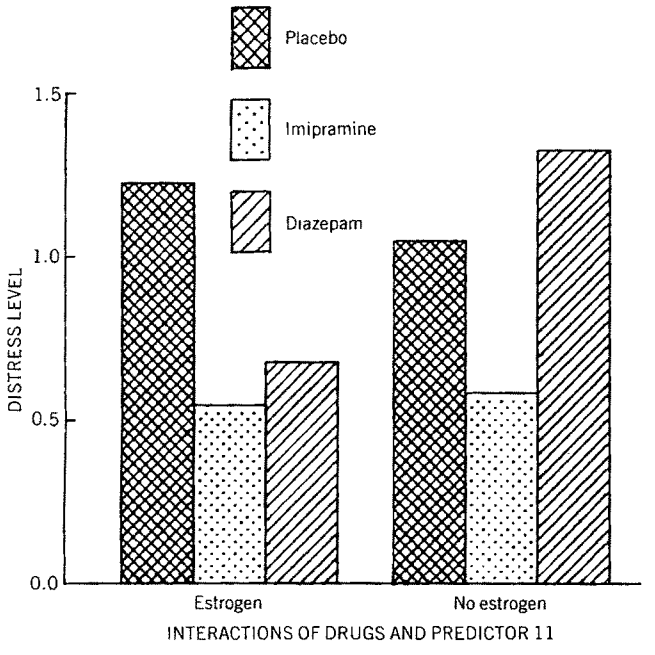
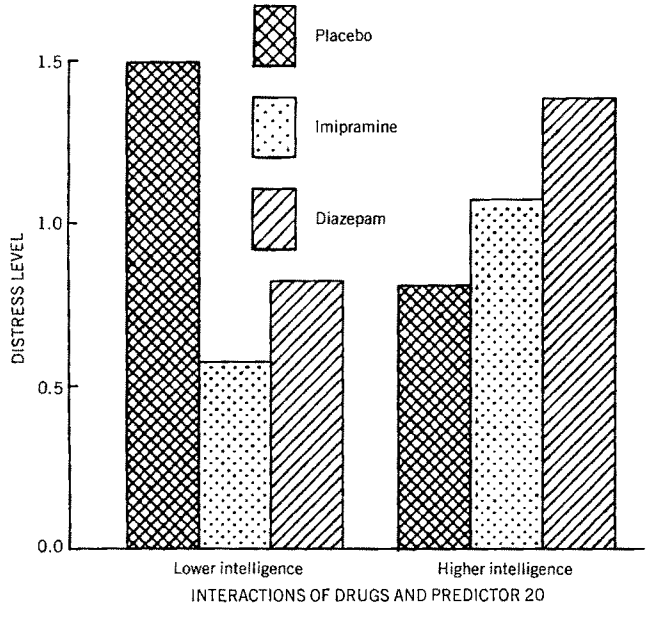


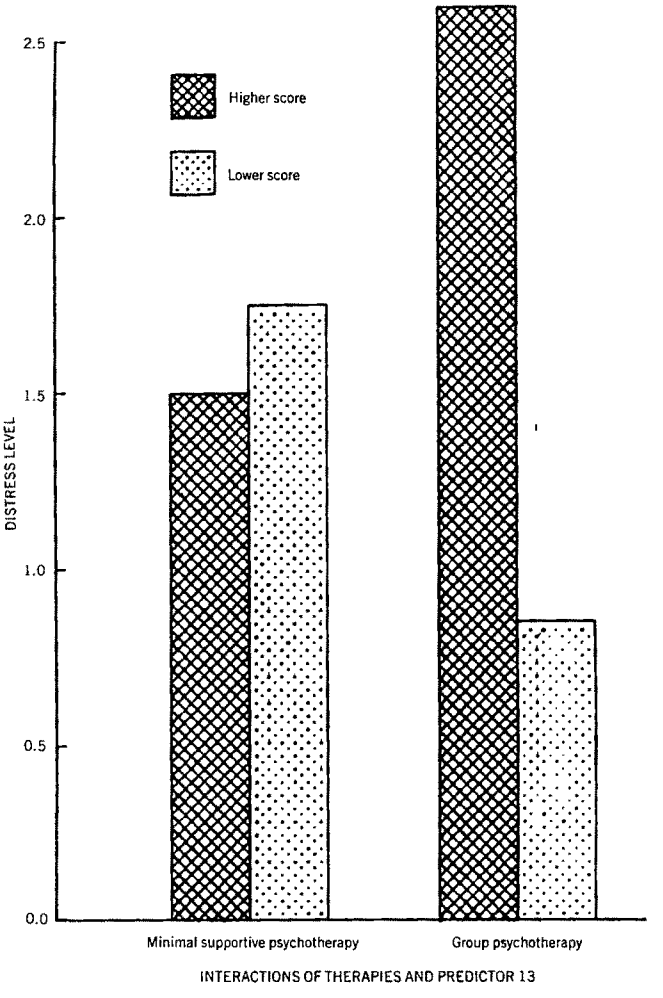
FIGURE 2
Representative Sample of Effects of Interactions of Drugs and Predictor 20 on Patients' Adjusted Mean Distress Levels



our statistical approach (17) have been minimized.

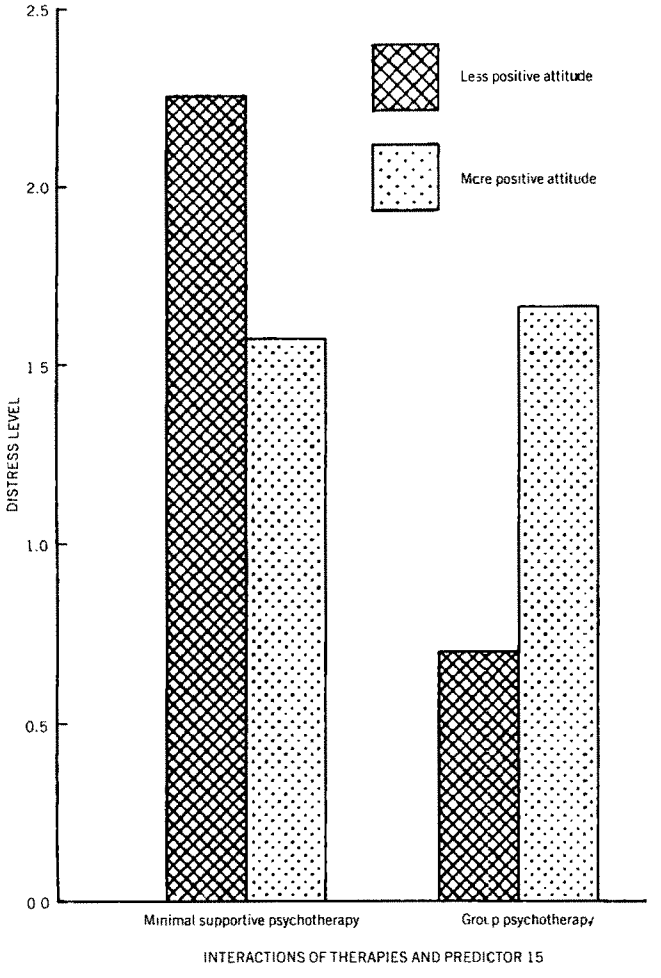
What practical implications can the clinician draw from this study? He can expect to see the most improvement in depression and anxiety by the end of 16 weeks of treatment among female patients who were most distressed at the onset of treatment and those who were initially favorably disposed toward more intensive psychotherapy, including group psychotherapy. On the other hand, female patients with a poor employment history would be expected to show relatively less improvement than those with a good employment history by the end of 16 weeks of treatment. He can count on reliably better results with imipramine than with diazepam or placebo, with either group or minimal supportive psychotherapy. On the other hand, our data suggest that female patients receiving estrogen hormones may respond quite well to diazepam. Patients who the clinican feels are less intelligent than average may do relatively better on active medication than patients he considers above average in intelligence, but the latter group may improve relatively more on treatment with inert medication.

FIGURE 3
Representative Sample of Effects of Interactions of Therapies and Predictor 13 on Patients' Adjusted Mean Distress Levels



The person who is easily hurt, who feels critical of others, and who is easily irritated should be expected to do less well in group therapy. On the other hand, if those who are important to a female patient are not favorably disposed toward psychiatric treatment, group therapy may well be the treatment of choice.

FIGURE 4
Representative Sample of Effects of Interactions of Therapies and Predictor 15 (Attitude of Significant Other) on Patients' Adjusted Mean Distress Levels



COMMENT

Despite our care in selecting consistent predictors, the results of the kind of multiple regression analyses presented in this paper are often difficult to replicate in independent samples. Until such replication is available, these findings should be considered tentative.

For instance, contrary to other, similar studies, our study has shown few demographic factors to be predictive of treatment response; this difference may be due to the homogeneity of our population, which was limited to white women between the ages of 20 and 50 years. It should also be noted that our predictor analyses were based on 16 weeks of treatment, in contrast to most other predictor studies, which employed much shorter treatment periods.

The finding of a positive therapeutic interaction between estrogen and diazepam is a most tentative finding, but it is suggestive of a possible direction for further investigation. In this regard, our initial expectation was that imipramine would be more influenced than diazepam by the presence or absence of estrogen (18). We would further caution that only a small number of patients (15 per medication), not randomly assigned, and on a variety of estrogen-containing medications of varying dosages of estrogen, were grouped together for analytic purposes. Further research is therefore needed to more clearly test the validity of this clinically important potential drug interaction.

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Short Versus Long Hospitalization: A Prospective Controlled Study. IV. One-Year Follow-Up Results for Schizophrenic Patients

BY IRA D. GLICK, M.D., WILLIAM A. HARGREAVES, PH.D., JOAN DRUES, M.A., AND JONATHAN A. SHOWSTACK

The authors compared treatment results for 141 schizophrenic patients randomly assigned to short-term or long-term hospitalization. Test results indicated that the long-term group was functioning significantly better one year after admission according to global measures only. The authors caution that the differences between the two groups, although statistically reliable, were modest and may have been confounded by the amount of psychotherapy the patients received after hospitalization. Although there appears to be a general advantage to the long-term approach, further work will be needed to identify patient subgroups for whom this more expensive treatment is cost effective.

THIS STUDY WAS DESIGNED to determine the relative effectiveness of short-term hospitalization (i.e., 21–28 days) compared with long-term hospitalization (i.e., 90–120 days) for schizophrenic patients in need of hospital care for whom both types of treatment are judged clinically feasible. We have previously reported limited preliminary results (1) and complete results (2, 3) of the inpatient phase of this study of a group of schizophrenic patients and an additional group of nonschizophrenic patients. This paper reports the results of a one-year follow-up of our study group of 141 schizophrenic patients; the next paper in this series, which follows in this issue of the *Journal*, reports on our group of 74 nonschizophrenic patients.

There have been virtually no controlled evaluations of alternative methods of in-hospital treatment for acute schizophrenic episodes, such as brief hospital stay versus more extended stay. Treatment approaches that involve longer hospitalization are being discarded even though there is very little evidence about the effect this has on the lives of patients.

We have discussed elsewhere (1) the few studies that have some relevance to this question. Two additional studies have since come to our attention. Smith and associates (4), using a nonrandomized design, compared outcomes of first hospital admissions to a regional community mental health center (which involved a mean of 47 days of hospitalization) with those of patients given traditional state hospital care (which involved a mean of 137 days of hospitalization). Most of the patients they studied were diagnosed as having personality disorder (64%), 18% were diagnosed as neurotic, and 13% as psychotic. Smith and associates reported that over a four-year follow-up period,

> The [community mental health] center group had fewer days hospitalized, less disability, less total cost per patient and per capita, as well as a lower cost-benefit ratio than traditional state hospitals. Neither system of care, however, was successful with a small hard-core group of long-term patients.

Schooler and associates (5), reporting on the patients studied by the National Institute of Mental Health-Psychopharmacology Service Center (NIMH-PSC) collaborative study group, found that patients who received placebo while hospitalized stayed in the hospital an average of 6 weeks longer than patients who received phenothiazines but were less likely to be rehospitalized. In this study as well as in that of Smith and associates (4) the length of hospital stay was confounded with other variables, preventing any confident conclusions about length of stay.

METHOD

The inpatient phase of our study was conducted on the Clinical Research Ward of the Langley Porter Institute. This is a coed ward with a 26-patient capacity, operated as an open door unit that can accept only voluntary admissions. Patients' fees are adjusted according

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The authors are all with the Clinical Research Ward, Langley Porter Institute, University of California, San Francisco, Calif. 94143. Dr. Glick is Associate Professor of Psychiatry in Residence, Dr. Hargreaves is Associate Professor of Medical Psychology in Residence, Ms. Drues is Staff Research Associate, and Mr. Showstack is Staff Research Associate II.

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to their ability to pay. It has a high staff:patient ratio, as is usual at a university hospital (1).

Treatment Assignment

All of the patients consecutively admitted to the Clinical Research Ward during a 26-month period were randomly assigned to either short-term (21–28 days) or long-term (90–120 days) hospitalization. After the study was explained to the patients, all of them agreed to participate. However, some potential patients probably chose not to seek admission because the nature of the study was explained to the referring person at the time of the initial inquiry about admitting a patient. Patients remained in the long- or short-term group regardless of final diagnosis. This removed any temptation to bias the diagnostic process to obtain some favored treatment for a particular patient. Although the definitions of "short-term" and "long-term" were target lengths of stay for patients in the study, in practice these produced a very good separation in the distributions of actual lengths of stay in the two groups, as has been previously reported (2, 3).

Diagnosis

The clinical diagnosis for research purposes was made by the staff psychiatrist in charge of the ward and by a research psychiatrist (I.D.G.) after the patient had been examined and studied (usually at least 10 days after admission). After a diagnosis of schizophrenia was agreed on as the most appropriate diagnosis for the patient, both psychiatrists independently rated each patient using the Certainty of Diagnosis of Schizophrenia Form developed by Mosher (6). The results of this assessment indicate that there was no significant difference between the short-term and long-term groups in the certainty of diagnosis of schizophrenia (chi-square test). Ninety percent of those diagnosed as schizophrenic were rated as either "definitely" or "almost definitely" schizophrenic.

Treatment Program

Goals and strategies for the short-term and long-term groups have already been described (1), including our strategy to balance the use of medication (2). We also measured the actual frequency of various treatment activities for a sample of patients and found that, within limits, the treatment was qualitatively and quantitatively close to that prescribed. Our impression is that the quality of the treatment was at least as good as, if not better than, that in other comparable clinical settings. Patients' impressions of the treatment were also measured. There was no difference between the short-term and long-term groups in patients' ratings of the helpfulness of treatment (7).

Aftercare can affect subsequent functioning. For a variety of reasons, we chose to use existing community services rather than provide a controlled aftercare program. Because variations in posthospital treatment can confound the direct effect of short versus long inpatient stay on one-year outcomes, these effects will be examined in some of the analyses reported below.

Patients

Of the 141 schizophrenic patients studied, 71 were short-term and 70 were long-term. The schizophrenic patients fit the selection criteria used in the NIMH-PSC multihospital study of phenothiazines (8), except that we relaxed the criterion requiring that the patient spend 12 months in the community after any previous hospitalization. There were no significant differences between the long-term and short-term groups in regard to sex, marital status, race, or age (chi-square test or t test). Despite random assignment, t tests showed that the long-term group had more education (mean difference, one year, $p<.05$), a higher socioeconomic status (mean difference, .5 on a 5-point scale, $p<.01$), and better premorbid adjustment (mean difference, .8 on a 13-point scale, $p<.05$). These variables will therefore be examined for their relation to outcome.

At one-year follow-up we obtained nearly complete data for 132 of the original sample of 141 schizophrenic patients. Some items are missing for these patients. We had no data for 3 patients and only partial data (obtained from interviews with significant others) for 6 patients, 2 of whom had committed suicide. This low attrition rate, plus inspection of the characteristics of short-term and long-term dropouts, indicated that differential attrition did not reduce the comparability of the two treatment groups.

Measuring Instruments

Seven instruments were used to evaluate outcome one year after admission: 1) the Health-Sickness Rating Scale (HSRS) (9), 2) the Psychiatric Evaluation Form (PEF) (10), 3) the Katz Adjustment Scales (KAS) (11) for patients, 4) the KAS for patients' relatives, 5) a historical information form (HIF) based in part on the Brief Follow-Up Rating Form developed by Soskis (12), 6) the Patient's Self-Evaluation of Current Status Form (PSECS) (13), and 7) the Family Members' Evaluation of Current Status of Patient Form (FMECS) (13). Baseline measures on the first five were obtained at admission, and the last two call for direct before and after comparisons. These measurements were more fully described in previous publications (1, 2).

For the analyses reported in this paper we computed a summary score for the social and treatment history data that consisted of the sum of standard scores on six items: 1) ratings of overall functioning, 2) ratings of role functioning, 3) ratings of percent of time functioning, 4) number of months unemployed, 5) number of hospitalizations, and 6) total number of days hospitalized. These scores were standardized separately at admission and at one year, using the total group of 215 schizophrenic and nonschizophrenic patients. In interpreting this summary score of historical data one should keep in mind that admission ratings apply to the average social functioning of the patient during the two

years before admission, but the other global ratings (the HSRS and the PEF overall severity of illness item) apply to only the week before admission. This was probably responsible for the fact that the baseline summary score of historical data proved to be the best predictor of all three global ratings at one year.

RESULTS

Global Outcome

Global outcome was measured using the HSRS, the overall severity item of the PEF, and the summary score of historical data (see table 1). The initial analy-

TABLE 1
Summary of Outcome Measures for Schizophrenic Patients One Year After Long or Short Hospitalization

Item	Range of Measure*	Short-Term Patients	Long-Term Patients	Test of Significance	Significance
Global measures (adjusted means)					
HSRS	1–100**	35.6	40.1	Analysis of covariance	$p<.05$
PEF overall severity item	1–6	3.7	3.2	Analysis of covariance	$p<.01$
Summary score of historical data	−1.20–+2.82	+0.2	+0.1	Analysis of covariance	n.s.
Posthospitalization treatment measures					
Number of readmission episodes	—	45.0	33.0	Chi-square test	n.s.
Mean number of days per episode	—	25.4	31.8	Two-tailed t test	n.s.
Number of patients rehospitalized	—	26.0	19.0	—	—
Number of patients not rehospitalized	—	43.0	48.0	—	—
Analysis (patients rehospitalized versus those not)	—	—	—	Chi-square test	n.s.
Mean number of days in hospital	—	16.5	15.7	Two-tailed t test	n.s.
Mean number of psychotherapy visits	—	12.6	19.9	Two-tailed t test	$p<.01$
Number of patients taking phenothiazines	—	28.0	37.0	—	—
Number of patients not taking phenothiazines	—	37.0	26.0	—	—
Analysis (patients taking drug versus those not)	—	—	—	Chi-square test	n.s.
Mean dosage of chlorpromazine equivalent per day for all outpatients (mg)	—	141.0	378.0	Two-tailed t test	$p<.01$
Mean dosage of chlorpromazine equivalent per day for outpatients receiving medication (mg)	—	328.0	644.0	Two-tailed t test	$p<.01$
Symptom measures					
PEF disorganization scale (adjusted means)	1–6	1.6	1.6	Analysis of covariance	n.s.
PEF subjective distress scale (adjusted means)	1–6	2.1	2.1	Analysis of covariance	n.s.
PEF antisocial scale (adjusted means)	1–6	1.4	1.5	Analysis of covariance	n.s.
PEF withdrawal scale (adjusted means)	1–6	2.8	2.4	Analysis of covariance	$p<.10$
PEF grandiosity-externalization scale (adjusted means)	1–6	2.1	1.9	Analysis of covariance	n.s.
PSECS emotional health item (means)	1–5	2.6	2.3	Two-tailed t test	n.s.
FMECS emotional health item (means)	1–5	2.5	2.2	Two-tailed t test	n.s.
Family functioning measures (means)					
PEF mate role item***	1–6	3.6	3.9	Two-tailed t test	n.s.
PEF parent role item***	1–6	2.9	2.0	Two-tailed t test	n.s.
PSECS family item	1–5	2.4	2.1	Two-tailed t test	n.s.
FMECS family item	1–5	2.2	2.0	Two-tailed t test	n.s.
Leisure time and social activities measures					
KAS for patients socially expected activities scale (adjusted means)	10–48**	30.3	29.8	Analysis of covariance	n.s.
KAS for relatives socially expected activities scale (adjusted means)	10–48**	27.9	29.4	Analysis of covariance	n.s.
KAS for patients free time scale (adjusted means)	11–66**	34.8	35.3	Analysis of covariance	n.s.
KAS for relatives free time scale (adjusted means)	11–66**	34.8	36.9	Analysis of covariance	$p<.10$
PSECS social life item (means)	1–5	2.8	2.7	Two-tailed t test	n.s.
FMECS social life item (means)	1–5	2.6	2.5	Two-tailed t test	n.s.
Work measures (means)					
HIF role functioning item	1–4**	2.9	2.7	Two-tailed t test	n.s.
Number of months unemployed	0–8	6.0	5.5	Two-tailed t test	n.s.
PEF wage earner item***	1–6	2.1	2.6	Two-tailed t test	n.s.
PEF housekeeper item***	1–6	2.3	2.2	Two-tailed t test	n.s.
PEF student trainee item***	1–6	2.9	2.8	Two-tailed t test	n.s.
PSECS work item	1–5	2.7	2.6	Two-tailed t test	n.s.
FMECS work item	1–5	2.6	2.4	Two-tailed t test	n.s.

* Unless otherwise noted, a low score indicates better adjustment.
** A high score indicates better adjustment.
***There were missing data for a large number of patients on these items. The numbers of patients for whom data were available on the PEF mate role item were 15 short-term patients and 9 long-term patients. The respective numbers for the PEF parent role item were 16 and 8; for the PEF wage earner item, 15 and 16; for the PEF housekeeper item, 25 and 18; and for the PEF student trainee item, 14 and 12.

ses of these three variables employed a three-way analysis of covariance: treatment assignment (short versus long hospitalization) × sex × diagnosis (paranoid versus nonparanoid); the admission score on each variable was the covariate. Details of these three analyses are shown in table 2. Significant treatment assignment effects favoring the long-term patients were found on the HSRS and on the overall severity item of the PEF (see table 2). These two variables also showed a significant main effect for sex on these two variables, i.e., women were rated as showing greater improvement. Neither the diagnosis effect nor any of the interactions was significant for any of the three variables.

Inspection of these analyses show that the HSRS and PEF global scores had a restricted range at admission. The admission summary score of historical data, in contrast, showed a wider range because it applied to average functioning over a two-year preadmission period. We found that this score was the best predictor of all three global ratings at one year. We therefore repeated the analyses of covariance for the HSRS and the PEF overall severity item, using the admission summary score of historical data as the covariate.

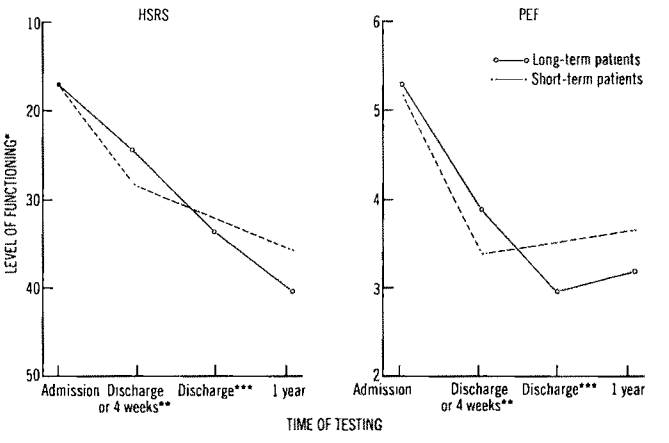
This provided a better control of prehospital differences in functioning and tended to weaken the main effect of treatment assignment. The HSRS significance level changed from $p<.05$ to $p<.06$, and the PEF significance level changed from $p<.01$ to $p<.05$. We further examined the effect of adding, as covariates, the three

TABLE 2
Analysis of Covariance of Three Global Measures for 138 Schizophrenic Patients*

Item	HSRS Score F	HSRS Score Significance	PEF Overall Severity Item Score F	PEF Overall Severity Item Score Significance	Summary Score of Historical Data F	Summary Score of Historical Data Significance
Treatment (short versus long hospitalization) (df=1, 129)	4.95	p<.05	8.28	p<.01	0.76	n.s.
Sex (df=1, 129)	6.96	p<.01	4.46	p<.05	1.76	n.s.
Diagnosis (paranoid versus nonparanoid) (df=1, 129)	0.99	n.s.	2.73	n.s.	0.01	n.s.
Treatment × diagnosis (df=1, 129)	0.75	n.s.	2.92	n.s.	0.02	n.s.
Treatment × sex (df=1, 129)	0.04	n.s.	0.46	n.s.	0.27	n.s.
Diagnosis × sex (df=1, 129)	0.56	n.s.	0.06	n.s.	0.01	n.s.
Treatment × sex × diagnosis (df=1, 129)	0.21	n.s.	0.75	n.s.	1.41	n.s.
Covariate (df=1, 129)	0.62	n.s.	7.57	p<.01	58.97	p<.01
Equality of covariate regression slope (df=7, 122)	0.26	n.s.	1.24	n.s.	1.91	n.s.

* The baseline value of each measure is the covariate.

FIGURE 1
Mean Scores of Short-Term and Long-Term Schizophrenic Patients on the HSRS and the PEF Overall Severity of Illness Item



*A score of 50 on the HSRS indicates a better level of functioning; a score of 10 indicates a worse level. A score of 2 on the PEF overall severity item indicates a better level of functioning; a score of 6 indicates a worse level.
**The second time of testing for short-term patients took place at discharge; for the long-term patients it took place 4 weeks after admission.
***Long-term patients only.

variables that significantly differentiated the short-term and long-term groups (premorbid adjustment, education, and socioeconomic status). Addition of these covariates tended to further reduce the treatment assignment effect, although it remained at $p<.05$ for the overall severity item of the PEF. Thus, although it appears that there is indeed an effect of treatment assignment favoring long-term hospitalization, this effect also seems to have been spuriously enhanced in our data by chance variation in the random assignment of the patients.

These effects of short and long hospitalization, as measured by the HSRS and PEF global ratings, are shown graphically in figure 1. As previously reported (2), in a similar analysis of covariance short-term patients were found to be more improved on the HSRS just before their discharge than were long-term patients at a comparable time after admission (4 weeks) ($p<.01$). At one year this effect was reversed, however: the long-term group showed significantly greater improvement (see table 1).

We next looked at the tails of the sample to find out which patients were most improved and which were least improved or had gotten worse. At one year, 15 of the 17 patients who improved most on the overall severity item of the PEF were long-term, and 14 of the 26 patients who improved least were short-term ($p<.02$, chi-square test). There was a similar trend on the HSRS, but it did not reach significance.

In summary, at one year there was a global effect favoring the long-term patients.

Posthospital Treatment

In interpreting these results, one must consider the possible intervening effect of the aftercare that many

of these patients received. Since we did not control aftercare experimentally, it was possible for the length of hospital stay to influence the amount of aftercare, which in turn could account for some or all of the differences in functioning seen one year after admission. The long-term patients did, in fact, receive a different pattern of treatment after hospitalization than did the short-term patients, as is shown in table 1. During the final 8 months of the first year after admission, when virtually all of the patients had been discharged from the index hospitalization, long-term patients made significantly more psychotherapy visits. At the one-year follow-up they were receiving medication at significantly higher dose levels (see table 1).

To check whether the number of posthospitalization psychotherapy visits might also be related to sex or baseline summary score of historical data, we computed two further analyses with the number of psychotherapy visits as the dependent variable: a three-way analysis of variance—treatment assignment × sex × admission summary score of historical data (high versus low) and a two-way analysis of covariance (treatment assignment × sex, with the admission summary score of historical data as covariate). Neither sex nor admission summary score of historical data was related to number of posthospital psychotherapy visits, either directly or by interaction with the treatment assignment. In both analyses the treatment assignment (short versus long) main effect was significant ($p<.05$ and $p<.01$, respectively). Similar analyses were computed using the one-year data on chlorpromazine-equivalent medication dosage at the time of follow-up as the dependent variable. Again, the main effect of treatment assignment was significant, but neither sex nor admission summary score of historical data showed main effects or interaction with treatment assignment.

With such complex confounding of the length of hospital treatment with the amount of posthospitalization treatment, it is not possible to be sure how much of the better performance of the long-term patients at one year was due to more aftercare. Nevertheless, we attempted to explore this issue further by including the amount of aftercare as if it were an independent variable (which it is not) in further analyses of global outcomes at one year. The patients were divided into three approximately equal groups according to whether they had a low (0–3), moderate (4–20), or high (21 or more) number of outpatient psychotherapy contacts. We computed analyses of covariance (treatment × sex × amount of psychotherapy) of the three global measures. The linear component of posthospital psychotherapy accounted for a significant ($p<.05$) amount of variance of two of the measures, the HSRS and the summary score of historical data. These are the two most closely related to social functioning, but the third, the PEF overall severity item, focuses more on symptoms. The short versus long treatment effect on the HSRS was entirely eliminated (or "accounted for") by including the aftercare variable. The treatment effect seen in the PEF was not eliminated, but it was somewhat weakened by adding the aftercare variable; it was also complicated by the appearance of two- and three-way interactions of aftercare therapy with short versus long treatment assignment.

Thus, in general, the aftercare variable does seem to relate to one-year outcomes and at least partially accounts for the independent effects of the short or long treatment assignment. In similar analyses of drug dosage at one year, no comparable attenuation of the treatment assignment effect was found. This suggests that the influence of length of stay on subsequent posthospitalization psychotherapy is an important mechanism—perhaps the primary mechanism through which length of stay influences outcomes at one year.

Other Outcome Measures

Analyses were also carried out on the 19 specific symptom ratings of the PEF and a variety of other specific ratings by the follow-up interviewers, by the patients, and by family members. Two of the 19 PEF items, daily routine and belligerence-negativism, showed significant differences ($p<.05$, t test) favoring long-term patients. On the emotional health item of the PSECS, more long-term patients judged their emotional health "much better" or a "a little better" than it was two years before admission, more short-term patients judged it to be the same, and equal numbers of short- and long-term patients judged their emotional health worse ($\chi^2=7.27$, $p<.05$). The remaining measures are summarized in table 1. Although the trends generally favor long-term patients, most do not reach significance.

COMMENT

This study attempts to answer the question of what are the relative benefits of short-term versus long-term inpatient treatment for those schizophrenic patients for whom inpatient treatment is indicated.

To the clinician, the pertinent question is which type of treatment (long-term or short-term) is more likely to result in improvement at one year in a patient who has to be hospitalized. Our data show a modest but statistically reliable difference in global outcome favoring the long-term group. There were no consistent differences between groups on symptoms, work, socialization, or family relationships, but when there were differences, they generally favored long-term treatment.

Our data are consistent with those of Caffey and associates (14), who conducted an experiment similar to ours, in that there was no significantly greater incidence of readmission or better community adjustment (as measured by the KAS) for either group, but our data do show differences (favoring long-term patients) in areas that Caffey and associates did not assess. Our data favoring long-term treatment also need to be considered in the context of two nonexperimental studies comparing community mental

health center inpatient treatment (short-term) versus traditional state hospital treatment (long-term) that favored shorter treatment (4, 15), although our study cannot be directly compared with these studies. Finally, it should be kept in mind that the differences found in favor of long-term hospitalization in the present study, although statistically significant, are of modest size.

The suggestion in our data that the effects of length of hospitalization on outcome are mediated by the amount of psychotherapy the patients received after hospitalization seems important. We speculate that the longer hospitalization allowed for acceptance of the need for therapy on the part of the patient and those important to him or her as well as the need for better arrangements to be made for such treatment. These are advantages sometimes suggested by those who favor long-term over short-term inpatient treatment. Our data do not indicate whether this could be done during a shorter hospitalization or through concerted intervention during the first weeks after hospital discharge. This is a question that must remain for a later study.

In our first paper (1), we began to look at the cost-benefit issue. It is obvious that in this first follow-up year the greater initial cost of the long-term hospital treatment is not offset by a lower cost after hospitalization. Given the differences in outcome, which cannot easily be converted to monetary value, is it worth the greater cost to keep patients in the hospital longer? If society bases the cost-benefit comparison solely on monetary outcomes, then short-term treatment will clearly be the treatment of choice. On the other hand, if society places a high value on the individual patient's adjustment, then long-term treatment might still be considered a viable alternative. A more comprehensive picture of the cost effectiveness of short and long hospitalization will be available when we have the follow-up results for the second year.

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Short Versus Long Hospitalization: A Prospective Controlled Study. V. One-Year Follow-Up Results for Nonschizophrenic Patients

BY IRA D. GLICK, M.D., WILLIAM A. HARGREAVES, PH.D., JOAN DRUES, M.A., AND JONATHAN A. SHOWSTACK

The authors studied the effect of long-term versus short-term hospitalization on a group of 74 patients with the diagnoses of affective disorder, neurosis and personality disorder, and hysterical personality one year after their admission to the hospital. Although they had found in an earlier study that short-term patients seemed to integrate more rapidly in the hospital, the results reported in this study showed no statistically reliable differences between the long-term and short-term groups. In contrast to the author's results for schizophrenic patients, their findings for nonschizophrenic patients do not support extended hospitalization.

WE HAVE PREVIOUSLY REPORTED our results for the inpatient phase of this study (1, 2). One-year follow-up results for the schizophrenic patients are reported in the preceding paper. In this fifth communication, we report results of the one-year follow-up of 74 nonschizophrenic patients.

METHOD

Treatment assignment, treatment setting, evaluation, and follow-up procedures for our nonschizophrenic patients were the same as those for our schizophrenic patients. The treatment program for patients with various diagnoses in the study group has been described (2). There were no significant differences between short-term and long-term nonschizophrenic patients in the variables of sex, marital status, race, age, years of education, and socioeconomic status (2). As previously described, the 74 nonschizophrenic patients were grouped for analysis into three diagnostic subgroups—affective disorder, neurosis and personality disorder, and hysterical personality disorder. We were able to locate and enlist the cooperation of 33 short-term and 34 long-term subjects (91% of the original sample of 74 subjects).

The authors are all with the Cinical Research Ward, Langley Porter Institute, University of California, San Francisco, Calif. 94143. Dr. Glick is Associate Professor of Psychiatry in Residence, Dr. Hargreaves is Associate Professor of Medical Psychology in Residence, Ms. Drues is Staff Research Associate, and Mr. Showstack is Staff Research Associate II.

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RESULTS

Global outcome at one year was measured by the HSRS, the PEF overall severity of illness item, and the summary score of historical data, described in the preceding paper. Two analyses of covariance were computed for each of these three measures: treatment assignment × sex and treatment assignment × diagnosis. The admission score on each measure was the covariate. We chose not to compute a single three-way analysis of covariance because cell frequencies were too small when sex and diagnosis were cross-classified.

The PEF overall severity item showed a significant main effect for treatment assignment favoring the long-term group in the first analysis ($p < .05$) and a similar trend ($p < .10$) in the second analysis. Neither sex nor diagnosis produced significant main effects in their separate analyses, nor were there significant interactions of treatment assignment × sex or treatment assignment × diagnosis. Parallel analyses of the HSRS and the summary score of historical data produced no significant main effects or interactions.

In other words, at one year there was no differential response to long-term or short-term treatment for all three nonschizophrenic groups except for a slight but statistically significant effect favoring long-term treatment on the PEF overall severity item. There were no differences either between men and women or among the three diagnostic subgroups (affective disorder, neurosis and personality disorder, and hysterical personality disorder) on any of the three global measures.

We had previously found that the admission summary score of historical data of schizophrenic patients was highly correlated to the one-year outcome scores; this was also true for the nonschizophrenic patients.

We therefore repeated the above analyses, using the admission summary score of historical data as the covariate. The results of these analyses are essentially the same as reported above.

Analyses parallel to those reported in the preceding paper were done for symptoms, family functioning, posthospital treatment, use of leisure time, socialization, and role functioning. There were essentially no differences between short- and long-term patients in these areas of functioning. The study results are summarized in table 1.

COMMENT

The results for nonschizophrenic patients suggest that at one year long-term treatment may be slightly more effective than short-term treatment, but the findings are much more equivocal than they were for the schizophrenic patients. There are no similar studies reported that support greater benefit for either type of treatment for nonschizophrenic patients. On the whole, our results do not provide strong support for the use of the more expensive longer hospitalization

TABLE 1
Summary of Outcome Measures for Nonschizophrenic Patients One Year After Short or Long Hospitalization

Item	Range of Measure*	Short-Term Patients	Long-Term Patients	Test of Significance	Significance
Global measures (adjusted means)					
HSRS	1–100*	48.3	53.1	Analysis of covariance	n.s.
PEF overall severity item	1–6	3.3	2.6	Analysis of covariance	$p < .10$
Summary score of historical data	−1.20–+2.82	−0.26	−0.37	Analysis of covariance	n.s.
Posthospitalization treatment measures					
Number of readmission episodes	—	7.0	13.0	Chi-square test	n.s.
Mean number of days per episode	—	19.3	34.8	Two-tailed t test	n.s.
Number of patients rehospitalized	—	5.0	8.0	—	—
Number of patients not rehospitalized	—	26.0	26.0	—	—
Analysis (patients rehospitalized versus those not)	—	—	—	Chi-square test	n.s.
Mean number of days in hospital	—	4.4	13.3	Two-tailed t test	n.s.
Mean number of psychotherapy visits	—	12.0	17.1	Two-tailed t test	n.s.
Symptom measures					
PEF disorganization scale (adjusted means)	1–6	1.4	1.3	Analysis of covariance	n.s.
PEF subjective distress scale (adjusted means)	1–6	2.5	2.2	Analysis of covariance	n.s.
PEF antisocial scale (adjusted means)	1–6	1.4	1.4	Analysis of covariance	n.s.
PEF withdrawal scale (adjusted means)	1–6	2.2	1.7	Analysis of covariance	n.s.
PEF grandiosity-externalization scale (adjusted means)	1–6	1.6	1.4	Analysis of covariance	n.s.
PSECS emotional health item (means)	1–5	2.3	1.8	Two-tailed t test	n.s.
FMECS emotional health item (means)	1–5	2.1	1.5	Two-tailed t test	n.s.
Family functioning measures (means)					
PEF mate role item***	1–6	2.9	2.9	Two-tailed t test	n.s.
PEF parent role item***	1–6	3.8	1.8	Two-tailed t test	$p < .02$
PSECS family item	1–5	2.5	2.2	Two-tailed t test	n.s.
FMECS family item	1–5	2.2	1.8	Two-tailed t test	n.s.
Leisure time and social activities measures					
KAS for patients socially expected activities scale (adjusted means)	9–48**	33.4	34.0	Analysis of covariance	n.s.
KAS for relatives socially expected activities scale (adjusted means)	9–48**	31.5	32.6	Analysis of covariance	n.s.
KAS for patients free time scale (adjusted means)	10–65**	37.7	39.0	Analysis of covariance	n.s.
KAS for relatives free time scale (adjusted means)	10–65**	40.5	38.8	Analysis of covariance	n.s.
PSECS social life item (means)	1–5	2.3	2.0	Two-tailed t test	n.s.
FMECS social life item (means)	1–5	2.3	1.7	Two-tailed t test	n.s.
Work measures (means)					
HIF role functioning item	1–4**	2.2	2.2	Two-tailed t test	n.s.
Number of months unemployed	0–8	4.1	3.6	Two-tailed t test	n.s.
PEF wage earner item***	1–6	2.5	1.9	Two-tailed t test	n.s.
PEF housekeeper item***	1–6	1.8	1.4	Two-tailed t test	n.s.
PEF student trainee item***	1–6	1.6	1.0	Two-tailed t test	n.s.
PSECS work item	1–5	2.3	2.3	Two-tailed t test	n.s.
FMECS work item	1–5	2.1	1.6	Two-tailed t test	n.s.

* Unless otherwise noted, a low score indicates better adjustment.
** A high score indicates better adjustment.
***There were missing data for a large number of patients on these items. The numbers of patients for whom data were available on the PEF mate role item were 7 short-term patients and 13 long-term patients. The respective numbers for the PEF parent role item were 5 and 10; for the PEF wage earner item, 15 and 16; for the PEF housekeeper item, 18 and 17; and for the PEF student trainee item, 7 and 5.

for nonschizophrenic patients. It is possible, of course, that the sample was too small, the measuring instruments used were insensitive to the relevant outcomes for this group, or greater differences between the two treatment groups will emerge in the two-year follow-up results.

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Journal Policy on Multiple Authorship

It has come to our attention that there has been some misunderstanding regarding the *Journal*'s policy on polyauthorship. The number of authors listed on a paper is not a factor in the review and acceptance/rejection procedure. However, once a decision has been made to publish a paper, we do apply *Journal* policy regarding the number of authors in the by-line: only principal researchers and writers of articles are listed in the by-line; collaborators who have provided technical and other assistance are acknowledged in a footnote. In papers that list more than 4 people as principal authors, we query the first author to verify that those individuals were substantial contributors to the research or ideas reported. Since certain types of psychiatric research may require multiple workers, we will of course include all of the names in these cases.

Limitations of Brief Hospital Treatment

BY STEPHEN REIBEL, M.D., AND MARVIN I. HERZ, M.D.

A total of 175 newly admitted inpatients who lived with their families were randomly assigned to three treatment groups: standard inpatient care and brief hospitalization with and without transitional day care. Case reports of 6 of the 9 patients considered "study failures" illustrate that effective postdischarge adaptation is limited by the patients' degree of impairment as well as the family and community capacity to accept them. Although rapid return to the community is beneficial to many patients, rigid adherence to this policy is neither wise nor clinically effective.

IN RECENT YEARS there has been a distinct trend toward brief rather than long-term hospitalization for the mentally ill and an attempt to provide psychiatric care within the patient's community rather than in large, geographically remote state hospitals. The deleterious effects of prolonged custodial care in large government institutions have been described by Goffman (1), Gruenberg (2), and others. Not only state hospitals but also psychiatric units of general hospitals and community mental health centers have attempted to shorten inpatient stays. While it appears that for most patients brief hospitalization is preferable to prolonged hospitalization (3–7), there are some patients who need long-term inpatient care. This paper describes a group of patients who were found to be unsuitable for brief hospital care and who required more lengthy inpatient treatment.

In 1971 a controlled study was begun on the Washington Heights Community Service of the New York State Psychiatric Institute to evaluate the relative efficacy of brief hospitalization (1 week or less) and discharge at the therapist's discretion (standard hospitalization) (3). Because we were interested in the effects on families as well as patients, only patients who lived with families were included in the study. The study design provided that no patient should be discharged from inpatient care if it were clearly detrimental to his own or his family's welfare.

In spite of intensive efforts to adhere to the study design, 9 out of 112 patients were considered "study failures" in that they could not be treated by brief hospitalization. They are to be distinguished from other patients assigned to brief treatment who did relatively poorly and required frequent rehospitalization but for whom there was no compelling reason to discontinue the "brief" paradigm. In the case of the 9 study failures, it was judged that any further attempt to limit the duration of hospitalization would be clearly detrimental to the welfare of the patient or his family. The purpose of this paper is to describe 6 of these 9 patients and their social situations. We feel that they document the limitations of brief hospital treatment and the continued need for lengthier hospital stays for a small but significant proportion of patients.

METHOD

Subjects

The study was conducted on the Washington Heights Community Service, which is responsible for a catchment area with a population of 90,000 in an ethnically diverse low-income area of upper Manhattan.

All patients admitted to the inpatient service were evaluated for possible inclusion in the study on the first weekday following their admission. The attending psychiatrist and the patient's therapist (a psychiatric resident or psychology intern) conducted the evaluation. A total of 175 patients entered the study. It is important to note that all patients accepted in the study had to have a responsible adult willing to provide some supervision at the time of discharge.

Treatment

Immediately after they were evaluated for inclusion in the study, patients who met the criteria were randomly assigned to one of the following treatment groups.

1. *Standard-out group* (N=63). Standard hospital procedure was followed, i.e., the length of hospital-

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The authors are with the Washington Heights Community Service, New York State Psychiatric Institute, 722 West 168th St., New York, N.Y. 10032, where Dr. Reibel is Director, Outpatient Service, and Dr. Herz is Director.

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ization was determined by the patient's therapist and the attending psychiatrist in consultation with the rest of the clinical staff. When inpatient care was no longer deemed necessary, the patient was discharged into the community and followed, if appropriate, as an outpatient.

2. *Brief-day group* (N=61). Every attempt was made to discharge patients from inpatient care to aftercare within 1 week. Patients and their families were told that in all probability the patient would be discharged from inpatient care within 1 week but would receive appropriate aftercare. When the patients were discharged from inpatient care, they could receive day care unless it was considered unnecessary. Patients who received day care came to the hospital each weekday at 8:30 a.m. and left to go home at 4:30 p.m. There was no separate program for day patients. While at the hospital, they participated in the same program with the same staff as the inpatients. Discharge from day care was at the therapist's discretion, with outpatient treatment provided when appropriate. Acceptable reasons for prolonging the inpatient hospitalization included suicide risk, possibility of violent behavior, and extreme disorganization. Whenever a patient was not discharged within 1 week, the reasons were recorded on a form.

3. *Brief-out group* (N=51). Inpatient treatment was identical to that of the brief-day group. Day care was not offered, i.e., brief hospitalization was followed by full discharge into the community with outpatient therapy when appropriate.

Once a patient had been assigned to a treatment group, he was reassigned to the same group on subsequent readmissions during the entire follow-up period of the 2 years after initial admission. All patients were treated on the same 55-bed inpatient ward, which has an open-door policy except for a closed 10-bed intensive care unit. Patients in the 3 groups were treated by the same staff and participated in the same activities during the day. The ward program was group oriented, with a strong emphasis on activation of patients. A full range of psychiatric treatments was used, including individual psychotherapy, group, family, milieu, and somatic therapy, and vocational rehabilitation. The inpatients generally spent 24 hours a day in the hospital. Except for weekend or overnight passes, all inpatients slept at the hospital. Day patients began their treatment by spending the full 8 hours during the day at the hospital from Monday through Friday. Later participation would be reduced if deemed appropriate.

Weekly meetings of the research and clinical staffs were held to review the status of each study patient. The determination that brief hospitalization was proving to be inappropriate or harmful to a patient or his family was made at these meetings. Once this determination was made, no time limit was placed on the patient's hospital stay and he was considered a "brief hospitalization failure." Reasons for this decision were recorded on a special form.

RESULTS

The 9 patients described in this paper who were study failures were compared to the total sample by means of the Social Background Record (SBR) (8) and the Psychiatric Status Schedule (PSS) (9). The SBR revealed that the "failure" subgroup was significantly more likely to be younger, single, and schizophrenic (paranoid or chronic) than the rest of the sample and more likely to have had more previous hospital admissions ($p<.05$, t test). The only differences noted on the initial PSS scores were significantly more overt anger in the "failure" subgroup ($p<.05$) and a tendency toward more impairment in their roles as parents and housekeepers.

By examining these cases carefully we hoped to define patients and situations that do not lend themselves to brief hospital treatment as well as to provide some basis for prescribing appropriate treatment on initial evaluation. A description of 6 patients whose psychopathology and life situations did not permit brief hospital treatment follows.

CASE REPORTS

The first 2 patients to be discussed were assigned to the brief-day group. The failure of this form of treatment is of particular significance since a short period of in-hospital treatment followed by partial hospitalization is being increasingly used as an alternative to long-term hospitalization.

Case 1. A 23-year-old single Ecuadorean man with a diagnosis of schizophrenia was brought to the hospital by his brother because of extreme social isolation, psychological withdrawal, and self-neglect. The brother professed his willingness to have the patient return to live with him following discharge. The patient was rehospitalized 5 times within 1 year; it was during the last hospitalization, which lasted 65 days, that the patient was considered a "brief hospitalization failure."

The staff's effort at short-term hospitalization was based on the assumption that the patient could return to his brother's home. Eventually the staff learned that the brother planned to marry and no longer wished to care for the patient. The treatment plan actually intensified the conflict between the patient and his brother. After the brother admitted that he could no longer care for the patient, a suitable plan involving residential placement was formulated. As a result both the patient's mental status and his relationship with his brother improved.

Case 2. A 21-year-old single, mildly retarded Puerto Rican woman was admitted to the hospital and placed in the brief-day group after a 3-day period of increasing confusion and agitation. She seemed to have a relatively stable home situation. Her behavior in the hospital was wildly bizarre and provocative as efforts were made to introduce her to the day hospital regimen.

In this case, the pressure for rapid return home with day hospitalization was inappropriate. The degree of the patient's psychopathology and disorganization precluded

her understanding and participating in a program that required that she adjust to living both at home and in the hospital. It was evident that her panic and disorganization required a longer period of hospitalization, and she was judged to be a "brief hospitalization failure" 5 months after admission. After several months of hospitalization in a distant state hospital she was able to return home, obtained a job, and began a promising relationship with a young man.

The following patients were randomly assigned to the brief-out group. This treatment placed more stress on the staff for rapid intervention and higher expectations on the patients and their families since day hospitalization was not available.

Case 3. A 17-year-old single girl, a high school senior, was admitted to the hospital following a suicidal gesture involving an overdose of barbiturates. The precipitating event seemed related to the impending dissolution of her parents' 25-year marriage. There had been severe and open discord for years. Attempts to discharge the patient into this deteriorating situation involved her repeatedly in pathological family interactions, and she required 4 readmissions in 5 months. She was transferred to and did well on a long-term inpatient service.

Case 4. A 20-year-old divorced mother of 2 was admitted to the study at the time of her fourth hospitalization in little over a year. At the time of admission she had stopped taking her medication and was hallucinating accusatory voices.

The patient was unable to live either with her abusive alcoholic mother or independently with her children. Her conflicts around this choice led to repeated suicide attempts and eventually necessitated involuntary commitment. When she was encouraged to live by herself in the sheltered setting of a halfway house, her suicidal behavior ceased.

Case 5. A 22-year-old single, unemployed man of massive proportions was admitted to the hospital in a confused and agitated state. This was his first hospitalization. The patient, who was black, had a long history of antisocial behavior and vague and grandiose delusions concerning the Black Muslims.

The patient was discharged within 1 week in accordance with the protocol but within 2 days was arrested for purse snatching. He was sent to prison to await trial. However, due to the hospital's intervention he was placed on probation and transferred to our custody. Because of these legal entanglements he was dropped from the study. Various efforts were made to engage him in rehabilitation through vocational counseling, training programs, etc., but he was unable to follow through on any of these. After 6 months of such efforts, the patient was discharged but was quickly readmitted because of another incident of purse snatching. His management in the hospital was difficult because of continued petty thievery and occasional violence. Efforts to place the patient in a residence for the mentally retarded were unsuccessful, and he was discharged to his family. Three months after his final discharge the patient was arrested for stealing a neighbor's television set and was sent to a correctional institution.

A combination of this patient's antisocial traits, limited intelligence, poor judgment, impulsivity, and episodic psychotic disorganization make life for him in the community a doubtful proposition. Such patients account for many of the sensational news media stories concerning discharged psychiatric patients. There are currently no institutions specifically designed to deal with them. Any effective treatment program would have to include legal sanctions. At the moment these patients are lost between the boundaries of criminal justice and psychiatric care systems. This neglect benefits neither them nor their potential victims.

Case 6. A 37-year-old separated mother of 5 from Santo Domingo was admitted to the hospital for the first time following an overdose of chlordiazepoxide (Librium). Her symptoms of insomnia, anorexia, and anhedonia were clearly related to the breakup of her marriage of 20 years and her continuing struggle with her husband over financial support. As time for discharge approached, the patient made dramatic assertions of suicidal intent that were sufficiently frightening to the staff for her to be placed in the intensive care unit.

This nonpsychotic woman was hospitalized because of provocative and largely manipulative suicidal behavior. She manifested this behavior in order to express her anger toward her husband and to avoid maternal responsibilities that she experienced as overwhelming. Pressure for early discharge led to a power struggle. When the staff adopted a policy of not setting a definite discharge date, the patient ceased her regressive behavior within a few weeks and requested discharge.

DISCUSSION

In a study of brief hospitalization conducted on the Washington Heights Community Service, all but 9 of 112 patients were treated according to the protocol. Standardized test scores showed that the subsample had more overt anger and more previous hospitalizations than the rest of the study sample. However, the differences were not great. Family, social, and individual factors were all relevant to some extent. The comments concerning each case represent our conceptualization of the relevant problems.

Persistent poor judgment and impulsive behavior, with or without mental deficiency, antisocial behavior patterns, and continued psychotic disorganization clearly limit effective postdischarge adaptation as well as the family's and community's capacity to accept and tolerate discharged patients. Patients who are suicidal or assaultive cannot be returned rapidly to the community until these symptoms have abated.

Family-related factors were of great importance in many of the cases. In some, the intensity of family crisis was so severe that rapid return home was clearly detrimental. This was particularly true for adolescents whose families were in severe crisis. In other families, specific patient-family interactions perpetuated and exacerbated the patient's symptoms.

Schizophrenic patients who decompensate after marrying and having children can present special problems. They may gravitate back to their parents and be encouraged to regress into dependent or symbiotic relationships. Brief hospitalization encourages the staff to return patients to their families, but in some instances a more prolonged hospitalization, with the goal of separating the patient from his family, may be more appropriate.

In the past, removing patients from society was felt to be best for the patients, their families, and their communities. More recently, however, mental health ideology has come full circle, advocating rapid return to the community. Our intent is to call attention to the limitations of a rigid adherence to this policy. The fact that all patients in our study had families is of crucial importance. As a result they were in a far better position to benefit from brief treatment than are most patients who are social isolates, such as those described by Reich and Siegel (10). These authors pointed out that the discharge of the chronically dysfunctional, isolated psychotic individual to a foster home he rarely leaves, a "welfare hotel" where he may become the victim of various depredations, or to a wandering skid-row existence hardly represents social concern or the exercise of meaningful personal freedom. At least our patients did have homes to which to return. Nevertheless, brief treatment did not appear effective in these 9 cases.

We believe that rapid return to the community enables most patients to use their assets and discourages regression. Families are unable to use the hospital as a repository for their problems. Nevertheless, a rigid adherence to the policy of brief hospitalization for all is neither wise nor clinically effective. Only a careful assessment of each case can determine the appropriateness of hospital discharge. It is necessary to recognize that hospitalization is only one component of a comprehensive health care delivery system that should include halfway houses, supervised residences, sheltered workshops, and aggressively carried out aftercare. Without the availability of such comprehensive community care, there is a distinct danger that enthusiasm will give way to cynicism and a return to custodial institutions.

Finally, there is a small group of chronically ill patients, characterized by gross psychotic disorganization, markedly impaired reality testing, and extremely poor judgment, for whom the most humane environment may be the state hospital with the protection and support that it affords. Certainly mental health care delivery systems must include some provision for patients who require more prolonged institutionalization.

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Psychosis, Thought Disorder, and Regression

BY NANCY C. ANDREASEN, M.D., AND PAULINE S. POWERS, M.D.

Although thought disorder is widely considered to be pathognomonic of schizophrenia, the role of thought disorder and regression in psychosis has not been systematically evaluated with psychometric techniques. The authors used a sorting behavior test to compare five cognitive processes in schizophrenic patients, manic patients, and normal children and adults. They hypothesized that the two psychotic groups would have scores similar to those of the children, which would reflect the tendency of psychotic individuals to display regressive thinking and autism. It was found that the cognitive styles of the children and the manic patients were strikingly different, but that those of the children and the schizophrenic patients were similar.

AT LEAST since the time of Bleuler, the concept of thought disorder has been central to the definition of schizophrenia. Bleuler tried to determine the fundamental disturbances that underlie the illness, rather than examining superficial clinical symptoms, and he thought that the cognitive disturbance was perhaps the most basic (1). In an era of "association psychology," Bleuler emphasized that schizophrenic patients suffered from loosening of associative threads, or an inability to see connections between things:

> Of the thousands of associative threads which guide our thinking, this disease seems to interrupt, quite haphazardly, sometimes such single threads, sometimes a whole group, and sometimes even large segments of them. In this way, thinking becomes illogical and often bizarre. (1, p.14)

Bleuler's influence on the concept of schizophrenia has been significant, and many psychiatrists follow his precedent and consider thought disorder pathognomonic of schizophrenia.

Later theorists, particularly those who use psychoanalytic concepts, have tended to use the idea of regression to earlier stages of adaptation to explain schizophrenic thought disorder. According to these investigators, schizophrenic thinking shows the same symbolism and lack of ordinary logic that one observes in dreams or in the thought and speech of children (2–8). Explanations of this similarity vary, depending on the point of view of the theorist. Some stress the importance of withdrawal in the face of overwhelming anxiety, while others hold that the breakdown of the governing power of the ego and superego may reveal uncensored primary process thinking. Whatever their particular emphasis, many theorists have stressed the regressive and primitive qualities of schizophrenic thinking.

Some recent discussions of thought disorder have suggested that reevaluation of these basic concepts and theories is needed and have seriously questioned the notion that thought disorder is pathognomonic of schizophrenia. Reed (8) argued that schizophrenic thought disorder differs from normal thinking in quantity rather than in quality. In one investigation, a group of clinicians was unable to distinguish speech and writing samples taken from schizophrenic patients, manic patients, and well-known creative writers (9). Other investigations have suggested that thought disorder occurs in manic as well as schizophrenic patients and that they may show even more thought disorder than schizophrenics (10, 11). Now that the biological model is being applied more widely to schizophrenia, the importance of psychological concepts like regression is being questioned by some. Although widely discussed on a theoretical level, the role of regression in producing schizophrenic thought disorder has not been subjected to quantitative scrutiny.

METHOD

This investigation examined, using quantitative psychometric methods, thought disorder and regression in psychotic individuals. We compared cognitive processes in four groups: schizophrenic patients, manic patients, normal children (6 to 10 years old), and normal adults. We assumed that the scores of the children could be used to measure regression and that the adults would act as a control group whose scores would represent a baseline measurement.

Cognitive Processes

The psychometric measure used was the Goldstein-Scheerer Object-Sorting Test (12). In this test, sub-

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When this work was done, the authors were with the University of Iowa College of Medicine, 500 Newton Rd., Iowa City, Iowa 52242, where Dr. Andreasen is currently Assistant Professor of Psychiatry and Dr. Powers was a resident in psychiatry. Dr. Powers is now Assistant Professor, Department of Psychiatry, University of Southern Florida, Tampa, Fla.

jects are asked to sort a set of 29 objects (e.g., nails, a hammer, a red rubber ball, a red ashtray) into "groups that belong together." The "handing over" method of administering the test was used, with a total of eight starting points, which were all predetermined by the examiner. After each sorting, the subject was asked to explain why he had sorted the objects in that manner and his answer was recorded. The test results were then coded to conceal name and diagnosis and were randomly mixed. Both investigators scored them independently on a blind basis for behavioral overinclusion, conceptual overinclusion, idiosyncratic or autistic thinking, richness, and underinclusiveness, using the criteria described by Harrow and associates (13–15)[1]. Ratings were usually quite close, but after rating independently both investigators discussed their ratings and reached a consensus score for each subject.

The cognitive qualities were measured according to the following criteria:

1. Behavioral overinclusion. Total number of objects sorted in response to eight starting points.

2. Conceptual overinclusion. Scored on a 1 to 5 scale, with 1 representing normal logical sortings and 5 maximum overinclusiveness. Sorting behavior leading to high ratings included using vague or unrelated concepts to arrange groups, arbitrarily changing starting points, or "force fitting" objects into a category in which they did not belong.

3. Autistic thinking. Scored on a 1 to 5 scale, with 1 representing no autistic behavior and 5 extreme autistic behavior. High scores were assigned for using the sorting starting point in reference to personal experiences, using it as a cue understandable only to the subject, or engaging in inappropriate or strange behavior while sorting.

4. Richness. Scored on a 1 to 5 scale, with 1 representing no richness and 5 maximum richness. High scores for richness were assigned when subjects saw dimensions in the starting point that were appropriate but were rarely seen by others. For example, symbolic sortings (recognizing a group of oblong objects as phallic symbols), seeing each starting point in a fresh or different way from other starting points so that many concepts were used in the course of testing, or consistently making clear logical sortings resulted in high scores for richness.

5. Underinclusiveness. Scored on a 1 to 5 scale, with 1 representing no underinclusiveness and 5 maximum underinclusiveness. Behavior leading to high ratings on underinclusiveness included inability to sort in response to a starting point, incomplete sortings, and repeatedly using the same categorizing principles.

Sample

The sample tested consisted of 18 schizophrenic patients, 16 manic patients, 30 normal children, aged 6 to 10, and 21 normal adults. Patients were screened prior to testing by one of the investigators to determine if they met the diagnostic criteria for inclusion in the schizophrenic or manic groups.

All patients included in the manic group were required to meet the following criteria: 1) presence of at least four of eight symptoms of mood disorder (racing thoughts, grandiosity, pressured speech, increased sociability, early awakening, increased sex drive, irritability, financial extravagance) for periods lasting more than 2 weeks; 2) presence of at least five depressive symptoms (e.g., dysphoric mood, decreased appetite, weight loss, decreased sex drive, insomnia, disturbed diurnal rhythm, suicidal thoughts, crying spells, guilt feelings, decreased concentration), including at least two physiological symptoms, for periods lasting more than 3 weeks; 3) full remission between episodes of mood disturbance.

Patients included in the schizophrenia group were required to exhibit: 1) absence of mood disturbance (depression or elation); 2) symptoms lasting more than 2 months; 3) at least one of three cognitive symptoms (Schneiderian first-rank symptoms, delusions, or auditory hallucinations) in the absence of recent drug intake or excessive use of alcohol; 4) at least one of three behavioral symptoms (social withdrawal, poor work history, or poor heterosexual adjustment).

All patients tested had been admitted because of acute onset or exacerbation of symptoms; they did not differ significantly in age, sex, or educational background. Both manic and schizophrenic patients had a mean of 12.5 years of schooling. The mean age of the manic patients was 32; that of the schizophrenic patients was 29. Patients were usually tested within 1 week of admission. Nearly all were receiving medication at the time of testing, usually phenothiazines or lithium or both, but none was close to clinical remission.

Fifteen first-graders (ages 6 to 7) and 15 fourth-graders (ages 9 to 10), who were randomly selected from all the students in these grades (except those evaluated by their teachers as having below-average intelligence) at an elementary school in a small rural farming community, were tested. The adult group consisted of 21 individuals with no history of psychiatric disorder. The adults tested had a mean age of 33 and a mean of 13.8 years of formal schooling.

Analysis of Data

Data were analyzed by analysis of variance. The manic and schizophrenic groups were compared by t tests to the adults, who were considered a control group; t tests were also used to compare the manic and schizophrenic patients to the children, who were considered to be an index of regression. Scores of the first- and fourth-grade children were pooled, although the older children showed more behavioral overinclusion and less underinclusion, because the children as a group varied in the same direction in comparison with the other three groups. It was hypothe-

[1]Reference (15) is available on request from M. Harrow, Department of Psychiatry, University of Chicago, Ill. 60637.

sized that the two psychotic groups would have scores different from those of the normal adults and similar to those of the children, which would reflect their tendency to regress cognitively and to express autism.

RESULTS

As table 1 indicates, the differences among the four groups revealed by an analysis of variance were significant in most of the areas assessed by the Goldstein-Scheerer Test. Only in richness did the four groups fail to show a significant difference. The variance among groups was highly significant in behavioral overinclusion, conceptual overinclusion, and autistic thinking; the groups also differed in underinclusiveness, but less strikingly.

Tables 2, 3, and 4 indicate the sources of variance among the specific groups based on a series of t tests. In table 2 the manic and schizophrenic groups are each compared to normal adults, who were considered to represent a normal baseline on the various aspects of conceptual style measured by the Goldstein-Scheerer Test. As that table indicates, manic patients differed more from normal adults than did schizophrenic patients. These two groups differed in behavioral overinclusion ($p<.01$), conceptual overinclusion ($p<.001$), and autism ($p<.001$). Schizophrenic patients were also more autistic than the normal adults ($p<.05$), but they were less autistic than the manic patients. Unlike the manics, schizophrenic patients showed significant underinclusiveness when compared to the normal adults ($p<.02$).

The manic and schizophrenic groups are compared to the children in table 3. The manics differed from the children even more strikingly than they differed from the normal adults. They showed more behavioral overinclusion, conceptual overinclusion, and autistic thinking ($p<.001$ for all), and less underinclusiveness ($p<.02$). The schizophrenic patients, on the other hand, tended to have scores quite similar to those of the children. They differed significantly in only one area, autistic thinking, showing more autism at the .001 level.

The hypothesis that data from children can be used as a baseline to measure regressive cognitive style is of value only if the psychometric test used discriminates normal children from normal adults. Table 4 summarizes a statistical analysis of the five conceptual variables measured in the two normal groups. The scores obtained by normal adults on these five conceptual variables are treated as a normal baseline and the scores of the children are compared to them. The children differed significantly from the adults ($p<.01$) on two aspects of the test that measure overinclusive thinking; they scored significantly lower than the adults on behavioral overinclusion and significantly higher on underinclusiveness. They shared this increased underinclusiveness with the schizophrenic group (see table 3), but resembled the normal rather than the schizophrenic adults in their lack of autistic thinking, as one might expect from individuals considered normal.

The mean scores obtained by each of the four groups for each of the five cognitive variables examined indicate that the manic patients are quite different from each of the other three groups. Both psychotic groups are more autistic than the two normal groups. However, if this variable is eliminated, the schizophrenic patients' overall cognitive profile is more similar to the childrens' than to the manic patients'. Thus there appears to be a distinct difference in cognitive style, with manics showing more thought disorder and schizophrenics more regression.

TABLE 1
Analysis of Variance of Scores of Manic Patients (N=16), Schizophrenic Patients (N=18), Normal Adults (N=21), and Normal Children (N=30) on Five Cognitive Variables*

Variable	F**	Significance
Behavioral overinclusion	14.23	.001
Conceptual overinclusion	20.81	.001
Autistic thinking	17.88	.001
Richness	1.42	n.s.
Underinclusiveness	4.96	.01

*As measured by the Goldstein-Scheerer Object-Sorting Test (12).
**df=3,81.

TABLE 2
Comparison of Manic and Schizophrenic Patients to Normal Adults on Five Cognitive Variables*

	Normal Adults (N=21)		Manic Patients (N=16)			Schizophrenic Patients (N=18)		
Variable	Mean	SD	Mean	SD	Significance**	Mean	SD	Significance**
Behavioral overinclusion	24.00	11.64	54.63	46.83	.01	18.83	11.06	n.s.
Conceptual overinclusion	1.23	.62	3.31	1.77	.001	1.50	.92	n.s.
Autistic thinking	1.57	.92	3.43	1.50	.001	2.33	1.32	.05
Richness	1.71	.90	1.50	.50	n.s.	1.22	.54	n.s.
Underinclusiveness	1.76	.99	1.62	1.20	n.s.	2.88	1.52	.02

* As measured by the Goldstein-Scheerer Object-Sorting Test (12).
** By t test.

TABLE 3
Comparison of Manic and Schizophrenic Patients to Normal Children on Five Cognitive Variables*

Variable	Normal Children (N=30)		Manic Patients (N=16)			Schizophrenic Patients (N=18)		
	Mean	SD	Mean	SD	Significance**	Mean	SD	Significance**
Behavioral overinclusion	14.93	8.35	54.63	46.83	.001	18.83	11.06	n.s.
Conceptual overinclusion	1.13	.35	3.31	1.77	.001	1.50	.92	n.s.
Autistic thinking	1.20	.48	3.43	1.50	.001	2.33	1.32	.001
Richness	1.47	.82	1.50	.50	n.s.	1.22	.54	n.s.
Underinclusiveness	2.87	1.61	1.62	1.20	.02	2.88	1.52	n.s.

*As measured by the Goldstein-Scheerer Object-Sorting Test (12).
** By t test.

TABLE 4
Comparison of Normal Children (aged 6 to 10 years) to Normal Adults on Five Cognitive Variables*

Variable	Children (N=30)		Adults (N=21)		
	Mean	SD	Mean	SD	Significance**
Behavioral overinclusion	14.93	8.35	24.00	11.64	.01
Conceptual overinclusion	1.13	.35	1.23	.62	n.s.
Autistic thinking	1.20	.48	1.57	.92	n.s.
Richness	1.47	.82	1.71	.90	n.s.
Underinclusiveness	2.87	1.61	1.76	.99	.01

* As measured by the Goldstein-Scheerer Object-Sorting Test (12).
** By t test.

DISCUSSION

The nature of thought disorder is a matter of considerable debate among both psychiatrists and psychologists. Assessing thought disorder by using the Goldstein-Scheerer Test taps only a few aspects among the many that have been discussed. It is useful to view this test as investigating two dimensions: a dimension measuring inclusiveness, with extremes of overinclusive and underinclusive thinking; and a dimension assessing originality of thinking, with extremes of autism and richness. The Goldstein-Scheerer Test does not examine other aspects of thought disorder that are often considered significant, such as inability to think logically, interpenetration of themes, or paraphasias and agrammatisms. It does, however, indirectly evaluate abstractness and concreteness of thinking, since the ability to sort abstractly is built into both the inclusiveness and the richness dimensions, although there is no direct measure of either abstractness or concreteness. In spite of the limitations inherent in the version of the Goldstein-Scheerer Test used in this study, one can make some generalizations about the quality and quantity of thought disorder in mania and schizophrenia.

Manic patients showed relatively more thought disorder than schizophrenic patients when compared to normal adults. In particular, they were markedly higher on the two measures of overinclusiveness and somewhat higher on autistic thinking. This does not necessarily mean, however, that manics have more of all types of thought disorder. Since overinclusiveness is more characteristic of mania than of schizophrenia, we should perhaps discard the concept of overinclusive thinking as a means of explaining schizophrenic thought disorder and should instead begin to apply it to mania. More particularly, we should perhaps speak of overinclusive versus underinclusive thought disorder. If we conceptualize thought disorder in this way, schizophrenic and manic patients are both disturbed in this dimension when compared to normal adult control subjects, but the schizophrenics are disturbed in the direction of underinclusiveness and the manics in the direction of overinclusiveness. Thus, rather than considering thought disorder as pathognomonic of schizophrenia, we should recognize that it occurs in both psychotic illnesses, but in different forms.

If one accepts the assumption that one can measure regression by using the conceptual style of children as a standard, then the notion that schizophrenic thinking is regressive receives some validation from this study. The conceptual profiles of the schizophrenics are similar to those of the children, with the exception that schizophrenics tend to be quite autistic in comparison with normal children. The quality that the schizophrenic group shares with the children is a marked underinclusiveness, and conversely, a lack of overinclusiveness, as well as a lack of richness. This seems to suggest that the poverty of thought often noted in

schizophrenic patients may be a return to more primitive levels of cognitive behavior. Whether this regressive pattern of thinking is due to psychological mechanisms (such as retreat in the face of overwhelming anxiety) or to organic ones (such as a biochemical lesion in areas of the brain devoted to complex integrative or associative functions) is an issue that cannot be resolved by an objective psychometric study of this sort.

Although manic and schizophrenic patients differ strikingly on this measure of regression in that manics show little resemblance to children in conceptual style, both psychotic groups have high scores on autism. Thus, autistic thinking and regressive thinking may not necessarily be the same, although it is often assumed that these terms may be used interchangeably. The autistic thinking measured in this study tends to be bizarre, personalized, and idiosyncratic. Both psychotic groups show this disturbance in thinking, while it is minimal or absent in normal children and adults. The regressive thinking displayed by the schizophrenics, on the other hand, tends to be primitive, narrow, restricted, and unimaginative. It may be of value to separate the concepts of regressive thinking and autistic thinking in future studies of thought disorder, seeing the former as characteristic of schizophrenia and the latter of all types of psychotic disturbances.

Comparisons of clinical symptoms in groups of patients are reliable and valid only to the extent that sampling is accurate. The samples used in this study were based on predefined diagnostic criteria. The boundaries of the schizophrenic sample, in particular, were narrowly drawn, to exclude schizoaffective patients and other schizophrenics with only brief episodes of illness. Although the patients were not all chronic, the schizophrenic sample was nevertheless skewed because patients having symptoms for less than 2 months were excluded. Furthermore, paranoid and nonparanoid patients were mixed because the sample was too small to permit subtyping. Future studies of thought disorder should examine subtypes within the various categories of schizophrenia (schizoaffective versus nonaffective, acute versus chronic, paranoid versus nonparanoid) to determine whether they manifest different types of thought disorder.

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Behavioral Changes Following Surgery for Obesity

BY MARK J. MILLS, J.D., M.D., AND ALBERT J. STUNKARD, M.D.

The authors interviewed 69 subjects who had had jejuno-ileal bypass surgery for obesity to determine the relationship between weight loss and eating behavior after surgery. All patients had been extremely overweight; at an average of 37 months after surgery, their mean weight was reduced by 39%. There were four significant postoperative changes: voluntary restriction of food intake, "normalization" of eating patterns, resistance to eating during intense emotion, and far less dysphoria during postsurgical weight loss than during previous weight loss efforts. The authors therefore suggest that factors other than impaired intestinal absorption may account for some portion of postsurgical weight loss.

JEJUNO-ILEAL BYPASS surgery for the treatment of massive and intractable obesity was introduced more than a decade ago (1, 2). The rationale for this procedure was that impairing the absorption of nutrients by radically shortening the small bowel would reduce caloric intake sufficiently to control obesity (3–7) and that this control would continue even in the presence of large intake of food. Favorable responses to this operation have resulted in its cautious and increasing acceptance (8–10), but the original rationale has received little examination (11).

The present study, which was undertaken to test hypotheses that questioned this rationale, derived from prior clinical experience with 12 bypass patients. The hypotheses were as follows: 1) jejuno-ileal bypass surgery is followed by voluntary restriction of food intake, 2) surgery is also followed by an apparently involuntary "normalization" in the pattern of food intake, 3) these reductions of food intake predict the degree of weight loss, and 4) postsurgical weight loss is accompanied by less dysphoria than previous weight losses. Briefly, we anticipated that reduced food intake as well as reduced absorption would be responsible for the weight loss of jejuno-ileal bypass patients.

When this work was done, Dr. Mills was a student at the Stanford University School of Medicine, where Dr. Stunkard is Professor of Psychiatry. Dr. Mills is now a resident in psychiatry, Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, Calif. 94305.

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METHOD

The study population consisted of all patients available for follow-up who had been operated on for obesity at the Harold D. Chope Community Hospital, San Mateo, Calif., more than 1 year prior to the study (N=69). All patients were operated on by the same surgeon (Basil M. Meyerowitz, M.D.); 46 received an end-to-side jejuno-ileal bypass wherein 36 cm of proximal jejunum and 10 cm of terminal ileum were anastomosed (12). The remaining 23 patients received modifications of this operation involving slightly less radical resection. After surgery, patients were placed on a recommended regimen of a diet low in fat, spices, and green vegetables. They were told not to drink alcoholic beverages and were given oral vitamins A and D, parenteral vitamin B_{12}, and oral potassium. Oral vitamins E and K, calcium, and magnesium were prescribed as needed. Sixty-two patients received diphenoxylate (Lomotil) to decrease the frequency of bowel movements.

Patients were chosen for surgery on the basis of four criteria: greater than twice predicted weight, good general health, age ranging from 16 to 55 years, and a screening interview with a psychiatrist.

At the time of interview, the population contained 56 women and 13 men whose ages ranged from 21 to 54 years (mean=41±9.9). Sixty-eight percent of the subjects (N=47) had become overweight by age 12, and 93% (N=64) were obese in their twenties (2 of the 5 exceptions were patients who received bypasses in their teens). All had been severely obese for years and had dieted many times without sustained weight loss. Fifty-two percent of the subjects were married, 29% divorced or separated, 6% widowed, and 13% single. Forty-eight percent were Protestant and 35% were Catholic. The mean income was $11,000 per year, and the mean educational level was 12.8 years.

A period of 1 year between surgery and interview was selected in order to ensure that the main weight loss had occurred. The mean interval between surgery and interview was 37±19 months. A total of 82 patients had been operated on at this facility at least 1 year before the study began. At the time of interview, 6 of the 82 had died—1 each from halothane hepatitis,

atherosclerotic heart disease, chronic liver failure, an unidentified neurologic disorder, suicide, and unidentified intraoperative causes. Five had moved out of state; we decided to study only those living in California. We obtained interviews with 69 of the 71 available patients.

Each subject was contacted in advance and interviewed at a place of his/her choice. A structured interview lasting about 50 minutes was conducted by one of us (M.J.M.). The interview involved 104 questions in the following categories: changes in eating habits (N=41), demographic information (N=20), patterns of weight change (N=18), complications of surgery (N=18), and weight loss (N=7).

TABLE 1
Weight Changes in 69 Patients Following Bypass Surgery (in kilograms)

Category	Reported Weight Mean	Reported Weight SD	Ideal Weight* Mean	Ideal Weight* SD	Excess Weight** Mean	Excess Weight** SD
Before surgery	150	26	60	7.6	89	22.0
After surgery	91	17	60	7.6	31	16.0
Difference	59	9	—	—	58	6.0

*Computed from height-weight tables compiled by the Metropolitan Life Insurance Company.
**Defined as reported weight minus ideal weight. Values for this category do not exactly correspond to those of the other two categories because excess weight could not be calculated for 1 subject.

RESULTS

The mean weight loss for all subjects was 59±9 kg. The mean preoperative weight was 150±26 kg, falling to 91±17 kg at the time of examination an average of 37 months after surgery (see table 1). This loss of 59 kg constituted 66% of the total preoperative excess weight and 39% of the total preoperative weight. The mean percentage overweight fell from 149% (range, 80%–238%) to 52% (range, −3.5%–156%) after surgery. At the time of interview, 70% of the subjects reported that their weight was stabilized, 23% that it was decreasing, and 6% that it was slowly increasing. Of the 69 subjects, all but 5 reported weight losses of more than 25%; those 5 had weight losses of 6%, 17%, 18%, 22%, and 24%.[1] There was no significant correlation between the time since surgery and weight (or percentage weight) lost, suggesting that weight had largely stabilized during the year after surgery.

These weight losses contrast sharply with the subjects' previous attempts at weight reduction. The mean maximum weight loss in previous attempts was 25.3±15.2 kg (range, 0–150). Furthermore, these losses were maintained for an average of only 11 days (range, 0–90) in contrast to the more than 25 months of weight loss reported by the patients after surgery.

Hypothesis 1: Voluntary Restriction of Food Intake

Sixty-one patients (88%) suffered from severe postoperative diarrhea. Bowel movements increased dramatically from a preoperative mean of 1.8±2.5 per day to 17±12 in the month following surgery. At examination an average of 37 months after surgery, bowel movements had decreased to a mean of 4.7±3.8. Differences between these means were significant ($p<.01$, Student's t test). All 61 subjects with diarrhea reported rectal tenderness; they also reported voluntarily restricting their food intake in an effort to alleviate this symptom. The 8 subjects without severe diarrhea did not report restriction of their food intake.

Hypothesis 2: Normalization of Eating Habits

The normalization of food intake following surgery was a most striking behavioral change. There was significant normalization in both eating patterns and resistance to eating during periods of intense emotion.

We predicted 10 changes in eating patterns: 1) decreased frequency of eating binges, 2) less night eating, 3) less snacking, 4) less difficulty in stopping eating, 5) reduced craving for sweets, 6) lower alcohol consumption, 7) decreased food consumption in general, 8) less food eaten during meals, 9) fewer meals, and 10) increased frequency of breakfast consumption.

Figure 1 depicts subjects' responses to 7 of the 10 structured questions designed to investigate these predicted changes. There were 5 possible responses; answers were assigned numerical values from 1 to 5, where 1 equals "much more than before surgery" and 5 equals "much less than before surgery." Means, standard deviations, and standard errors were then calculated and Student's t test was applied. Since all of the reported behavioral changes were in the predicted direction (that of normalization), only that part of the scale is presented in figure 1. The 7 predictions related to these questions were confirmed: all values were significantly different ($p<.01$) from no change, the null hypothesis. In addition, the number of meals consumed fell from 3.5±1.7 (range 1–9) before surgery to 2.6±0.8 (range, 1–4) after surgery ($p<.01$). Similarly, 38 persons reported increased frequency of breakfast consumption, while only 11 reported a decrease ($p<.05$, Student's t test). Thus 9 of our 10 predictions were confirmed—the only prediction not borne out by the data was that related to decreased alcohol consumption. Thus, the part of the normalization hypothesis dealing with eating patterns was strongly confirmed.

We also predicted increased resistance to eating during times of intense emotion. Figure 2 depicts the reported changes in eating behavior during periods of intense emotion (6 negative and 2 positive emotions). Re-

[1]Subjects were asked two questions about their weight during the structured interview, one on their weight at the time of surgery and one about their current weight. Therefore it is not possible to report the pattern of weight loss for the group or to determine when the maximum postsurgical weight loss occurred; however, only 4 subjects reported that their weight was increasing at the time of the interview.

FIGURE 1
Changes in Eating Patterns of 69 Jejuno-Ileal Bypass Patients after Surgery

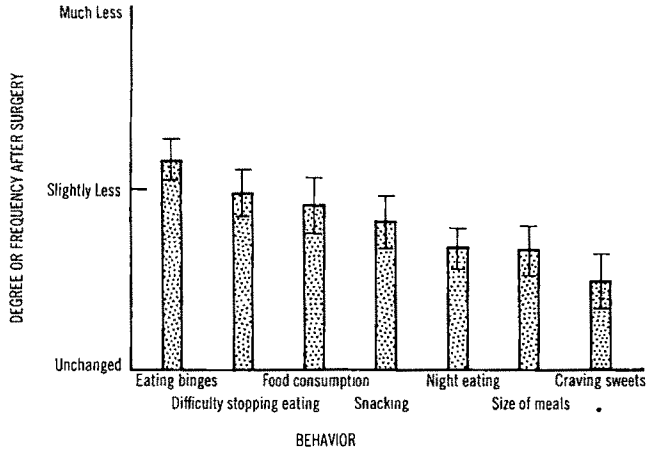
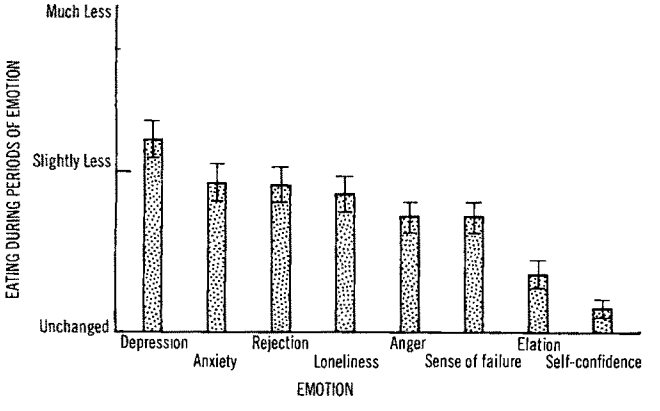


FIGURE 2
Changes in Bypass Patients' Eating Behavior During Periods of Intense Emotion (N=69)



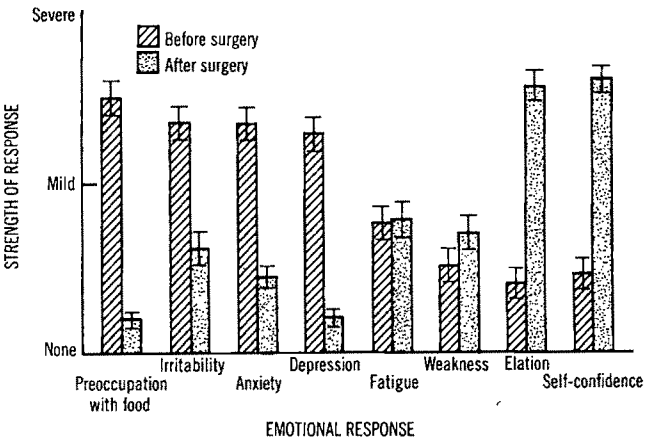
sponses were ranked in the same way as those related to normalization of eating patterns and similar analyses were carried out. All of the values presented in the figure were significantly different from no change ($p<.01$, except self-confidence [$p<.05$]). The second part of the normalization hypothesis was thus strongly confirmed.

Hypothesis 3: Reduced Food Intake and Reduced Weight

The correlations between percentage of weight lost and both voluntary restriction of food intake and normalization of eating habits were ascertained. The first part of this hypothesis was not confirmed: subjects who reported voluntary restriction of food intake did not differ significantly in weight loss from those who did not.

Since two aspects of the normalization hypothesis were tested, we explored two relationships: 1) the relationship between changes in eating habits and percentage of weight lost and 2) the relationship between

FIGURE 3
Emotional Responses to Weight Loss Before and After Jejuno-Ileal Bypass Surgery (N=69)



increased resistance to eating during periods of intense emotion and percentage of weight lost. The former relationship showed no statistically significant pattern. The latter variables, however, were positively correlated; subjects reporting the largest increase in resistance to eating during intense emotions also reported the largest percentage of weight lost. Table 2 presents the individual changes in eating in response to emotions that were significantly correlated with increased percentage of weight lost ($p<.01$, Spearman rank-order correlation).

Hypothesis 4: Responses to Weight Reduction

Our fourth hypothesis was that subjects would experience less dysphoria during weight loss after surgery than they had experienced during previous attempts at weight reduction. In order to investigate this hypothesis, subjects were given structured questions regarding emotional responses to weight loss, with answers ranked from 1 to 3 (none, mild, and severe). Mean values were calculated for responses related to the preoperative and postoperative periods and differences were analyzed via matched-pairs t tests.

Figure 3 depicts the contrast in emotional responses to weight loss before and after surgery. The four most common untoward responses to weight reduction before surgery (preoccupation with food, irritability, anxiety, and depression) were present to only a slight degree postoperatively ($p<.01$). Two responses to weight reduction, fatigue and weakness, were present to approximately the same degree postoperatively. Two positive emotions, elation and self-confidence, had been experienced by few patients before surgery but were common after surgery ($p<.01$).

PREDICTORS OF WEIGHT LOSS

Three variables in this study were predictors of the percentage of weight lost by each subject at the time of

TABLE 2
Mean Percentage of Weight Lost (± SE), by Susceptibility to Eating During Times of Intense Emotion (N=69)*

	Susceptibility After Surgery					
	More		Unchanged		Less	
Emotion	N	Weight Lost	N	Weight Lost	N	Weight Lost
Depression	1	62±38	21	34±2	47	39±2
Anxiety	1	62±38	29	33±2	39	40±2
Loneliness	1	30±9	35	35±2	33	40±2
Sense of failure	—	—	41	35±1	28	42±2
Rejection	—	—	35	34±2	34	41±2
Anger	—	—	42	34±2	27	42±2
Elation	2	28±11	52	37±1	15	40±3
Self-confidence	1	27±7	62	38±1	6	37±7

* Percentage of weight lost is significantly correlated with emotional susceptibility in each case ($p < .01$, Spearman rank-order correlation).

the interview. There was a significant positive correlation between initial weight and percentage of weight lost ($r=.394$, $p<.01$). The heaviest subjects not only lost the most weight but, surprisingly, actually lost a greater *percentage* of their excess weight. A significant negative correlation was found between age and the percentage of weight lost ($r=-.308$, $p<.01$); younger subjects lost more weight following surgery. There was also a significant positive correlation between decrease in eating in response to anger and weight loss ($r=.359$, $p<.01$). The multiple regression coefficient of the percentage of weight lost versus these three variables was .548 ($p<.01$). More than 30% of the individual variance between subjects in terms of percentage of weight lost was thus accounted for by these three variables.

DISCUSSION

The most striking finding of this study is the degree to which the eating patterns of the subjects tended to change in the direction of those of nonobese people. This change took place both in the general pattern of food intake and in the consumption of food during periods of intense emotion. Some of these changes have been recently reported: less food with each meal (13, 14), less eating between meals (13, 14), eating fewer meals (14), and less night bingeing (13). This kind of change does not occur in the vast majority of patients undergoing weight reduction (15, 16), nor had it occurred in the past experiences of the subjects. This suggests that the circumstances of weight reduction following surgery are quite different from those following traditional methods of weight reduction. The difference applies to such habits as eating binges, night eating, and snacking, as well as to the reduction in eating during intense dysphoric emotions. For the first time in their memory, subjects found that following surgery these emotions did not have the power to elicit eating responses.

The finding that three variables (initial weight, resistance to eating during periods of anger, and age) accounted for more than 30% of the variance in percentage of weight lost suggests, as has been previously proposed (8–10), that subjects for jejuno-ileal bypass surgery should be carefully chosen. Since subjects who were the youngest and the most overweight lost the greatest percentage of their weight, these factors can be added to the criteria for selection of candidates for this surgery.

The final major observation of this study has been previously reported (11) and denied (13) and therefore merits emphasis. This finding is the dramatic decrease in untoward responses and dysphoria during weight loss following surgery as compared with the subjects' previous weight reduction experiences. Weight losses of the degree achieved by our subjects are frequently associated with severe untoward responses, particularly of an emotional nature (17, 18), and this had been true of our subjects. The dramatic decreases in such responses following surgery (illustrated in figure 3), provide support for the use of bypass surgery in the treatment of massive and intractable obesity.

The findings of this study suggest that a major change may occur in the biology of obese individuals following jejuno-ileal bypass surgery. Only some of the changes we have reported were achieved by voluntary efforts on the part of the patients; many occurred without special effort. It was as if the operation had lowered a set point about which the subjects' body weight had been regulated (19). There are at least three factors that may contribute to these profound changes in regulation of body weight. First, surgery may have provided the subjects with "medical evidence" that their obesity was not their fault and in effect gave them a new opportunity to choose more appropriate eating behavior (11, 13). A second explanation is that subjects were aversively conditioned to limit their food intake; the vast majority of the subjects had severe diarrhea and rectal tenderness following surgery, and these symptoms were exacerbated by ingestion of food. Such symptoms provided a powerful

incentive to curtail eating. However, in this study aversive conditioning is not a sufficient explanation of the weight loss of the subjects—those who did not report these symptoms lost as much weight as those who did. A third explanation is that as subjects lost weight they became more active and thus accelerated their weight loss.

In summary, this study has demonstrated that impaired absorption may not account for all the weight loss of patients following jejuno-ileal bypass surgery and that decreased food intake in a variety of circumstances for a variety of reasons may play a part in that loss.

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The Significance of Childhood Neurosis for Adult Mental Health: A Follow-Up Study

BY SHERWOOD WALDRON, JR., M.D.

The author compared the mental health of 42 young adults who had suffered from a neurosis in childhood with that of 20 control subjects. Various aspects of functioning were clinically assessed using the Current and Past Psychopathology Scales and the Health-Sickness Rating Scale. More than 75% of the former patients were at least mildly ill at follow-up, compared with only 15% of the control group. Intergroup differences in diagnoses and symptoms are described. The findings establish the fact that neurotic children require much more effective treatment than they have traditionally received in order to attain a good prognosis for adult mental health.

NEUROTIC CHILDREN may be referred for treatment because the presenting symptom itself is distressing, but more commonly treatment is considered on the basis of concern about the child's future development. Because of a lack of adequate follow-up studies, we do not know for certain whether neurotic children have an unfavorable long-term prognosis. Even the demonstration of short-term improvement with treatment (1) does not tell us whether such improvement predicts ultimate adult mental health, and there is reason to think that it does not (2).

The few long-term follow-up studies available (3, 4) suffer from deficiencies in design because they lack either a clinical evaluation of the patients or a control group. It is too hazardous to compare their findings with those of epidemiological studies of psychiatric disturbance in the general population (e.g., 5, 6) because the latter generally use a questionnaire-interview approach by nonprofessionals that may result in misleading conclusions when compared to the results of assessment by clinicians (7).

Several long-term follow-up studies of children suffering from various specific neurotic symptoms (8–13) have suggested the importance of these difficulties to adult mental health. I thought that a long-term follow-up study of neurotic children in general would be more interesting if we studied one of these subgroups as well. I chose children with school phobia because of the evidence that impairment continued into adolescence (13), while the symptom itself tends to remit during childhood with most short-term therapies. Therefore extensive psychotherapy for this condition would ordinarily be indicated only if the long-term prognosis were unfavorable.

For two reasons, I selected the control group from children who were in the same elementary school class as each of our former patients. This sample would presumably reflect the normal expectable outcome for an unselected population, with the omission only of those children too disturbed or damaged to maintain attendance at an ordinary school. In addition, the selection method would serve as an effective control for age, sex, and social class.

We wanted to use a clinical appraisal of these young adults because of the tenuous relationship between indirect measures of mental health and clinical findings. Clinical assessment itself has often been found unreliable (14–16). Fortunately, two instruments have been developed, based upon clinical appraisal, which have shown excellent reliability: the Menninger Clinic's Health-Sickness Rating (HSR) Scale (17, 18), in which ratings are anchored to case descriptions, and the Current and Past Psychopathology Scales (CAPPS), a semi-structured clinical interview with rating scales developed by Endicott and Spitzer at Columbia University (19). These two instruments made possible a follow-up that would be both clinically meaningful and reliable.

Our research group independently assessed the clinical status of the children and their families when they were seen in our clinic in order to provide baseline values that could be compared with outcomes and to clarify the syndrome or syndromes of school phobia. This systematic study has already been reported (20).

Dr. Waldron is Clinical Assistant Professor, Department of Psychiatry, Albert Einstein College of Medicine, Bronx, N.Y. His address is 35 East 85th St., New York, N.Y. 10028

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METHOD

Subjects

A child psychiatrist screened the charts of all patients under age 13 who were first evaluated at our clinic between 1955 and 1962 (N=627). Using the classification of psychopathological disorders in childhood of the Group for the Advancement of Psychiatry (21), we could differentiate between those children who suffered from neurotic disorders or other milder conditions (e.g., reactive disorders or developmental disturbances) and the sicker children. Those who had experienced a school phobia were identified from among the neurotic group. Criteria of classification were first refined in a pilot series of charts, resulting in excellent interrater reliability.

Two-thirds of the over 600 children were considered sicker than neurotic and were excluded from further study. Forty-five of the 203 neurotic children were identified as having had school phobia. We drew up a panel of 35 subjects from the group with school phobia; this was the maximum number that we could study. We then matched one other neurotic child to each of these children with school phobia on the basis of sex, age, age at referral, and year of referral to the clinic. We selected our control subjects from the class that each child with school phobia was attending when the phobia first appeared; from each class we selected the child whose surname alphabetically followed that of the phobic child.

Procedures for locating the subjects were similar to those described by others (4, 22). Considerable persistence and ingenuity were required. Introductory letters were followed by a home visit unless the subject refused. Thus we visited the homes of nonresponders. The home visits were made as far as 200 miles from New York.

Primary Follow-Up Instruments

The semistructured clinical interview (the CAPPS) is of sufficient flexibility to permit the interviewer to explore areas of particular clinical significance as they emerge during the interview. The assessment includes various scales of symptomatology as well as measurements of sexual and interpersonal relationships and functioning in the roles of spouse, parent, housewife, student, and employee. We also added a few other individual scales of particular interest to us. Summary scales have been previously derived by factor analyses of the more than 170 CAPPS scales. A computer program has also been developed which produces reliable and valid psychiatric diagnoses when applied to these scale scores of each individual (23).

Most of the CAPPS scales use a 6-point rating ranging from "none" through "minimal," "mild," "moderate," and "severe" to "extreme" on each dimension rated. We were particularly interested in the overall rating of severity of illness. There is a 6-point severity of illness scale of the CAPPS, which we coordinated with the 100-point HSR scale by achieving a consensus among 3 clinicians as to what points on the 100-point scale corresponded to the 6 degrees of severity of illness specified above. Each time we rated an individual for severity of illness on the CAPPS, we used the case descriptions provided with the HSR scale, gave a rating on the 100-point HSR scale, then converted it to the corresponding score for severity of illness expressed on the 6-point scale (see footnote to table 1 for score conversions). We developed 4 subscales of this overall health-sickness rating, reflecting different aspects of mental health and based upon the 7 subscales originally developed at the Menninger Clinic.

Follow-Up Interviews

I conducted most of the interviews; the rest were done by another child psychiatrist. All of the interviews were tape recorded. Each of us was first trained and evaluated for reliability. Subsequently, ongoing reliability was monitored by independent ratings of an unselected sample of the tape recordings of interviews. Reliability was excellent for the various scales; for example, the most important judgment, the HSR score, showed an intraclass correlation coefficient of .87 between the two raters. Neither interviewer had any knowledge of the old records or of the classification of the subject at the time of the interview, except as the subject revealed it himself in the course of the interview.

RESULTS

We were able to locate 91% of our 105 subjects, of whom two-thirds agreed to be interviewed. We saw 24 phobic subjects, 18 subjects with other neuroses, and 20 controls. Fifty-eight percent of the follow-up group were men. Most subjects were of middle- to lower-class origin. The subjects averaged 22 years old at follow-up; fewer than 5% were younger than 18.

The interviews lasted an average of 3½ hours. The interviews were so lengthy because we encouraged the subjects to discuss their life story in their own way. The interviewer then pursued pertinent lines of inquiry as in a regular initial psychiatric interview. Visiting homes in the absence of a response was important to our success, since one-third of those interviewed had not answered our letters.[1]

We could compare the subjects who were interviewed with those who were not because we had extensive ratings of clinical aspects of the parents and families of virtually all of the former patients in childhood and of the former patients themselves as children. There were negligible differences between the 2 groups of subjects on all ratings from childhood. (See reference 20 for descriptions of the dimensions rated.) This finding reassures us that the 40% of our 105 subjects

[1]Comparison of responders and nonresponders showed negligible mean differences in HSR scores.

who were not seen in the follow-up were probably not very different from those who were seen.

Former Patients

When we compared those persons who had had a school phobia with those who had had other neuroses, we found that the phobic subjects 1) seemed more dependent (29% versus 6%), 2) had a greater tendency to somatize (42% versus 11%), and 3) had had more difficulty completing their secondary school education (25% versus 6%). Aside from these 3 findings, the groups were not distinguishable on our multiple measures. These measures included a number of factors postulated in the literature as typical of school phobics in later years, such as leading lives more centered around the home of origin, tending to avoid new situations, and being more inhibited in the expression of aggression (13). Both groups of former patients were more inclined toward these behaviors than the control subjects were. Because there were so few differences between the 2 groups of former patients, it seemed reasonable to contrast the former patients as a whole with the control group for the remainder of our analysis.

Diagnoses and Health-Sickness Ratings

The school-phobic patients did not differ as a whole from the other neurotic patients in distribution of diagnosis or degree of illness at follow-up. However, there were striking differences in these areas between the former patients as a whole and the control group (see table 1). We found that 40% of the former patients, but none of the controls, received specific diagnoses from the computer program ($p<.01$, chi-square analysis). Twenty-one percent of the former patients were diagnosed as having a specific neurosis,[2] 14% as having a personality disorder including drug dependence, and 5% as being psychotic, although neither of these 2 patients had ever been hospitalized. Thus we found a wide range of psychopathology in the former patients.

The HSR scales showed an even more striking difference between the former patients and the controls: more than 75% of the former patients were at least mildly ill, compared to only 15% of the controls ($p=.001$).

Since diagnoses were derived from computer analysis of the scores on specific variables, whereas the level of illness was a global rating by the interviewer, it is interesting to examine the relationship between the two. There were no specific diagnoses for those who were not ill or minimally ill, whereas close to half of those who were mildly to moderately ill received a specific diagnosis. The author, who is psychoanalytically trained, reviewed the cases of patients who were found to be ill but not diagnosed. All of them appeared to fit into the category of character disorders in the psychoanalytic sense (24), although they were not suitable for a diagnosis from DSM-II (25).

The relationship between the nature and degree of illness of the 2 groups can be summarized as follows: 64% of the former patients were mildly to moderately ill, predominantly with a neurosis or character disorder, while another 12% were severely ill, predominantly with a personality disorder or psychosis. In contrast, only 15% of the control subjects were mildly to moderately ill, all with character disorders. None of the controls was more severely ill.

We were also interested in which of the different aspects of mental health was impaired. The data in table 2 demonstrate that the former patients were quite impaired in the severity of their symptoms and in their interpersonal relationships and less impaired occupationally and in breadth of their interests. In all aspects they were substantially less healthy than the control subjects.

Individual CAPPS Variables

The clinical significance of the subscale differences between the two groups (table 2) can be understood by examining specific clinical differences between them (table 3).

The former patients showed greater difficulty than the controls in functioning as wage earners and as students, with about one-third being impaired. However, the differences between the 2 groups were not so great as in some other areas.

Disturbances in interpersonal relationships were extensive. More than one-fourth of the former patients were more than minimally socially isolated, compared to none of the controls; almost two-thirds had substantial problems in adult sexual adjustment, compared to only one-fifth of the controls. Because many of the subjects were just entering adulthood and therefore would not usually have married yet, we added an assessment of functioning as boyfriend or girlfriend to that of spouse if the relationship seemed enduring and serious. If we examine spouses and boyfriends and girlfriends together, we find that only 50% of the former patients had such a relationship, compared to 80% of the controls ($p=.025$). Of the former patients who did have such a relationship, 52% of them were at least mildly impaired, compared to only 6% of the controls.

There were various symptomatic differences between the 2 groups. Nearly half of the former patients suffered from at least mild depression, compared with only 1 of the control subjects. They also suffered more from anxiety and phobias than the controls, but the differences were not statistically significant. Almost one-fourth of the former patients showed more than a minimal degree of narcotics abuse and/or antisocial behavior and/or belligerence, compared with none of the controls. There were other symptomatic manifestations that were present in small numbers of the former patients; 2 patients had a history of homosexual acts and 1 patient had a perversion. More of the former patients also showed impaired use of leisure time and incapacity for pleasurable activities. In all, there were

[2]Four suffered from depressive neurosis; 2 from hysterical neurosis, conversion type; 1 from phobic neurosis; 1 from anxiety neurosis; and 1 from obsessive-compulsive neurosis.

TABLE 1
Health Sickness Ratings and Diagnosis at Follow-Up of 42 Former Patients (FP) and 20 Control Subjects (C)

Health-Sickness Rating*	No Specific Diagnosis		Neurosis**		Personality Disorder		Psychosis		Total		Percent	
	FP	C	FP	C	FP	C	FP	C	FP	C	FP	C
1—None	1	10	0	0	0	0	0	0	1	10	2	50
2—Minimal	9	7	0	0	0	0	0	0	9	7	21	35
3—Mild	11	2	1	0	1	0	0	0	13	2	31	10
4—Moderate	4	1	7	0	3	0	0	0	14	1	33	5
5—Severe	0	0	1	0	2	0	2	0	5	0	12	0
6—Extreme	0	0	0	0	0	0	0	0	0	0	0	0
Total number	25	20	9	0	6	0	2	0	42	20	—	—
Percent	60	100	21	0	14	0	5	0	100	100	100	100

* HSR scores on the 6-point scale correspond to the original 100-point scale as follows: 1=86–100, 2=76–85, 3=66–75, 4=51–65, 5=26–50, 6=0–25.
** The numbers for neurosis have been corrected for computer overdiagnosis of phobic neurosis when the clinical picture did not warrant this specific diagnosis.

TABLE 2
Percent of Former Patients and Control Subjects with More Than Minimal Impairment on the Health-Sickness Rating Scale and Subscales

Item	Former Patients (N=42)	Control Subjects (N=20)	Significance*
Overall HSR*	76	15	$p < .001$
Occupational role*	48	5	$p < .001$
Interpersonal relationships*	79	25	$p < .001$
Severity of symptoms*	74	20	$p < .001$
Breadth and depth of interests	52	10	$p < .01$

* Significance was determined by analysis of variance over entire 6-point range (Student's t test).

many ways in which the former patients appeared to be more impaired than the control group.[3]

Relationships Between the Various Measures

The differences between the former patients and the control subjects on individual variables were reflected in significant differences between the 2 groups on the relevant summary scales. This set of findings bears out the significant differences found in subscales of the HSR (table 2) and adds a partly independent confirmation to the validity of our principal findings. This is because the summary scale scores are founded upon the individual variables, which are usually much more concrete and specific than the HSR subscales, leaving less latitude for rater bias. Intercorrelations between the HSR subscales and summary scales demonstrated the same relatively high degree of relationship between different aspects of mental health as had been previously demonstrated in the extensive study at the Menninger Foundation (26). We know from that study's sophisticated methodology that this is not simply a halo effect but that it reflects a true interdependence between various aspects of mental health.[4]

Unreliability of Symptomatic Assessment

The relationship between symptomatic and clinical aspects of mental health alluded to in the previous section may be examined by classifying a subject as symptomatically ill if he obtained a score representing more than minimal impairment on at least 1 of the symptom variables in table 3. Then one can correlate this measure of illness with clinical illness as defined by a score representing more than minimal illness on the HSR scale. The degree of correlation between the two measures was relatively small for the 2 groups (κ=.46 for the former patients and .49 for the control subjects) (27).

However, separate examination of the degree of illness determined for the former patients and controls reveals an apparent bias in the results of the symptomatic assessment. Among the former patients, almost the same proportion was found to be symptomatically and clinically ill (69% versus 76%). However, a much higher proportion of the controls was found to be symptomatically than clinically ill (35% versus 15%). This difference between the 2 groups was just short of significance despite the small sample sizes (p=.052). Thus we add further evidence to the findings of Dohrenwend and associates (7) and Masterson (28) that symptomatic assessment of controls leads to overdiagnosis of mental illness[5] and that one must be very cautious in making conclusions based upon questionnaire-interview assessments of mental health.

[3]We might not have discovered the actual differences between the former patients and controls if we had assessed them during adolescence instead of young adulthood. Our retrospective assessment of functioning in adolescence showed only small differences in the quality of adolescent friendships, sexual adjustment, and academic performance. A substantial number of the control subjects, as well as the former patients, showed malfunction.

[4]A table of intercorrelations is available from the author upon request.

[5]Robins and Braroe (29) reported a similar finding, although they interpreted it differently.

TABLE 3
Percent of Former Patients and Control Subjects with More Than Minimal Impairment on Various CAPPS Ratings

Variable	Former Patients		Control Subjects		Significance**
	Number Rated*	Percent with Impairment	Number Rated*	Percent with Impairment	
Occupational functioning					
Wage earner	34	32	16	6	$p < .05$
Student	21	43	8	0	$p < .05$
Interpersonal relationships					
Social isolation	42	26	20	0	$p < .025$
Adult sexual adjustment	34	62	16	19	$p < .01$
Spouse	10	50	10	0	$p < .025$
Spouse and boyfriend/ girlfriend	21	52	16	6	$p < .005$
Symptoms					
Depression	42	40	20	5	$p < .005$
Anxiety	42	43	20	25	n.s.
Phobia	42	19	20	10	n.s.
Narcotics abuse/antisocial behavior/belligerence	42	24	20	0	$p < .025$
Interests					
Impaired use of leisure time	42	21	20	5	n.s.
Incapacity for pleasure	42	21	20	0	$p < .05$

* Not all patients qualified for a rating on each of the variables.
** One-tailed chi-square analysis was used since the overall trend had been established.

DISCUSSION

Our principal finding is that most of the neurotic children had impaired mental health in adulthood, whereas most of the ordinary school children were not impaired. Is the different proportion of illness found in these 2 groups comparable to findings of previous studies and therefore of general significance? The only controlled study, that of Robins (4), suffers from a lack of direct clinical assessment. Even so, in that study there were significantly more healthy controls than former patients (4, p. 137).

I examined in detail the reported findings of the various clinical follow-up studies (7–13) and found that case descriptions and descriptions of the frequency of various degrees of impairment permit some assessment of whether the degree of illness of those groups is generally comparable with ours. For instance, Levy (10) provided detailed case descriptions of the clinical follow-up picture of 19 children whose mothers were overprotective. Only 2 or 3 of these 19 former patients would appear to be well enough to have been rated healthy or only minimally ill on the HSR. In all, I feel that the findings of all of these studies are quite compatible with ours and demonstrate substantial impairment when account is taken of differing methodology.

It is more difficult to find clinical studies of subjects who are comparable to our control group. We cannot compare our findings with those of the large-scale epidemiological studies for the reasons discussed earlier. I know of only one clinical study of a normal sample in which the levels of illness can be compared with those of our subjects. In that study (7) the low rates of illness for heads of households are quite comparable to those found in our control group. Therefore, such studies as we are able to find support our most important conclusion: children who come to a psychiatric clinic because of neurotic difficulties or developmental disturbances have an unfavorable prognosis for mental health in adulthood. This group of children is not identical to the large proportion of children who develop transient symptoms in the course of development, particularly during the oedipal period (2, 30, 31). For most of these children psychiatric help is not sought.

We cannot determine directly the cause-and-effect relationship between referral for neurotic symptoms in childhood and adult mental health. But it is reasonable to assume that those children who cannot find a more adaptive way to resolve conflicts than the formation of such troublesome symptoms may be demonstrating an impaired capacity for neutralization or an impairment in ego strength that will lead to continuing unhealthy development (32, p. 235).

We cannot say on the basis of our sample whether the unfavorable prognosis applies to all groups of neurotic children. However, we may anticipate the findings to be reported in a later paper by saying that generally, it was not possible on the basis of the clinical pic-

ture in childhood to differentiate those neurotic children who would have good outcomes from the others. It may eventually be established that certain types of neurotic difficulties do not usually result in adult impairment, but there is no evidence from this study to support such a comforting conclusion.

The relationship between outcome and treatment received will also be the subject of a later report. It will suffice here to say that no patient received adequate treatment by psychoanalytic standards. If the treatment received resulted in some improvement for some of these children, then without intervention the former patients would have been, if anything, sicker. Thus our findings make necessary the conclusion that these children need effective treatment. What remains to be established is the validity of psychoanalytic clinicians' impressions that sufficiently careful and intensive treatment in childhood frequently leads to good outcome in adulthood.

One methodological point deserves emphasis: the diagnostic approach to mental health is of somewhat limited utility and there is good reason to emphasize a quantitative assessment of degree of illness, which can be described as a dimensional approach to psychiatric disorder (33). Many areas of medicine have required just such grading of cases as the Health-Sickness Rating Scale provides (for instance, in the outcome of different grades of cervical cancers) before significant clarifications about prognosis can be made. Thus, although the clinical grading of psychiatric cases may not permit us to predict whether a child will develop a character disorder, a specific neurosis, or personality disorder, it does permit us to predict with some accuracy the later degree of illness.

CONCLUSIONS

This study has provided evidence in support of the following points.

1. Children with neurotic difficulties sufficient to bring them to professional attention have a much worse prognosis for adult mental health than normal control subjects. Therefore if any extensive psychotherapeutic technique such as child analysis can be shown to alter this prognosis, it should be used.

2. Depending on the population involved, estimation of the degree of psychiatric disturbance through questionnaire-type interviews administered by nonprofessionals can lead to overestimation of the degree of illness and to substantially incorrect conclusions. Clinical assessment appears to be the only currently available method to avoid this pitfall.

3. Approaching psychiatric illness from the point of view of degree of disturbance offers promising opportunities to enlarge our understanding and predictive powers, which may be obscured by too exclusive an adherence to a nosological approach.

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Prompt Publication Policy

The *Journal* would like to remind authors who are particularly interested in early publication (whether to establish priority of an idea, reveal innovative results of new research, or for other pressing reasons) of its prompt publication policy. Under this policy, short (maximum of six double-spaced pages) manuscripts will be considered for publication in the Brief Communications section of the first available issue. These manuscripts must still undergo expert scrutiny before being accepted, and two months must be allowed for the printing process. However, the time required for publication of these short articles can be lessened considerably.

Authors who would like to have their manuscripts considered under this policy should indicate this desire, as well as the reasons for it, when they submit their manuscripts.

Evolution of Behavior Disorders Into Adolescence

BY ALEXANDER THOMAS, M.D., AND STELLA CHESS, M.D.

The New York longitudinal study has followed the development of 136 subjects from infancy into adolescence. The authors report the distribution of diagnoses and outcome of 43 children who developed behavior problems before the adolescent period and the characteristics of 5 new cases diagnosed in adolescence and discuss issues of prediction. The analysis of the longitudinal data for subjects with and without behavior disorders permits discussion of two of the general issues in the literature: the significance of adolescent turmoil and the distinctness of adolescence as a developmental stage.

ADOLESCENCE, with its complex, rapid, and dramatic developmental processes, is a period of special interest to all students of human behavior. It is during this period that the individual comes to physical maturity, establishes his or her emotional independence, begins to achieve an adult level of cognitive functioning, and crystallizes a separate individual psychological and legal identity (1).

For the psychiatrist, the problems of adolescence are indeed challenging. Many of the serious mental illnesses of adult life begin to be evident during this period, but their symptomatic manifestations may be very different than they are in the older individual. The adolescent patient is no longer a child but not yet an adult, and this makes his treatment especially demanding.

Theories of the developmental dynamics of adolescence generally tend to emphasize the conflict of the strivings for individuation, independence, and the establishment of a mature self-identity in the context of the persistent dependent ties to parents and family. Many writers view the tension, rebelliousness, and impulsivity of adolescents as resulting from this conflict and consider such turmoil a normal and necessary consequence of the adolescent struggle for separation, independence, and the beginning of mastery of the adult world (2–6). Others question this concept and caution that adolescent turmoil may in fact signal the development of significant psychiatric disorders (7, 8). Psychoanalytic theory interprets much of the behavior of the adolescent as a "reawakening of oedipal attachments," a period in which "the work of the oedipal period has to be carried out once again" (9).

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Dr. Thomas is Professor of Psychiatry and Dr. Chess is Professor of Child Psychiatry, New York University School of Medicine, 550 First Ave., New York, N.Y. 10016.

THE NEW YORK LONGITUDINAL STUDY

The New York longitudinal study has provided a special opportunity to investigate several issues in the development from childhood into adolescence. This study has followed the behavioral development of 136 children from early infancy into adolescence. The group is primarily a middle and upper-middle class population with native-born parents. (Demographic characteristics of the sample have been detailed previously [10].)

Forty-three of the 136 youngsters have developed behavior disorders of varying types and severity at different age periods in childhood. The data on the total sample include information gathered longitudinally and anterospectively at sequential age levels from early infancy on related to the child's individual characteristics of functioning at home, in school, and in standard test situations; parental attitudes and child-care procedures; special environmental events and the child's reactions to such events; and intellectual functioning. In addition, psychiatric evaluation has been done for each child presenting symptoms and, wherever necessary, has included neurological examination or special testing. Clinical follow-up of children with problems has also been carried out systematically.

The anterospective longitudinal behavioral and psychometric data, the clinical psychiatric evaluation and follow-up, and the availability of those subjects in the sample without problems as a comparison group have provided a special opportunity to investigate the genesis and evolution of behavior disorders and of specific symptomatology and to test the validity of various theoretical formulations advanced to explain these phenomena. We have also explored the usefulness of counseling procedures for parents that emphasize the development of consonance between the child's temperamental characteristics and the environmental demands and expectations he or she faces. The findings of these studies through the early school years have been reported previously (10–14).

We have conducted separate extended open-ended interviews with each child and his or her parents when he or she reaches the age of 16. The interview covers such subjects as the adolescent's school functioning,

extracurricular activities, sexual involvement, drug use, current problems, future plans, and attitudes toward parents, siblings, and peers. Out of our total sample of 136, 95 have thus far reached age 16; only 5 have refused to be interviewed. All the parents have responded. The 90 adolescents who have been interviewed were generally open and frank in their discussions.

CLINICAL SAMPLE

Of these 95 adolescent subjects, 43 had been diagnosed as clinical cases at an earlier age. Forty of them were described in an earlier work (10); 3 additional cases were identified in the childhood period following that report. Each case was followed up at periodic intervals; the frequency of follow-ups was determined by clinical considerations. With children in direct treatment, the therapist was consulted as to diagnosis and course. On the basis of these data and the new interviews at age 16, the clinical course and outcome was defined for each youngster.

After clinical psychiatric evaluation by one of us (S.C.), 5 additional clinical cases were identified as having started during the adolescent period. These youngsters were referred for this evaluation either by direct parental request or as the result of discussion with our staff interviewer. Other subjects clinically evaluated for similar reasons were judged as behaviorally normal. The criteria for the various diagnostic categories were detailed in our previous report (10).

A number of our findings regarding the adolescent period are currently in preparation (15, 16). This presentation, our second for the adolescent period, will concentrate on the course of the clinical group, the dynamics of the evolution of symptomatology, and predictive issues related to outcome. (Our first report was on the course of 3 youngsters with brain damage [17]).

Table 1 shows the age at onset of symptoms in the 48 cases and the original diagnosis assigned to each case. The largest number of cases came to light during the ages 3–5; the second largest number occurred in children aged 6–8. These cases are, for the most part, a mild reactive type of behavior problem. Very few additional children developed problems during the ages of 9–14. A larger number of new cases then appeared in children 15–18 years old, and these were considerably more serious: 2 neurotic character disorders, 2 neurotic behavior disorders, and 1 moderate reactive behavior disorder.

The findings in this sample conform to general clinical experience. A number of children developed behavior problems, mostly of a mild nature, in the preschool and early school years, very few new cases appeared in the later childhood and early teen years, and then a new group of clinical problems of greater severity developed in adolescence. The disorders in the younger age periods were presumably reactive to the demands for socialization and school adaptation and those in older children to the stresses and conflicts of the adolescent period.

Although the total number of clinical cases seems high, this is a cumulative figure; at any one age period, the number would be much lower due to the number of recovered cases. Also, some of those in the mild reactive category might have recovered spontaneously and not come to psychiatric attention if they had not been subjects in the longitudinal study.

OUTCOME IN ADOLESCENCE

Table 2 shows the outcome in the adolescent period for 42 of the 43 clinical cases diagnosed in the childhood period. (The 43rd case, originally diagnosed as mild reactive behavior disorder at 7.3 years, has not yet been followed up.) As can be seen in the table, 19 cases (almost 50%) recovered, 5 improved, 3 were unchanged, 8 became mildly or moderately worse, and 7 became markedly worse. There was no significant correlation between the severity of the original disorder and outcome in adolescence. It is of interest that very few cases were unchanged—if there was no recovery or improvement, the problem almost always became worse. In all of the cases originally diagnosed as reactive behavior disorder, this worsening led to a diagnostic change to neurotic behavior disorder or neurotic character disorder.

Those youngsters who recovered during the childhood years have remained well in adolescence and appear to be functioning on the same level as the adolescents who did not show behavior disorders in childhood. Recovery cannot be considered spontaneous, since in all cases we extended guidance to the parents. Nineteen cases had direct treatment; 4 of these improved or recovered and several others are still in treatment, the ultimate effect of which cannot yet be evaluated. Of course, some of the children, especially the milder cases, might have recovered spontaneously without any therapeutic intervention.

In a number of the milder cases with rapid recovery, the improvement could be ascribed to the effectiveness of the guidance given to parents, which was directed toward correcting excessive stress on the child created by demands and expectations that were dissonant with his temperamental characteristics. Similarly, lack of improvement or worsening in other cases may have resulted from parents' unresponsiveness to such guidance and advice (10, 14). In some of these cases, the unhealthy parent-child relationship became more stressful and conflict-ridden as the youngster grew older, with corresponding worsening of the behavioral disorder.

In other instances, the reasons for favorable or unfavorable outcome are not obvious, In several cases the overall parent-child relationships were positive, the parents carried through the necessary changes to lessen the excessive demands on the child, and no other sources of unusual stress were evident, yet there was

TABLE 1
Age at Onset of Symptoms for 48 Children with Clinical Diagnoses

Original Diagnosis	Age at Onset of Symptoms (years)						Total
	Less than 3	3–5	6–8	9–11	12–14	15–18	
Reactive behavior disorder							
Mild	1	20	7	0	0	0	28
Moderate	1	5	2	0	0	1	9
Severe	0	2	0	1	0	0	3
Neurotic behavior disorder							
Moderate	0	0	0	0	1	2	3
Severe	0	1	0	0	0	0	1
Neurotic character disorder							
Mild	0	0	0	0	0	1	1
Severe	0	0	0	0	0	1	1
Brain damage with behavior disorder							
Moderate	0	0	1	0	0	0	1
Severe	1	0	0	0	0	0	1
Total	3	28	10	1	1	5	48

TABLE 2
Original Diagnosis and Outcome

Original Diagnosis	Outcome					Total
	Recovered	Improved	Unchanged	Mildly or Moderately Worse	Markedly Worse	
Reactive behavior disorder						
Mild	15	0	0	3	6	24
Moderate	2	2	2	4	1	11
Severe	1	1	0	1	0	3
Neurotic behavior disorder						
Moderate	0	0	1	0	0	1
Severe	1	0	0	0	0	1
Brain damage with behavior disorder						
Moderate	0	1	0	0	0	1
Severe	0	1	0	0	0	1
Total	19	5	3	8	7	42

no improvement in the child's disturbance. In others, a pathological family environment continued, but the child improved or even recovered.

In cases where a pathogenic parent-child relationship persisted into adolescence, we have thus far identified one significant difference between the youngsters who improved or recovered and those who became worse. This involved the adolescent's ability to shift the center of his activities, interests, and emotional commitments from the home and the parents to extrafamilial individuals and groups. Adolescents who achieved this outside identity improved; those who did not grew worse. However, we do not as yet know why one youngster achieved this shift of identity and another did not, or whether the development of a separate identity was the cause or effect of the improvement. Our further analysis of the data is exploring possible correlations with the youngster's temperamental characteristics and special talents or attributes, as well as specific kinds of family pathology.

NEW CASES IN ADOLESCENCE

As noted previously, we have identified 5 new cases with adolescent onset. As a group, these disorders are more severe than those which started in childhood, although there are no instances of psychosis. The symptoms are varied, including depression, hostility, rebelliousness, and antisocial behavior. All of these youngsters exhibit poor performance in school, both academically and behaviorally, and poor impulse control. One girl has a history of neurotic promiscuous heterosexual behavior followed by a homosexual affair. There is no history of sexual problems in the other 4 cases, nor in the clinical cases that started in childhood. Our observation of the total study sample, nonclinical as well as clinical, indicates a wide range of sexual attitudes and behavior (16).

These 5 cases do not permit quantitative comparisons to temperamental characteristics with the rest of the study sample, as was possible with the larger

group of clinical cases starting in childhood (10). In the qualitative analysis of the longitudinal data it has been possible, as with the earlier cases, to trace the evolution of the behavior disorder in terms of an excessively stressful maladaptive interaction between the youngster's temperamental and other characteristics and his family and other environmental influences. However, our review of the anterospective longitudinal data of the childhood period has not as yet revealed any specific characteristics of these 5 adolescents or their families that could have been predictive of the development of pathology. However, our analyses of these data are still in progress and our conclusions are therefore incomplete.

COMMENT

The analysis of the longitudinal data on our subjects, both with and without behavior disorders, enables us to comment on two general issues—the significance of adolescent turmoil and the distinctness of adolescence as a developmental stage.

Many of the youngsters in our study showed one or more of the manifestations usually associated with adolescent turmoil—rebelliousness, conflict with parents and authority figures, negativism, impulsivity, tension, mood swings, and vacillation in goals and plans. In some cases these phenomena were symptomatic of definite behavior disorder; in other instances they occurred in youngsters who were coping effectively and constructively with the demands of the adolescent period and who did not show clinically significant psychopathology. Other adolescents who were also coping and developing on a healthy level showed no evidence of turmoil. Finally, some of the behavior problem group with symptoms in the direction of passivity and inhibition of functioning also had few or none of the characteristic manifestations of turmoil despite their clear-cut psychopathology and disturbed functioning. It is our impression that these differences may be related at least in part to individual patterns of temperament, although our data analyses are still incomplete in this regard. It will also be of interest to determine what correlation, if any, exists between the presence or absence of adolescent turmoil and psychological functioning in early adult life.

In any case, the evidence from this group of adolescents indicates that no a priori conclusion can be drawn as to the significance of the presence or absence of adolescent turmoil in any specific youngster. Each case has to be evaluated individually and in the perspective of overall behavior and functioning.

In reviewing the sequential histories of our study population we have found it most fruitful to view adolescence as a qualitatively new stage of development, in which the youngster faces new demands, expectations and issues, and to which he brings to bear his past experiences and characteristics and his new physiological, cognitive, and emotional capacities. Psychological development always represents a continuously evolving individual-environment interactive process, with qualitatively new elements in both the individual and the environment introduced at all sequential age-stages. This is especially true in adolescence, when there is a dramatic combination and interplay of the old and the new in psychological development.

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The Role of Neurosis in Phylogenetic Adaptation, with Particular Reference to Early Man

BY LEON SLOMAN, M.R.C.S., L.R.C.P.

The author hypothesizes that neurosis results from maladaptation and leads to further maladaptation, thus creating a vicious cycle that can be viewed as having played a significant role in human evolution. Individuals who were less successful in the struggle for survival during prehistory would have been more likely to become neurotic, a maladaptation that would magnify existing genetically transmitted differences in their viability and affect both the choice of a mate and the viability of their offspring. This process would have accelerated human evolution by favoring the most viable. The author relates this theory to his concepts of assortative mating and to other theories of the role of neurosis.

CURRENT THEORIES about the biological role of neurosis tend to be mutually contradictory and offer only partial explanations. One of the central issues is whether or not an adaptive or maladaptive role should be attributed to neurosis.

As a result of Freud's influence, clinicians tend to see neurotic symptoms as having a positive function, that of providing solutions to problems that otherwise seem insoluble. We postulate that this source of satisfaction is what allows them to persist for long periods almost unabated. In clinical practice, we tend to focus on the adaptive and compensatory components of neurosis. However, patients seek help because of symptoms of anxiety or depression, which they view as clearly maladaptive.

Within a psychodynamic framework, one can delineate a snowball effect in psychoneurosis whereby maladaptation leads to further maladaptation. For example, problems in the oedipal phase may contribute to subsequent problems in obtaining satisfaction of affectional needs or in productively harnessing aggressive drives. Alternatively, adaptive failures at a later age can lead to regression to earlier fixation points, which may precipitate neurotic symptoms and thereby compound the initial maladaptation. The view taken within this formulation is that neurosis results from maladaptation and then causes more maladaptation.

Dr. Sloman is Assistant Professor, Department of Psychiatry, University of Toronto, and Senior Psychiatrist, Clarke Institute of Psychiatry, 250 College St., Toronto M5T 1R8, Ont., Canada.

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Referring to neurosis, Gantt (1), a noted experimenter in the area of animal neurosis, commented that "maladaptation is as inherent in the physiology of the organism as is adaptation and, like adaptation, it has its basis on physiological structures." Gantt implies that maladaptation is an active process. It is easier to reconcile the concept of active adaptation than that of active maladaptation with what we know of biological function; Gantt's concept of active maladaptation may therefore be difficult to understand. In this paper, I will present a hypothesis to explain how individual maladaptation could have played an adaptive role for the human species. I will introduce the discussion by describing what I mean by the terms "maladaptation," "adaptation," and "neurosis" and presenting a brief summary of the theories that try to account for the maladaptive role of neurosis.

DEFINTION OF ADAPTATION AND MALADAPTATION

Adaptation has many meanings (2); here, it is used in a phylogenetic sense. The definition of natural selection as differential reproductive success between contemporary genetic alternatives implies that adaptation demands the historical context of becoming and cannot be discussed in absolute terms.

Maladaptation is a concept diametrically opposite to adaptation, but I will introduce an additional distinction in this paper. Whereas I will use a phylogenetic concept of adaptation, the term maladaptation will be used in an ontogenetic context. Maladaptation would therefore be likely to diminish the likelihood of reproductive success as well as the individual's ability to discharge his responsibilities toward himself and other members of the group. For example, maladaptation may hamper functioning within both the mating role and the parental role. This use of the term maladaptation (because of the inapplicability of the term natural selection, as discussed below) is not readily applicable to modern Western society. As is argued below, the series of individual maladaptations is seen as promoting the phylogenetic adaptation of the species as a whole.

NEUROSIS

Estimates of the prevalence of psychoneurosis in the general population vary with methodology and

diagnostic criteria. One study suggested a figure of about 20% (3). Although there has been some question as to whether experimental neurosis in animals meets the criteria of neurosis as set by clinicians (4), lower animals also seem to be susceptible to stress (5, 6).

There is evidence that neurosis is disadvantageous to man; individuals diagnosed as neurotic have been found to have a greater incidence of physical illness (7, 8) and higher mortality (9). Hinkle and Wolff (10) demonstrated that man develops illness most frequently at those times when he is having the most difficulty adapting to his environment. These illnesses tend to occur in "clusters," each of which consists of a variety of illnesses involving several body systems. They argue that "Man's relation to his social environment has relatively small influence upon the form which illness takes, but it has a major influence upon the time and the situation in which illnesses will occur and the course which they will pursue."

One might say that man has a generalized psychophysical propensity to disease (7). The greater the difficulty in adapting to the social environment, the more severe the illness, be it neurotic, physical, or both. This vicious cycle of neurotic maladaptation (which may extend to physical disease) will be discussed below.

Definition

I will use Hebb's definition of neurosis as an "undesirable emotional condition which is generalized and persistent . . . occurs in a minority of the population and has no origin in a gross neural lesion" (4). I will interpret "undesirable emotional condition" as referring to patterns of anxiety and depression without (in the interest of brevity) attempting to clarify the distinction between neurosis and psychosis. This definition will exclude the adaptive reactions of personality to anxiety or depression.

Current Theories of Neurosis and Adaptation

Prima facie, maladaptive behavior cannot be adaptive, yet neurotic maladaptation is very common in our society. This problem has challenged theorists. Jonas and Jonas (11) interpreted neurotic behavior as an "archaic response" and attributed a purely vestigial role to it. It is difficult to reconcile this hypothesis with the prevalence of the so-called vestigial phenomenon of neurosis. Clearly, if neurotic behavior were vestigial, it should occur very infrequently.

Barnett (12) proposed a relationship between psychoneurosis and psychosomatic disorders in man and "displacement behaviour" in animals, thereby implying no specific adaptive or maladaptive role for neurosis. Price (13), however, believed that neurosis could play an adaptive biological role. Although he viewed human neurotic reactions characterized by depression, anxiety, and lack of self-confidence as painful and incapacitating, which implies a maladaptive function, he hypothesized that they evolved as the "yielding" component of "ritual agonistic behaviour." Agonistic behavior covers all aspects of conflict between members of the same species, including threat, fighting, domination, submission, and retreat. Ritual agonistic behavior is designed to achieve the settlement of a dispute by an exchange of signals rather than blows (13). Because it avoids combat, yielding behavior apparently prevents unnecessary injuries or fatalities. Frank (14) also claimed that depression can play a biologically adaptive role, but he attributes this to the function of depression as a means of conserving energy.

Rodger (15) suggested that depression may be a biological mechanism for preventing incapable and rejected members from rejoining their herd. He thus views depression as maladaptive for the individual but adaptive for the group as a whole.

Although none of these theories is sufficiently comprehensive to explain the biological role of neurosis, neither Price's nor Rodger's theory is incompatible with the hypothesis I will advance below.

VICIOUS CYCLES OF MALADAPTATION

The view that neurosis results from maladaptation and leads to further maladaptation is basic to my theory. This vicious cycle operates in a situation where the inability to cope leads to anxiety, which compounds the person's difficulty in coping. A depressive vicious cycle may also manifest itself when repeated failure leads to a loss of initiative and drive, with feelings of hopelessness and helplessness that make further failure inevitable.

In behavioral studies, it has been demonstrated that increasing anxiety or "arousal above the optimal condition" leads to a deterioration in performance on certain tasks (16, 17); this is what happens in chronic psychoneurosis. Anxiety has also been shown to reduce the ability to improvise responses, which is likely to make adaptation more difficult (18). Inability to cope may therefore eventually lead to excessive "arousal," which results in greater maladaptation, creating a vicious cycle.

In order to understand the neurophysiological basis of this vicious cycle, one can refer to Hill (19), who differentiated two physiological patterns: a state of arousal experienced by the patient as anxiety or tension, and a state of "inhibition" or "depression-withdrawal." Hill noted that "both inhibition and arousal coexist in many patients and that inhibition is not merely a function of low arousal." It is interesting that this hypothesis was suggested in part by research evidence that inhibition may be an active process (20).

Furthermore, Malmo (21) described the concept of "behavioral arousal," which he related to psychoneurosis and anxiety. According to Malmo, chronic anxiety is a "disease of overarousal" or, in Selye's terms (22), a "disease of adaptation." The critical neural change is thought to be produced by a process of attrition from excessive and extended overarousal.

THE HYPOTHESIS

The biological mechanisms I will describe below are thought to have operated in prehistoric times and to probably no longer play any very significant evolutionary role. As Huxley said, "The human situation is so different from the biological, that it may prove best to abandon the attempt to apply concepts like natural selection to modern human affairs" (23). Therefore, one cannot rely too much on direct clinical observations to evaluate the validity of these theories.

According to the theory of natural selection, differences in viability between competing members of a group influence their relative chances of survival. If the genetic differences in viability are extremely small, they would have only a limited differential impact on the chance of survival.

My hypothesis is that those who were less successful in the competitive struggle for survival were likely to become more neurotic. According to this hypothesis, neurotic reactions represented maladaptive vicious cycles, which would have led to a magnification of small existing differences in viability. This would have accelerated the rate of human evolution in the prehistoric era.

It is thought that neurotic maladaptation results from the interaction between the individual and his environment. In primitive human societies, where there was an active struggle for survival, the less successful groups or group members were more prone to develop neurotic maladaptive patterns. As a result, those humans who were less viable due to genetically determined weakness in given areas relative to given environmental demands would have suffered from a greater degree of neurotic disability. In this neurotic vicious cycle, those who were least fit genetically would have been more rapidly eliminated in the struggle for survival. Although this mechanism would have been disadvantageous to some, it could have been advantageous to the survival of the group as a whole. The more active elimination of those who were least fit genetically would have resulted in increased genetic fitness in the surviving members. In this way, a small genetic difference between competing members of a group could have had a major impact on their relative chances of survival. Neurotic maladaptation could therefore be viewed as a mechanism that greatly accelerated the evolution of particular groups and of the human species in general by favoring the survival of the most viable individuals among early man.

GROUP SELECTION

If the potential to develop neurosis can only operate to the disadvantage of the individual, one might expect this potential to be eliminated by natural selection. How then is it passed on? This question could be answered by the concept of group selection (24, 25). Wynne-Edwards broke natural selection down into several categories that are functionally as well as conceptually distinct. He proposed the concept of group selection, which operates through success or failure of entire groups of individuals. Simply stated, this is natural selection operating at the group level. He feels group selection does no more than "correcting in part . . . the shortcomings of individual selection from the standpoint of the species as a whole." Whereas all members of the group have the potential to develop neurotic maladaptive patterns, only some would actually do so. This mechanism would have aided the survival of the group as a whole by accelerating the process of natural selection, which would lead to a more rapid adaptation of the group members over a period of generations.

In Darwinian natural selection, the most viable member of the group is likely to have the largest number of descendants and the least viable few or none. I propose that natural selection was accelerated by the following mechanism. Those who were less successful in the competition for survival were likely to become more neurotic. The experience of failure results in decreased self-esteem, feelings of inferiority, and reactions of anxiety and depression, characterized by indecision, hesitancy, and lack of confidence. According to this theory of neurosis, the severity of neurosis in prehistoric society would have been a rough index of the degree of maladaptation of the individual.

As the competitive struggle for survival led to the less successful members becoming more neurotic, there was a vicious cycle wherein the less viable developed maladaptive reactions, which led to further failure, resulting in even greater anxiety and depression. Faster elimination of those who were genetically less fit would have led to a more rapid spread of positive genetic mutations.

I will use as an example a group of Ojibway Indians before the advent of the white man whose survival depended on their success in hunting and trapping in a period of food shortage. One adolescent member of the group is a little more skillful than his peers. His success wins him general approbation and his parents feel proud of his achievements. This young man develops what Erikson termed a "sense of mastery" and a strong positive "sense of identity" (26). He will be labeled as "highly viable and minimally neurotic." By contrast, one member of the group is conspicuously unsuccessful. He becomes an object of derision and develops strong feelings of inferiority and a sense of identity diffusion. He will be labeled as having "low viability" and being "highly neurotic." One can examine the probable outcome of this situation in light of the different theories.

According to the theory of natural selection, the ability of the most viable member of the group to find food would have increased his chances of survival and vice versa. However, if the group of Indians shared the small amount of food that was available (as was probably the case), the variation in hunting skills might

have had little or no impact on differential chances of survival.

According to the theory of neurotic maladaptation, the most viable and therefore least neurotic member of the group would have had an added psychological advantage over his peers in the competitive struggle for survival. The least viable and therefore most neurotic member would have been at a particular disadvantage. His neurosis would have handicapped him in the struggle for survival by lowering his adaptive efficiency and increasing his predisposition to illness. His neurosis would also have influenced him in his selection of a mate, as will be described below.

NEUROSIS AND ASSORTATIVE MATING

The theory I have described dovetails with a theory of assortative mating that I have explained more fully elsewhere (27). In order to demonstrate this interrelationship, I will give a brief outline of that theory.

I have argued that the degree of neurotic maladaptation was at one time an index of environmental maladaptation. Neurosis is characterized by anxiety and depression, which can influence the attractiveness of personality, poise, and appearance. It has also been said that neurotics marry other neurotics (28). These ideas have been incorporated into a theory about the evolutionary role of human assortative mating. The theory states that assortative mating is a mechanism that, in ancient times, favored the mutual selection of those who were more viable. According to this theory, the most viable and least neurotic members found each other more attractive and chose each other as mates. This would have led to a segregation of "more viable" genes from the "less viable." As a result of assortative mating, the strong members united so that they in effect became stronger and the weak became weaker. By this mechanism, the genetic differences that already existed would have been increased.

Whereas neurotic maladaptation would have contributed to the elimination by natural selection of those who were less fit, assortative mating would have increased the differential advantage of the strong. The mechanisms of action of neurotic maladaptation and assortative mating would have been closely interrelated. Through their combined effect, they would have greatly accelerated the action of natural selection and thereby accelerated the rate of human evolution.

The relationship among neurosis, assortative mating, and viability can be illustrated by the use of the term "impotent." Impotence is often a manifestation of neurosis and obviously leads to problems in selection of a mate. The term also refers to an inability to master difficult situations. This linguistic linking of general ability, neurotic problems, and sexual ability might reflect the biological link that I am proposing.

Parents who are poorly endowed genetically are likely to have children who are also genetically handicapped. In addition, parents transmit neurotic patterns to their progeny by direct interaction (29). The descendants of those who are most viable will have multiple advantages. They will be genetically well endowed, less neurotic, and will enjoy a strong advantage in the struggle for survival.

To return to the example of the Ojibway Indians, the highly viable and minimally neurotic member would possess the poise, serenity, and other criteria of attractiveness that would lead to his mating with an attractive female, who would also be highly viable and minimally neurotic. The highly neurotic group member of low viability would be likely to select a very neurotic partner of low viability.

The descendants of neurotic individuals of low viability are at a disadvantage both genetically and because of being highly neurotic. Therefore, one might expect their strain to die out. Conversely, the descendants of the most viable individual, who would be minimally neurotic and highly viable, should continue to multiply.

ADDITIONAL ASPECTS OF THE HYPOTHESIS

The mechanism of neurotic maladaptation would have particularly favored the evolution of the most complex structures, such as the brain. Chance mutations with a favorable influence on man's intelligence were probably rare and of minuscule effect. However, as a result of the long-term interaction between competing individuals, these small differences could have led to the development of a maladaptive vicious cycle in the less well-endowed individual. Neurotic maladaptation would in this way have specifically increased the impact of these small genetic differences. This process would have greatly accelerated the evolution of man's intelligence. There is considerable ethological data to support this theory—these data have been summarized elsewhere (30). However, my main interest is in clinical implications (31).

Whereas the focus of this paper has been on a negative concept, namely neurotic maladaptation, I might point out the positive aspect of healthy adaptation (using the term now within the context of emotional maturity) as did Saul (32) in his description of the mature personality: "Freed from childhood patterns, he is discriminating and highly adaptable. And, among the many results of such development, his anxiety is at a minimum." Achievement of emotional maturity could be seen as a response that would have greatly aided the struggle for survival. Today, it might be seen as a way of making that survival more meaningful.

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Principles of the New Sex Therapy

BY WILLIAM H. MASTERS, M.D., AND VIRGINIA E. JOHNSON

The authors review the development of their rapid treatment dual-sex therapy for sexual dysfunction. The basic tenets of the new sex therapy include 1) sound knowledge of physiology, endocrinology, and metabolic function, 2) institution of psychotherapy only when organic factors have been identified or ruled out, 3) treatment of the couple as a unit by dual-sex therapy teams, 4) a short-term intensive program, and 5) education in techniques of verbal and nonverbal communication. Since its inception in 1958, the new therapy has been adopted, modified, and examined by clinicians and researchers all over the world. The authors suggest that professionals interested in effective treatment for sexual dysfunction should improve techniques, train personnel, and encourage sound research.

SINCE THE PUBLICATION of *Human Sexual Inadequacy* (1) in 1970, there have been many attempts by other health care professionals to interpret and extend the original concepts of dual-sex psychotherapy for the rapid treatment of sexual dysfunction. As part of the original research design, the Reproductive Biology Research Foundation has continued to reexamine its own philosophy and treatment methodology subsequent to further clinical experience, additional follow-up information, and constructive criticism from other sources. Although such an evaluation process might be expected to lead to broad changes in methodologies, the foundation's basic conceptual framework for treating sexual problems has not substantially altered. In view of the increasing clinical interest in both the theory and practice of therapy for sexual dysfunction, a glance at the original treatment tenets from a current perspective may be of interest.

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The authors are the Co-Directors of the Reproductive Biology Research Foundation, 4910 Forest Park Blvd., St. Louis, Mo. 63108.

SEX THERAPY IN 1958

In 1958, as we began to make plans for a clinical research program in the psychotherapy of human sexual dysfunction, we conducted a detailed review of existing methods and findings in the field. It was immediately apparent that the available research was fragmented and unsystematic, with a marked dichotomy between biological and behavioral data that reflected the scientific bias of the times. Clinical techniques were both time-consuming and unreliable, and there were many indications that these methods derived more from the therapist's personal investment in the psychotherapeutic process than from an objective knowledge of sexual function or a practical application of behavioral principles. Both published data and reports shared with us by people in the field clearly established the fact that health-care professionals were essentially ignorant not only of human sexual physiology but of the potential clinical application of such knowledge. It seemed that sexually dysfunctional patients were being treated with professional insight drawn either from the psychotherapist's own sexual experience—good, bad, or indifferent as it may have been—or from anecdotal material provided by previous patients.

The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

This perspective was even bleaker when viewed within the constraints imposed by unrealistic dogma and prevailing modes of psychotherapy that peremptorily dismissed any nontraditional approach as worthless. Given this situation, innovation appeared to offer at least a realistic chance to improve treatment efficiency and efficacy. To assess this potential, we felt it necessary to document extensively the effectiveness of our experimental therapy model along at least two dimensions—the number and diversity of cases treated and the duration of therapy follow-up. Therefore, the research format that was implemented in 1959 included a plan to gather five-year follow-up data on treatment outcome.

Simply stated, the research was designed to evaluate a two-week intensive psychotherapeutic approach to sexual dysfunction working in a format that examined the psychological and social components of sexual dysfunction along with the organic factors involved. This rationale was based on recognition of the fact that contemporary social values and pressures interact with individual sexual attitudes, experiences, and feelings derived from the past, and that no better microcosm of these elements could be found than a relationship in which one or both partners were sexually distressed. To ignore the relationship seemed not only to deny the importance of a partner's potential positive or negative contribution but also to waste an opportunity to investigate the social laboratory provided by the relationship.

ORGANIC ASPECTS OF SEXUAL DYSFUNCTION

At the outset, it seemed logical to assume that a thorough knowledge of male and female pelvic anatomy, endocrinology, and metabolic function would be a strong asset in any approach to the treatment of sexual problems. This scientific background seemed essential for accurate evaluation of the organic aspects of sexual dysfunction; it also offered an opportunity for enhancing the psychotherapeutic process by providing a factual data base from which questions on sexual physiology could be answered objectively. It has become increasingly apparent that such expertise also provides therapists with objective criteria for assessing the dimensions of clinical progress, both psychological and physiological, during the treatment process. Detailed knowledge of anatomy, physiology, and metabolic function will never replace the skills of a well-ordered and sensitive psychotherapeutic approach to sexual dysfunction, but neither can therapists realistically continue to ignore these areas in the psychotherapeutic process.

It is important that prompt and thorough assessment of the organic aspects of sexual problems be carried out prior to treatment. Approximately 5% of all cases of sexual dysfunction are of physical origin and a higher but undetermined percentage are of metabolic origin; thus psychotherapy should not be instituted until the possibility of organic etiologies of sexual distress has been either eliminated or identified and treated when necessary. It is just as unconscionable to treat sexual distress of metabolic or physiological etiology exclusively with psychotherapy as it is to treat a psychosomatic illness directly with surgery. In 1958 patients with complaints of sexual dysfunction were rarely checked as thoroughly as possible, either physically or metabolically, before psychotherapy was initiated. Unfortunately, comprehensive assessment of these factors before a commitment to long-term treatment is still far from routine.

LEARNING AND INSTINCT

An original and continuing premise of our treatment model is that sexual response is a natural function. Human sexual behavior is clearly a complex set of learned and instinctive phenomena interacting with personality dynamics, hormonal factors, and cultural influences. Penile erection and vaginal lubrication are just as clearly inborn reflexes, not learned behavioral responses. Just as the natural physiological functions of respiration, digestion, and urination are not taught to the newborn infant, the reflex pathways of sexual response cannot be taught. Sexual functioning is influenced by psychosocial input, just as other natural physiological functions are, and can be disrupted by anxiety, depression, or physical stress.

A major misconception that was at one time shared by patients and health care professionals was that sexually dysfunctional individuals can be taught to respond effectively (i.e., the impotent male can be taught to achieve erections or the sexually dysfunctional woman can be taught vaginal lubrication). This is like believing that we can be taught how to sweat or to make our hearts beat. Certainly, we do learn the behavioral aspects of any natural function. Because sexual functioning involves social as well as psychophysiological components, we must emphasize learning as it relates to sexual behavior, but our potential to respond to effective sexual stimulation is instinctual rather than learned. This does not mean that we cannot learn ways of facilitating our biological potential; unfortunately, it also implies that we *can* learn ways of disrupting our natural physiological processes.

Many individuals seek counseling on the assumption that there are reliable methods of teaching sexual response. This is not quite accurate. What does exist is the possibility of identifying the obstacles to effective sexual functioning that have removed sex from its natural context and suggesting ways to alleviate and/or circumvent these obstacles. Similarly, attitudes, behavior, and emotional environments conducive to individual sexual response can be identified and encouraged. When this is done, natural function usually takes over with surprising ease. This is one of the reasons why a brief but highly concentrated form of psychotherapy can be very effective in treating sexual dysfunction.

THE COUPLE AS THE UNIT

Another reason for the effectiveness of short-term intensive therapy for sexual dysfunction that was also among the original treatment tenets stems from the emphasis on the couple—the contention that there is no such thing as an uninvolved partner in any committed relationship where there is sexual dysfunction. This partnership component of sexual functioning would seem obvious; however, the frequent use of individual psychotherapy indicated that the importance of mutual involvement in the problem was not always fully appreciated. In our nontraditional methodology, we felt it was imperative to direct treatment to the education of both partners simultaneously rather than focusing exclusively on the sexual distress of the chief complainant.

We were particularly concerned about the established practice of sending marital partners to separate therapists, especially when husband and wife individually described separate sexual dysfunctions. When two marital partners, each with his or her own sexual dysfunction, are treated by different therapists with different professional orientations, professional capacities, and levels of interest in and knowledge of the field of sexual response, the chances of returning effective sexual response patterns to the marital partners individually or to the marital relationship are negligible.

This principle of involvement of both partners has been widely misinterpreted. No implication of causality is necessarily intended—it is often apparent that the genesis of one partner's sexual difficulties antedated his/her entrance into the relationship that presents for treatment. Alternatively, when sexual problems arise for the first time in a relationship where there had previously been satisfactory sexual interaction, the dysfunction often arises from circumstances that are totally unrelated to the functional partner. However, the functional partner is certainly involved in the problem to the extent that he/she may experience sexual frustration, disruption of other areas in the relationship, and reverberations in occupational performance, social patterns, and self- or partner deprecation.

THE DUAL-SEX THERAPY TEAM

A psychotherapeutic format using a male and female cotherapist team seemed particularly indicated in the treatment of the sexually distressed. Men generally have a limited experiential frame of reference from which to identify with female sexuality and have essentially no concept of the subjective aspects of female sexual function. Women often do not fully comprehend the subjective aspects of male sexual functioning or empathize with the depth of most men's ego involvement with their sexuality. A man simply does not know what woman's orgasmic episode feels like, and a woman will never experience the ejaculatory process. Neither sex finds it easy to evaluate for the other the relationship between effective sexual function and sexual identity.

Earlier therapeutic techniques often failed to produce the desired return to effective sexual function because the therapists (most of whom were men) simply presumed expertise in both the sexual function and the sexuality of the opposite sex. As we gain knowledge of male and female sexuality and as our experience with the basic science aspects of human sexual function increases, it is to be hoped that specific gaps in our appreciation of the sexual response patterns of the opposite sex will be reduced. However, in 1958 we had—and unfortunately we still have—no real concept of the depth of general ignorance of the sexuality and the sexual functioning of the opposite sex.

Another advantage of the dual-sex team is related to the tendency of therapists to inadvertently impress their own social or sexual value systems on sexually distressed patients. The dual-sex therapy team is, in theory, an excellent counterbalance to this unfortunate therapeutic tendency. A primary function of the team is to acquire knowledge of the sexual value system of each marital partner by careful interview techniques. The basic therapeutic approach is then to encourage the couple to achieve their mutual sexual goals and to identify and enjoy their own concepts of satisfying marital interaction rather than the therapists' suggesting or impressing their own social or sexual value system in the treatment. If each partner's goals can first be understood and appreciated by the therapists and can then be explained to both partners, the committed couple will have a far better chance of attaining effective functioning.

EDUCATION

We presumed in 1958 on the basis of 13 years' experience in working with problems of infertility that simple education of patients in the factual anatomy and physiology of human sexual response might, in some instances, relieve a significant amount of sexual dysfunction. Certainly, the practice of seeing the partners in a distressed sexual relationship as a couple rather than as separate individuals has enabled us to educate patients more effectively. In this context, when a couple is treated for sexual distress, the learning opportunity becomes a combination of individual gain and mutual enrichment. Rather than just learning of self, a man and a woman treated together have the opportunity to consider the dynamics of their relationship. Man learns of woman as woman learns of self and vice versa. This mutual educational opportunity is particularly effective as a therapeutic procedure in dual-sex therapy, where the cotherapists can educate by modeling interactive communication, by interpreting material from both male and female perspectives, and by reinforcing therapeutic content in different individual styles and contexts.

An example of the use of simple education in treat-

ing sexual problems is our approach to vaginismus and premature ejaculation. It has long been obvious that these two problems rarely respond to specifically directed psychotherapeutic approaches. When we started developing the new sex therapy in 1958, these sexual distresses were falsely aligned with such psychosocial problems as impotence and nonorgasmic states. In fact, some psychiatric textbooks listed and defined premature ejaculation as a form of impotence. The foundation took full exception to this concept. We felt that premature ejaculation and vaginismus would respond far better to simple education of both marital partners and to the techniques of specific psychophysiological reorientation of the individual's nonproductive sexual response patterns with the partner's cooperation. There is no doubt that conditioning of reflex response patterns, usually during initial opportunities for sexual interchange, plays a major role in both rapid ejaculation and vaginismus. Therefore, specific techniques to teach partners to help each other recondition these unfortunate reflex response patterns are far more effective in reversing these distresses than any known form of formal psychotherapy.

PERFORMANCE FEARS AND THE SPECTATOR ROLE

Education is only one component of psychotherapy, and it is far more valuable when combined with some degree of insight. In treating sexual dysfunction, insight often centers on the fears of sexual performance that are inextricably woven into the genesis of sexual problems. When fear of sexual performance dominates in a relationship, either or both partners sacrifice their spontaneity as they become spectators of their sexual action. As spectators, they watch and measure their own or their partner's sexual response patterns in an attempt to overcome their performance fears by noting obvious physical signs of sexual excitation. The sexual experience ceases to be a mutually shared expression of feelings and needs and is reduced to individuals attempting to act out their expectations. The tragedy of the spectator role is that it often directly confirms the participant's worst fears—the individual or his/her partner *is* slow to respond (or does not respond at all), which confirms the spectator's feelings of sexual inadequacy. This role takes sexual response out of context and usually results in impaired function.

Neither fears of sexual performance nor the closely related spectator role has been sufficiently recognized as a primary deterrent to effective sexual functioning. In addition, the depth of male performance fears usually is not fully understood even by committed female partners, since women have had little personal frame of reference from which to appreciate the devastating effect of these fears on the male ego. Too often, a woman's well-intentioned attempt to help her sexually apprehensive partner by masking her own sexual interest tends to prevent the sexual stimulation that he might experience in response to her obvious sexual involvement.

Techniques of directly confronting fears of sexual performance and providing specific suggestions to help overcome these fears became an integral part of the new approach to sex therapy. For example, carefully structured exercises that present an opportunity to appreciate tactile sensations without any demand for overt sexual responsiveness provide one avenue for reducing cultural pressures to perform and concomitant fears of performance. Direct verbalization of these fears, both in the therapy interaction and between members of the sexually dysfunctional couple, provides another means of defusing the magnitude of anxiety because unspoken fear often looms larger than verbally acknowledged fear. Reducing the anxiety arising from self- or partner-imposed expectations for sexual performance, whether real or imagined, usually results in a move toward more spontaneous sexual interaction. Similarly, as fears dissipate, the need to be a spectator decreases and the sexual experience improves as it becomes one of involvement rather than performance.

Once either or both marital partner's fears of sexual performance have been reasonably neutralized, spectator roles can generally be easily minimized. The technique of reversing sexual dysfunction by concentrating on neutralizing performance fears and minimizing spectator roles is based on the knowledge that sexual response patterns are natural phenomena and that dysfunction of these response patterns is reversed by removing psychogenic barriers to response, not by attempting to teach the individual how to function sexually. This technique also recognizes that patterns of behavioral interaction are learned aspects of a relationship that can be modified on the basis of mutual motivation and cooperative effort.

Within this conceptual framework, it was possible to view the specific sexual dysfunction within the context of an intimate interpersonal relationship. A sexual problem may represent an isolated facet of an otherwise healthy relationship, but sexual distress usually arises from hostility, poor communication, maintenance of a double standard, unrealistic expectations, deception, differences in reproductive goals, or a host of other factors. Unless therapy focuses primarily on the relationship as a whole rather than being restricted to the sexual component, important treatment dynamics may be overlooked. For example, when a couple's system of communication is improved, the status of the relationship often stabilizes. Then, in a time frame that allows the couple to develop reasonable familiarity with the new concepts, dysfunction can often be reversed with simple educational techniques and a knowledge of the specifics of sexual physiology. Although sexual response cannot be taught, effective means of communication can be, and the newly established state of effective communication often provides a direct catalyst to the pleasurable experience of satisfying, responsive sexual interaction.

NEW SEX THERAPY COMPARED WITH TRADITIONAL TECHNIQUES

It is most important to emphasize that although psychotherapeutic dogma has been challenged in these new therapy techniques, traditional psychotherapeutic principles have not been obliterated. What has changed, of course, is the focus of therapy. Rapid treatment for sexual dysfunction emphasizes the personal interactions of men and women as they seek to enhance a total relationship rather than treatment of individuals concerned with their own sexual needs or inadequacies.

Perhaps the most significant departure from past psychotherapeutic dogma was our contention that sexual dysfunction is not necessarily a symptom of underlying psychopathology. In 1958, it was almost universally believed that if underlying psychopathology could be identified and treated successfully, symptoms of sexual inadequacy would automatically be resolved, since they were only surface manifestations of a more deeply rooted neurotic or psychotic process. We have never denied that sexual dysfunction may be a symptom of underlying psychopathology, but we do believe most emphatically that sexual inadequacy is an entity all its own at least as often as it is a symptom of severe psychopathology. This marked departure from established psychotherapeutic concepts not only encouraged the development of the rapid treatment techniques but also stimulated dissemination of the concept of sexual response as a natural function.

We also had a strong clinical feeling that when the sexual inadequacy *was* obviously a symptom of existing psychopathology, real psychotherapeutic gain might result from a direct approach to symptom removal. This position is directly antithetical to the idea that symptoms should not be approached directly because their persistence serves the useful purpose of keeping the patient in therapy. The therapy program that we developed did not ignore individual psychodynamics. Rather, it recognized that identification of factors such as stress reactions, problems of self-esteem, psychopathology, and maladaptive defense mechanisms was essential to the efficiency and efficacy of therapy.

Time Frame of the New Therapy

We presumed that there would be a number of specific therapeutic advantages to the limited time frame of the new therapy, which usually consists of daily sessions over a two-week period. First, there is significant cumulative return for patients who are exposed to sexual material on a day-by-day basis, since they become involved in mutual eroticism with increasing ease. Sexual thought and action seem to arrive at a healthy accord in a progressive manner that leads the distressed couple toward a sense of erotic well-being as a unit. Second, it is much easier to enhance communication skills when each day's successes and problems can be freely recalled and analyzed during the next day's visit. Third, it is far more clinically effective to evaluate and deal with sexual problems and questions as they develop rather than after the fact. Fourth, daily exposure to the therapists provides an ideal opportunity to handle treatment-related crises that may develop. Such crises should be approached when they occur rather than after delays of days or even weeks. When two people are involved in a treatment crisis, any delay in dealing with their distress may be disastrous.

COUNTERTRANSFERENCE ISSUES

Despite the obvious advantages of rapid treatment, we recognized that there were significant problems inherent in this mode of therapy. For example, a rapid treatment format does not allow time for the development of traditional transference interaction and insights. However, we felt that therapeutic emphasis on classical patterns of transference would obviously distract the focus of therapy from the marital relationship to the relationship between the individual and either or both cotherapists. Individuals with prior experience in analytically oriented psychotherapy often demand, overtly or covertly, a chance to nurture and discuss transference dynamics. This frequently slows progress in therapy because it diminishes the level of shared feelings within the marital relationship.

In general, the dual-sex therapy team minimizes the importance of transference. By its very design, dual-sex therapy tends to emphasize and encourage emotional identification and commitment between marital partners rather than developing the patients' emotional dependency on their therapists. It is important, however, to recognize the distinction between a full working through of transference and ways in which elements of transference phenomena can be used in particular situations.

A good example of the use of transference in the new sex therapy is the psychotherapeutic approach to vaginismus, which includes education and instruction in use of a series of vaginal dilators. It is usually necessary to foster an extraordinary degree of trust on the part of the patient toward the therapist who is performing the pelvic examinations and introducing the dilators into use. For a day or two, as the woman is gaining confidence in her ability to overcome a painful and frustrating problem, her partner's participation in the use of the dilators is typically kept to a minimum. Deliberate development of this aspect of transference has been found to be highly effective, resulting in prompt reversal of the vaginismus so that the couple can focus on their sexual interaction without fear of interference from vaginal spasms. A brief transition period is then required to downplay the transference between patient and therapist; this is usually accomplished by repeatedly stressing to the woman that *she* is the one who brought about the change and emphasizing the catalytic role played by the cotherapists.

Obviously, the therapist's authoritative position is of major import. Acceptance of authority makes the

process infinitely easier and enables it to progress with the necessary rapidity. Closely directed transference is sometimes used to set the stage for a couple to develop a more effective focus on their own interaction. Needless to say, this use of transference must be individualized. The concept of controlling or channeling transference during rapid treatment for sexual dysfunction should be seen as unique to this method; it in no sense reflects denial of the importance of the objective use of transference phenomena in other therapeutic programs.

We thought that if patients' emotional identification with the therapists (beyond that necessary to establish the authority figure) was to be discouraged, it was necessary that the cotherapist team take particular care to avoid problems arising from countertransference. The sexually dysfunctional man or woman is incredibly vulnerable to any suggestion of sexual connotation from the authority figure. Sexual seduction, whether actual or fantasized by either patient or therapist, clouds any therapeutically encouraged trend toward emotional identification within the sexually dysfunctional marital unit. Overt evidence of countertransference can create a distraction of such magnitude that the rapid treatment technique can easily be prejudiced.

We have seen direct evidence of the ultimate in countertransference many, many times in histories of patients who had other types of psychotherapy for sexual dysfunction before considering dual-sex rapid treatment techniques. The ultimate in countertransference is, of course, a therapist seducing a patient into overt sexual activity. We feel that this approach to the extremely vulnerable patient with a dysfunction is professionally and personally inexcusable. The use of dual-sex teams provides a significant degree of built-in protection against any extensive devlopment of countertransference, much less seduction of patients.

We feel that when sexual seduction of patients can be firmly established by due legal process, regardless of whether the seduction was initiated by the patient or the therapist, the therapist should initially be sued for rape rather than for malpractice, i.e., the legal process should be criminal rather than civil. Few psychotherapists would be willing to appear in court on behalf of a colleague and testify that the sexually dysfunctional patient's facility for decision making could be considered normally objective when he or she accepts sexual submission after developing extreme emotional dependence on the therapist.

PEER REVIEW

Peer review is an integral part of patient care, and self-regulation certainly has been established and supported by the medical fraternity. There is, however, a lack of peer review for psychotherapists of any persuasion in the private practice of their art; not only has this been a major handicap to the therapist professionally but also an obvious obstacle to good patient care. There is currently less peer review of professional productivity in psychiatry than in any other major discipline in medicine. Once a psychotherapist has completed training, his or her work is rarely if ever subjected to peer review. This clinical tragedy slows and may prevent professional growth and maturity. Inevitably, it is the patient who suffers.

We believed that a dual-sex therapy team would provide a built-in opportunity for a modest degree of continuing peer review, since the members of the team would be immediately accountable to each other. While we did not consider this approach to peer review completely satisfactory, and did not feel it should be employed to the exclusion of formalized peer review programs, we did think that objective criticism from a co-therapist was far better than any self-review technique imaginable.

IMPROVING COMMUNICATION WITHIN THE COUPLE

Satisfying sexual interaction has always represented a mutually enhancing means of communication between man and woman. When sexual interaction is not initiated merely for the release of accumulated sexual tensions, what is its potential? As a means of communication, its potential is vast. In one important dimension, sexual communication can be viewed as the medium or opportunity for exchanging trust and vulnerability. Sexual interaction represents a uniquely vulnerable occasion for committed participants on both a physical and an emotional level. Within this framework of sex as a form of communication, we decided to concentrate much attention on developing techniques to improve intimate communication skills. We presumed that if effective communication were established outside the bedroom and sexual anxieties were reduced, sexual functioning would often improve. This presumption has certainly been supported by our clinical experiences of the last 18 years.

Sensate Focus

Our original approach was to focus on the nonverbal aspects of communication. Because the sense of touch is most identified with nonverbal communication and is the special sense used most often in sexual interaction, we developed the concept of sensate focus.

In order to achieve optimum effect, sensate focus should be used as a means of physical awareness by the partner doing the touching and not specifically or solely for the sensual pleasure or even sexual excitation of the partner being touched. Sensate focus in the early stages of therapy tends to dissipate anxieties related to sexual performance on the part of either or both spouses. Thus, in nonverbal communication, the therapeutic emphasis is on touching as a personal sensual experience and only secondarily, if at all, as a sexual opportunity. The sensual experience consists of exploring the textures, contours, temperatures, and con-

trasts of the partner's body in a manner and at a pace chosen by the individual doing the touching. By specifically structuring the sensate focus opportunities at the onset of therapy, it is usually possible to significantly reduce the constraints imposed by old habit patterns of sexual interaction. Concomitantly, removing stereotyped expectations of what sexual interaction "should be" often leads to an awakening of spontaneous natural response that has long been forgotten and sometimes never recognized.

We have noted with regret that the technique of sensate focus has been introduced out of context countless times by therapists and has been used indiscriminately as a means of sexual stimulation, even in public settings where the individual's sexual response has little opportunity of being supported by fulfillment of related needs. In these cases, the specifically designed function of self-awareness in the use of touch as an excellent means of initiating nonverbal communication usually is lost.

Verbal Techniques

Verbal communication, although often emotionally charged in a couple with sexual problems, is more familiar and thus more comfortable than nonverbal communication for many individuals. Because of this, a significant focus of the new therapy was education in more effective verbal communication. Many couples are surprised that the majority of time in therapy is devoted to this area. It is indeed dramatic to find how often improvement in verbal communication skills releases a couple's sexual feelings toward one another and thereby enhances their sexual relationship.

Isolation During Therapy

We felt that a period of uninterrupted attention to the relationship was necessary to allow the couples to integrate the new techniques of verbal and nonverbal communication into the existing patterns of their lifestyle. It was anticipated that the quality of this time would be enhanced by social isolation, which would minimize interference from the exigencies of daily life. There is a decided therapeutic advantage when couples are temporarily freed from the demands of children, friends, relatives, work, and other responsibilities in a therapeutic program. This is certainly not a new concept; in fact, it follows long-proven tenets of learning theory. Once the intense phase of learning new skills is mastered, briefer exposure will suffice to maintain or improve these skills.

CONCLUSIONS

In this paper, we have briefly described the original principles of treatment that were the foundation of the new therapy techniques for sexual dysfunction. Of course, there has been both significant modification of these principles and expansion of concepts of treatment as the clinical therapy program has been closely observed during its 18 years of existence, first at Washington University School of Medicine and since 1964 at the Reproductive Biology Research Foundation.

Obviously, there have been further modifications of concept, format, and technique in various centers throughout the United States and abroad. Some have represented significant innovations and additions, others have been poorly conceived and casually conducted. All of these improvements, alterations, and even distortions simply point to the immense public demand for adequate professional support in this hitherto ignored health care field. Now that the surface has been scratched, it remains for those interested in effective treatment programs to improve techniques, train personnel, and encourage both basic science and clinical investigation.

We all have such a long way to go.

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The Role of Sex Therapy in Marital Therapy

BY CLIFFORD J. SAGER, M.D.

Marital and sex therapy are clinically interrelated in most cases because they deal with different symptoms of overlapping aspects of the couple's total relationship. The connection between marital discord and sexual dysfunction determines the emphasis and course of treatment. For example, sex therapy is the immediate treatment of choice when sexual dysfunction produces secondary marital discord but not when severe discord precludes the possibility of good sexual functioning. The author presents case reports to illustrate the relationship of remote and immediate causes of marital and sexual problems and to emphasize the value of a multimodality approach to treatment.

THE ROLE of sex therapy in marital therapy has been vague and inconsistent over the past several decades. Marital therapy generally has included some sex therapy. New developments in our knowledge of the physiology of sex and the treatment of sexual dysfunctions have led to changes that can currently be described only as trends because of their newness. In this paper, I will discuss the role of the new knowledge of sex therapy as it affects marital therapy based on my experience in private practice and in a large family, marital, and sex therapy clinic and training facility.

Like the relationship between sex and marriage, the relationship between sex and marital therapy is highly complex. It would be wrong and an oversimplification to claim that sex therapy is merely a subspecialty of marital therapy. The goal of marital therapy, as I conceive it, is not simply or necessarily to make the relationship work but also to foster the growth of two individuals by harnessing the forces within the marital system and directing them toward constructive ends. Therapeutic interventions cover a wide gamut from insight-oriented interpretations to behavior modification and from individual treatment to groups and couples' groups. However, there is one common factor that categorizes all marital therapists: the use of the marital system in some conceptual and technical fashion as an integral part of the therapeutic program.

Revised version of a paper presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Sager is Psychiatric Director, Jewish Family Service, 33 West 60th St., New York, N.Y. 10023, and Clinical Professor of Psychiatry, Mount Sinai School of Medicine, City University of New York.

Sex therapy and marital therapy deal with different aspects of the same entity and consequently are almost always clinically interrelated. However, each has a validity of its own, rooted in its own theoretical substructure, and each employs a body of techniques that require specialized training and experience. Many clinicians see no clear-cut distinctions between sexual and marital symptoms. I have found that almost three-fourths of the couples I see have a mixture of marital discord and sexual problems in varying proportions regardless of whether their chief complaint is strictly of marital discord or of a specific sexual dysfunction. The problem is deciding when to focus on sex therapy, marital therapy, or both forms of therapy.

THERAPEUTIC FOCUS

Sex is one strand in the cable of bonding and therefore is intricately entwined with the other strands that keep the couple together and allow them to develop and grow. When several of these strands begin to fray, they must be identified and reinforced if the weakening relationship is to be strengthened. Occasionally it is only the sexual bond that needs reinforcement, in which case sex therapy is certainly the treatment of choice. More often, however, sexual dysfunction or dissatisfaction is so intimately connected with other interpersonal problems that treatment of the dysfunction alone will be of relatively little value in mending the overall fabric of the relationship. It is therefore of primary importance to be aware of the interrelatedness of the various areas of the couple's relationship and to define the connection between the sexual dysfunction and other aspects of marital disharmony.

The relationship of the discord to the dysfunction seems to be the crucial factor in determining the initial therapeutic focus in the treatment of couples with both significant marital disharmony and sexual difficulties. In evaluating the qualitative nature of the discord and the temporal relation of the dysfunction to the discord, I found that couples could be divided into three descriptive categories, reflecting the extent to which the discord causes or results from sexual dysfunction (1).

1. Sexual dysfunction produces secondary marital discord. Sex therapy is generally the treatment of choice in this situation, especially when the dysfunction existed before the relationship between the spouses.
2. Marital discord in other areas impairs sexual

functioning. While conjoint marital therapy may be the treatment of choice, sex therapy may be indicated if the couple's positive feelings and desire to improve their marriage outweigh their negative feelings and the impairment in their relationship as is the case in the majority of such couples. A trial of sex therapy with these couples may result in rapid relief of symptoms and consequent increase in the self-esteem of both partners, creating a more beneficial milieu for dealing with other pressing marital problems.

3. Severe marital discord, usually with basic hostility, precludes the possibility of good sexual functioning. Immediate sex therapy would be contraindicated in this situation, since the couple's hostility would not allow them to attain the level of cooperation necessary for the rapid treatment of their dysfunction.

While extreme hostility augurs poorly for sex therapy, it is not a deterrent to marital therapy provided there is a genuine desire on the part of both partners to improve their relationship. After or during this improvement, more direct sex therapy may be initiated.

The couples model developed in marital therapy and applied by Masters and Johnson (2, 3) to sex therapy has been fed back into the mainstream of psychiatry by Kaplan's delineation of sexual dysfunctions as psychosomatic disorders (4). Kaplan elaborated a multicausal theory of the sexual dysfunctions, separating immediate forces in the couple's interactions that contribute to malfunctioning from etiological factors based on earlier experiences that predispose the individual to be dysfunctional. Sexual dysfunctions are not necessarily caused by deep intrapsychic problems that reflect disturbance in gender identity or unconscious conflicts. Most symptoms have multiple determinants. Often the sexual malfunction results from the couple's specific interaction, which may then serve as the overdetermining factor to produce symptoms (immediate cause) in an individual made susceptible by remote causes. The person without a partner often cannot be treated as effectively for a sexual dysfunction because the therapeutic leverage of interactional factors can be used only when an involved partner participates in the treatment program (5).

The recognition that the immediate cause occurs within a susceptibility diathesis due to remote causes within the person has important theoretical and clinical implications. It provides the theoretical rationale for combining task-oriented therapy with other treatment methods based on the therapist's knowledge of the remote etiological factors. The remote causes may be bypassed but not ignored by the therapist.

The terms used to describe intrapsychic pathology often carry questionable value judgments as, for example, in a situation in which a wife's transactions with her spouse appear to "cause" his impotence; the woman is often labeled as castrating, while the man may be described as weak, dependent, or passive. These adjectives are grave indictments of both partners' intrapsychic structure. However, impotence may also be conceptualized as resulting from the quality and content of an interaction at a particular time. No negative character traits need be involved; the man may not be weak, dependent, or passive in his general conduct, and the wife may not have a need to control or humiliate her husband. We may then expect that the impotence would respond to forms of treatment that will modify the couple's interactions without necessitating long-term therapeutic efforts to change their individual intrapsychic structures. This approach is quite different from that which might be undertaken when the therapist is skilled only in individual insight and conflict resolution modalities.

RELATIONSHIP BETWEEN MARITAL AND SEX THERAPY

The task-oriented, symptom-removal approach in marital therapy has been given much impetus by our having witnessed its effective use in sex therapy. For example, the use of erotic tasks in sex therapy has encouraged us to use tasks in marital therapy. I am currently applying this approach within the systems treatment model concept of the marital pair.

Treatment is facilitated when both marital and sex therapy are readily available to couples, regardless of their chief complaint, as is the case at the Jewish Family Service Clinic. Professionals competent in both marital and sex therapy increasingly appreciate that we cannot always focus simply on brief sex therapy and must be more flexible about length and type of treatment. There must often be a significant amount of time allotted to marital therapy within the context of the sex therapy program, particularly if the interaction between the partners is not consistent with the prerequisites for sex therapy. When sex therapy has been initiated, the therapist must be cautious of the tendency to regard the upsurge of conflicts in other areas of the marriage merely as indicators of resistances to treatment of the sexual dysfunction. The emergence of such marital conflicts may indeed signal resistance, but it may also be a valid indication for referral or for increased emphasis on marital therapy.

I do not think of sex and marital therapy as two isolated specialties that view the couple from different perspectives. Both try to examine and affect the same system of two persons. Hence, the sex therapy specialist must also be a well-trained marital therapist who is able to bring interactional and intrapsychic as well as sexual problems into focus. Conversely, the specialist in marital therapy must feel comfortable and competent in dealing with his patients' sexual problems. The marital therapist's attainment of some competency in sex therapy (and vice versa) need not preclude referral to experienced specialists when he knows he is getting beyond his expertise or into areas that he is not comfortable with or interested in.

In my first published opinion on this issue (1) I stated definitely that the therapist who is qualified to use both marital and sex therapy should keep the two

sharply separated. With additional experience, I have become less rigid. The interplay between immediate and remote causes often requires a frequent shift of therapeutic emphasis. However, both the therapist and the couple should know when the focus is on sex therapy and when some remote factor dealing with hostility or trust has to be explored before one can risk changing their behavior.

The important principle is that a couple's sexual functioning is only one aspect of their total dyadic system. It is readily influenced by and in turn readily influences other functions and processes. The therapist can feel free to move between the special techniques of each modality while appreciating that he is always working with numerous interrelated functions. Failure in one area may deeply affect other functions.

The presenting problem of many couples is such that rapid treatment of sexual dysfunction seems to be called for; however, more thorough examination may indicate that the problems are interactional rather than specifically sexual. The sexual dysfunction may be the sign that points to the interactional "dyscrasia." For example, the dysfunction of many couples can be best described as an infrequent desire to initiate or pursue sexual relations, a syndrome I refer to as sexual ennui. I prefer this term to "lack of libido" or "low sex drive" because it implies a psychological rather than a physical etiology or a mystical crisis of libidinal energy. When such a couple has sex, both partners may experience it as pleasurable and there may be no evidence of dysfunction. I find that treating this syndrome usually requires marital therapy in conjunction with various erotic and nonerotic tasks to be carried out at home. Treatment varies, of course, in accordance with the etiological factor(s). Often there must be several sessions focusing on the more remote aspects of the marital problem in order to achieve behavioral and attitudinal changes.

Some sex therapy techniques are very helpful in marital therapy and can be used in conjunction with more traditional techniques deriving from other schools of psychotherapy. For example, sensate focus with no genital stimulation (6) can be prescribed by the marital therapist as a way of highlighting and helping the couple work through hostilities, poor communication, fear of emotional closeness, and difficulty in verbalizing desires, and as a concrete demonstration of the importance of quid pro quo maneuvers. The procedure of taking turns giving and receiving pleasure taps deep emotional reactions in both partners. This task can be used as an evaluative test to determine several interactional and intrapsychic factors, thus helping the therapist to arrive at the most urgent and fruitful priorities for therapeutic intervention.

CASE REPORTS

I will present a few case reports to illustrate my contention that sex therapy cannot and should not be isolated from other areas of the marital system. When a sexual dysfunction is the major cause of marital discord, it may be best to focus treatment on the dysfunction—but this cannot be done in isolation from the marital interactions that have also contributed to the problem. When marital disharmony produces sex dysfunction, it is generally best to resolve some of the interactional problems first. When sexual dysfunction and marital disharmony have become a circular or reverberating system, the therapist might begin with sex therapy in an attempt to break the cycle, but he must be prepared to move freely between the two forms of treatment or to bring in another specialist if necessary.

Case 1. A woman of 30 and a man of 31 had originally been referred to me for sex therapy after the wife told her internist that, although she was orgasmic with her husband, she no longer desired sex. There was no apparent cause. I took a complete sexual history, as well as an appropriate history of their relationship. The woman was helped to appreciate her fury at her husband for what she perceived as insensitivity to her emotional needs. A short course of marital therapy, including sensate focus and other giving and receiving reciprocal tasks that were designed to increase communication and sensitivity, led both partners to an understanding of her anger and his evocative behavior, which were then modified by treatment.

The way was thus prepared for working through the immediate determinants of the woman's lack of sexual desire, which were primarily interactional. However, the wife's contribution to the couple's malfunction was also rooted in intrapsychic fears of rejection and feelings of inadequacy that she had experienced since childhood. The man's input was less specific and more reactive. Their overall relationship improved, and frequent pleasurable sex returned.

Case 2. A young couple was referred for marital therapy to the Jewish Family Service by a private psychiatrist. The caseworker who saw the couple originally felt that the wife was extremely hostile and near decompensation. A staff psychiatrist then interviewed the couple in consultation. Although the psychiatrist felt that the woman was not schizophrenic, he believed that at that time marital therapy would be too anxiety provoking for her and recommended individual treatment for the husband and wife. The husband's therapist elicited a history of marked sexual dysfunction. The wife had always been anorgasmic by any means and the husband had a history suggestive of premature ejaculation. It was also discovered that their sexual relationship was further limited by the wife's recurrent vaginal infections. A consultation with a gynecologist was arranged, and her therapist saw that she completed an adequate course of medical treatment. In the past, she had stopped taking medication prematurely and her infections then worsened. Her willingness to eradicate this physical barrier to coitus augured well for her readiness to work on her anorgasmia.

When a conference was held with all the concerned therapists, the patients also participated in the decision-making process. It was decided that individual treatment would be discontinued within a few sessions and the wife's therapist would see the couple in sex therapy, emphasizing the overall relationship problems, particularly those in the areas of trust and closeness. The wife rapidly became orgasmic by self-masturbation and by her husband's masturbation of her. His

premature ejaculation had been situational and cleared as he felt welcomed as a sex partner and respected as a person by his wife. Formal sex therapy was then stopped and the emphasis was changed, with the couple's agreement, to a new contract for time-limited marital therapy.

If this couple had continued with individual therapy instead of changing to a flexible multifaceted therapeutic approach, they would probably have proceeded to a divorce. Both partners might then have been left feeling scarred and inadequate. The case illustrates the advantages of a multimodality approach to the treatment of the total relationship.

Case 3. Two married professionals (a woman aged 26 and a man aged 30) came to therapy with a chief complaint that they were growing apart. The husband stated that he was subject to depressions that caused him to withdraw; the wife reacted to his withdrawal initially with anger and later with resignation and hopelessness. They were compatible in many areas, believed they loved one another, and wanted to make the marriage work.

When the sexual history was taken, the partners were both very definite that sex was fine mechanically; they were both invariably orgasmic during coitus. However, sex was not emotionally satisfying to the wife, and for several months prior to their seeking professional help she had allowed sex only sporadically. She had had sex with other men before marriage and with two men when she and her husband separated for a few months, and she had found that sex could be emotionally as well as physically satisfying for her. She sensed a distance between herself and her husband when they were having coitus, particularly immediately after orgasm. He would turn away and withdraw, while she wanted to snuggle and talk. "That's the time I can be most open," she said, "and he just isn't there." The husband confirmed this, describing an intolerable desire to get away after they had intercourse. He sensed her vulnerability but felt controlled and threatened by her desire for closeness.

The man's mother and father had had separate bedrooms from his first memory until he was 10 years old. During these years he slept with his mother. She was seductive toward him, which he found frightening. As an adult, he disliked and distrusted his mother and was ambivalent toward his father. His parents had a distant relationship in all aspects.

The wife had slept in the same room with her father from age 3 to age 8. Although he was seductive toward her, she recalls no actual sexual acting out. Unlike her husband's experience with his mother, she was close to her father.

Although they were both aware of these facts in each other's lives, neither had ever put these unusual "coincidences" together. The wife apparently yearned for the closeness she had or wished for with her father, and sex held no fears, anxieties, or inhibitions for her. The husband, although he did not suffer inhibition of sexual performance, experienced terrible anxiety at the prospect of emotional closeness, which was clearly related to his experience with his mother. Such a couple might provide a field day for our psychoanalytic concepts of the etiology of sexual inadequacy. Although there was no impairment of sexual functions they were as distressed about their sexual performance as other couples with actual dysfunction.

This situation required a therapist versed in marital and sex therapy and psychoanalytic psychotherapy. The therapist saw the couple conjointly to help them openly discuss their feelings regarding conflictual aspects of their sexual and emotional contracts. The marriage contract approach (7) uncovered many hitherto unconscious conflicting contractual terms. The source of the difficulty was found to reside largely within the man's intrapsychic dynamics (remote causes), which caused him to precipitate transactions with his wife (immediate causes) that were terribly destructive. She played the game with him. He had to see her as he had seen his threatening seductive mother. He feared being drawn into emotional closeness with his wife and reacted to this anxiety by manifesting hostile depression and withdrawal. The woman reacted in a way that was consistent with her vulnerability and reinforced his fears. Without major changes in their basic adaptations, the prognosis for their marriage was poor. Individual therapy for the man was recommended as the first step in an attempt to interrupt their circular negative interactions. Marital therapy had been helpful diagnostically but left us at a therapeutic impasse.

The husband sent me a note recently thanking me for the referral. He liked his therapist. He said things were quiet and he and his wife were more optimistic. Outcome awaits further developments.

Although sex was mechanically good for this couple, it was affectively inadequate and dissatisfying. This is a syndrome we are seeing with increasing frequency. Inappropriate feelings (or lack of feelings) during or after sex may be due to immediate causes within the couple's relationship (as they were for the wife) or to remote and intrapsychic determinants (as they were for the husband).

CONCLUSIONS

The sexual components of a couple's relationship cannot be separated from their total relationship, either in the reality of their daily life or in treatment. Sex therapy has had and will continue to have a profound effect on marital therapy. The effectiveness of the new sex therapy has substantiated the value of the systems approach and of a multifaceted, goal-directed, task-oriented form of marital therapy. This approach involves the fullest range of the therapist's skills in dealing with those factors that interfere with the patients' collaboration in their own change. Symptom removal and tasks mobilize resistance; an eclectic approach to treatment allows us to make use of a panoply of new and old treatment methods and techniques to deal with these deterrents to change.

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Profiles of Couples Seeking Sex Therapy and Marital Therapy

BY ELLEN FRANK, M.A., CAROL ANDERSON, M.S.W., AND DAVID J. KUPFER, M.D.

Assessment of 29 couples seeking marital therapy and 25 seeking sexual therapy at the same institution permitted the delineation of two distinct profiles. Although the two groups were similar in the degree of their sexual and marital difficulties and in demographic characteristics, the relationships of the sex therapy couples were generally characterized by satisfaction and affection, whereas those of the marital therapy couples were often antagonistic. In addition, the sex therapy couples tended to be less conservative and more thoughtful in their approach to life and their problems.

FOR MANY YEARS marital therapy was the sole province of social workers or marital counselors, who concentrated on the overt interactional difficulties being experienced by the couple. The marriage itself was the focus of the therapy, and the sexual aspects of the relationship were rarely, if ever, discussed. However, the recent explosion of scientific knowledge about human sexual experience, coinciding with the women's movement and the new freedom granted women to acknowledge their own sexuality, has focused considerable attention on this aspect of human interaction (1, 2).

At present there are thousands of clinics and individual therapists throughout the country offering what is commonly called "sex therapy." Symptoms of sexual dysfunction that were once considered manifestations of deep-seated psychological problems amenable only to long-term individual psychotherapy are now considered not only eminently treatable, but treatable almost exclusively within the context of short-term intervention involving both members of the affected couple (3). Perhaps because of their divergent origins, marital therapy and sexual dysfunction therapy continue to be thought of as different forms of treatment that attack quite different problems. Yet often the two seem to be inseparable aspects of a complex interplay of action and reaction.

The Western Psychiatric Institute and Clinic at the University of Pittsburgh, which provides both marital therapy and sexual dysfunction therapy in separate specialty clinics, offers a unique opportunity to study couples coming for one form of treatment or the other. This report is a preliminary attempt to examine the similarities and the differences between those who seek marital therapy and those who seek treatment for sexual dysfunction.

Revised version of a paper presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

The authors are with the Department of Psychiatry and the Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, 3811 O'Hara St., Pittsburgh, Pa. 15261, where Ms. Frank is Research Assistant in Psychiatry, Ms. Anderson is Associate Professor of Clinical Psychiatry and Director, Family Therapy Clinic, and Dr. Kupfer is Professor of Psychiatry and Director of Research.

METHOD

In order to study the characteristics of the couples coming for these two modes of therapy, we examined the initial marital evaluation form (KDS-15) given to all couples seeking marital therapy in the Family Therapy Unit and to all couples seeking treatment for sexual dysfunction in the Sexual Behavior Center over a 6-month period. Couples were given the questionnaires at their first appointment and instructed to complete them without consulting one another and return them at their next appointment. KDS-15 is an extensive self-report questionnaire that covers many aspects of a marriage, including individual development and parental family situation, psychosocial history, courtship, current marital relationship, current living situation, premarital and marital sexual activity, the impact of children on the marriage, work and social activities, the use of leisure time, medical history, and attitudes about role behavior in the marriage.

The entire sample of 108 patients was divided by

type of therapy, and overall differences between the two groups were examined. Data on men and women, divided by the type of therapy they were receiving, were also examined. Nonparametric techniques (chi-square tests) were used in this investigation.

RESULTS

Both groups were demographically quite similar, with the modal patient being 31½ years old, married for 7 years, either Catholic or Protestant, white, with a college degree, and 3.4 children. Although an analysis of the husband's type of employment and salary revealed no significant differences between the two groups, it was noted that a larger percentage of wives in the sexual therapy group were working (60% versus 32%, difference not significant).

An analysis of the data on the 29 couples seeking marital therapy and the 25 couples seeking sex therapy revealed that the overall frequency of sexual difficulties was quite similar in the two groups (see table 1). Many of the women who were seeking marital therapy were bothered by inability to reach orgasm, as well as difficulty getting excited and difficulty maintaining excitement. Nearly two-thirds of these women were unable to relax prior to intercourse, and almost half were generally disinterested in sexual activity. Women who were seeking sex therapy had a somewhat higher frequency of individual sexual difficulties than the women in the marital group, although none of the differences reached statistical significance.

The husbands of the women who were seeking marital therapy also tended to be bothered by sexual disinterest. Some men reported difficulty getting and/or maintaining an erection, and more than one-third reported premature ejaculation. The inability to relax and differing sexual habits or practices also constituted problems for a considerable number of these men. Although the men who were seeking sex therapy had more problems with premature ejaculation and were generally less disinterested in sex, there were no significant differences between the two groups.

Just as the couples seeking marital therapy were beset with numerous sexual difficulties, the couples seeking sex therapy seemed to be experiencing considerable marital discord. Outstanding among the interpersonal difficulties experienced by the sex therapy couples were decreased interest in talking to one another, feeling that their spouse did not understand them, frequent arguments, and feeling that their spouse did not fill their emotional needs (see table 2). Most of these couples reported that they were experiencing difficulty discussing problems.

A comparison of the marital complaints of the two groups indicated that marital therapy patients more often complained that their spouse did not understand them, complained about frequent arguments, financial disagreements, and the division of responsibilities within the marriage. A larger proportion of these couples were concerned about their different social needs or desires (see table 2).

More of the sex therapy patients felt that their marriage had exceeded their expectations for compatibility and independence, while more of the marital therapy patients found that their marriage had failed to meet their expectations for social contact (see table 3). Over half of the sex therapy couples rated their marriage as "happy" or "very happy," compared with only about one-fourth of the couples in marital therapy. Not unexpectedly, most of the couples in sex therapy rated their sex life as less satisfying than the rest of their marriage; however, in spite of the high frequency of sexual difficulties among the marital therapy couples, over

TABLE 1
Frequency of Sexual Difficulties in Couples Presenting for Sex Therapy and Marital Therapy, in Percents

Sexual Difficulty	Sex Therapy	Marital Therapy
Women		
Difficulty in reaching orgasm	75	58
Inability to have an orgasm	76	57
Reaching orgasm too quickly	0	10
Difficulty getting excited	80	57
Difficulty maintaining excitement	71	52
Inability to relax	72	63
Disinterest	68	46
Men		
Difficulty getting an erection	13	19
Difficulty maintaining an erection	36	27
Ejaculating too quickly	60	38
Difficulty in ejaculating	8	17
Inability to relax	25	28
Different sexual habits or practices	16	31
Disinterest	24	34

TABLE 2
Interpersonal Difficulties of Couples Presenting for Sex Therapy and Marital Therapy, in Percents

Difficulty	Sex Therapy	Marital Therapy	Significance
Less interested in talking to each other	22	28	n.s.
Difficulty discussing problems	52	69	n.s.
Spouse does not understand them	24	45	$p < .05$
Spouse does not fill emotional needs	46	50	n.s.
Frequent arguments	30	62	$p < .001$
Sexual dissatisfaction	80	60	$p < .05$
Infidelity	8	9	n.s.
Boredom	16	22	n.s.
Too little time spent together	30	28	n.s.
Financial disagreements	12	38	$p < .01$
Disagreement about division of responsibilities	14	38	$p < .01$
Different social needs or desires	16	35	$p < .05$

TABLE 3
Differences Between Couples Presenting for Sex Therapy and Marital Therapy, in Percents

Item	Sex Therapy	Marital Therapy	Significance
Satisfaction			
Marriage is "happy" or "very happy"	56	26	$p < .01$
Sex life is more satisfying than rest of marriage	2	26	$p < .01$
Sex life is less satisfying than rest of marriage	70	33	$p < .001$
Expectations			
Marriage has exceeded expectations for compatibility	20	4	$p < .01$
Marriage has failed to meet expectations for social contact	26	51	$p < .05$
Marriage has exceeded expectations for independence	33	7	$p < .01$
Marriage has exceeded expectations for fun	26	6	$p < .05$
Marriage has exceeded expectations for arguments	42	67	$p < .05$
Patterns of disagreement			
Threaten violence	0	18	$p < .01$
Fight	18	42	$p < .05$
Threaten to leave	11	42	$p < .01$
Talk it over calmly	72	46	$p < .01$
Leave briefly	17	42	$p < .05$
Leave for a day or more	3	22	$p < .05$

one-fourth of them rated their sex life as more satisfying than the rest of their marriage.

DISCUSSION

Our preliminary results lead us to a number of tentative conclusions. It is clear that the two groups did not separate in what would have been the expected fashion, with marital therapy couples experiencing a significantly higher frequency of marital complaints and sex therapy couples experiencing many more sexual complaints. However, questions concerning 1) their expectations about the marriage, 2) specific complaints about the marriage, 3) how they handled disagreements, and 4) various ratings of their overall satisfaction with their sex life and marriage did provide sufficient data to delineate differences between the two groups. Since our goal was to develop a profile of the couples that seek each of these forms of treatment, a closer scrutiny of the results in these four areas may best establish profiles of "personality style" within the relationships being studied.

Although the two groups appeared to be very similar in terms of their demographic characteristics, the personality profile derived suggests major differences between them. The couples were the same age, came from the same ethnic and religious backgrounds, and had approximately the same education. Yet an analysis of the problems they were experiencing, their modes of disagreement, their expectations about marriage, and the extent to which those expectations had been fulfilled indicates two groups that separate along what look like social-class lines.

While the sex therapy couples projected the image of the cerebral, upper-middle-class intellectual, the marital therapy couples projected a blue-collar "Archie Bunker" image. The sex therapy couples seemed to be more comfortable with one another, less conservative in their outlook, and more thoughtful in their approach to their life and their problems. In general, the sex therapy patients were happy with their spouse and involved in basically affectionate relationships, whereas the relationships of the marital therapy couples were characterized by antagonism. Perhaps this dichotomy can be characterized as one between couples who are able to disagree in socially acceptable ways and couples who are not. While the sex therapy couples allowed each other a good deal of independence and privacy, the marital therapy couples seem to be involved in more dependent relationships and did not or could not allow one another privacy. It also appears that a dichotomy existed between couples who had enough money to meet their needs (sex therapy) and couples who did not (marital therapy).

At first we thought that these differences, which seem to be blatantly class-related, could perhaps be explained on the basis of occupation, since neither the husband's earnings nor the level of education had provided significant differences between the two groups. However, the two groups contained similar numbers of professionals and academics at one end of the scale, as well as blue-collar workers at the other end. It may be that personality variables account for the differences, with the preliminary evidence suggesting that the sex therapy patients were clearly less impulsive and more mature than the marital therapy patients. While the latter threatened violence, threatened to leave, or actually left when they and their spouse disagreed, the sex therapy patients tried to talk things over calmly.

Sexual dysfunction therapy at the Western Psychiatric Institute and Clinic is conducted on a slightly modified version of the Masters and Johnson model, requiring two therapists working in conference with both the couple and one another, as well as comprehensive physical examinations and extensive laboratory studies. Marital therapy may involve any of a number of different approaches depending upon the needs of the particular couple, but it rarely requires more than one therapist. Marital therapy lasts an average of 8 weeks and sex therapy an average of 10 weeks. There is a vast difference in the cost of the two types of treatment; sex therapy costs an average of $1,000, compared to about $160 for marital therapy.

This difference may provide interesting leads about the nature of the relationships between the partners in the two groups. Sex therapy represents a considerable investment that can only be interpreted as evidence of the couples' high degree of commitment to their mar-

riage. Thus the decision to come for sex therapy most probably represents a joint decision rather than a unilateral one. In contrast to the marital therapy couples, who had a relatively negative view of their relationship by the time they presented for treatment, the sex therapy couples may have sensed that they had "something worth saving," an idea supported by the fact that they rated their marriages in general as happy. Thus it is not necessarily inconsistent that a number of the marital therapy couples rated their sex life as more satisfying than the rest of their marriage, since this may be a reflection of how bad the remaining aspects of the marriage were.

This study represents the first step in an attempt to assess who should receive marital therapy and who should receive sex therapy. The study asks simply, "Who comes to each type of therapy?" and, in spite of many similarities in the types of complaints the couples were experiencing, it manages to delineate two distinct couple profiles. The next step, of course, is to evaluate treatment outcome in each of the two groups. Only then can we begin to make judgments about whether these couples came to the right clinic for treatment or whether another form of treatment might have been more appropriate.

The present study does, however, have clear-cut implications for the training of both marital and sexual dysfunction therapists. It is obvious from our results that any therapist who is assessing a couple with marital difficulties, but is unable or unwilling to confront the issue of sexual compatibilities, may be performing only half the required task. Equally obvious is the fact that any therapist in sexual counseling must have more than a minimal ability to deal with interpersonal conflicts within the marriage.

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Sex Therapy and Psychophysiological Research

RAUL C. SCHIAVI, M.D.

The author discusses two areas of psychophysiological research that may be relevant to the diagnosis and treatment of sexual dysfunction: investigations of sexual arousal in men and studies of nocturnal penile tumescence. There has been no convincing evidence to date of a predictable, generalizable autonomic response pattern to sexual arousal, but neuroendocrine research in this area has shown promise. A close temporal relationship has been found between REM sleep and penile tumescence in normal men–deviations from this pattern may be related to sexual dysfunction. The author suggests that further research is necessary in these areas of potential diagnostic and prognostic significance.

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Dr. Schiavi is Associate Professor of Psychiatry, Mount Sinai School of Medicine, 11 East 100th St., New York, N.Y. 10029.

UNTIL FAIRLY RECENTLY, our lack of information on human sexual functioning was abysmal. Long after the results of the monumental work of Kinsey and associates on the epidemiology and social aspects of human sexual behavior had been reported, biological research in the United States on human sexual functioning was extremely limited. Masters and Johnson's pioneering investigations have contributed substantially to the current trend of considering human sexual behavior a proper area of biomedical research. This attitudinal change has been illustrated by the increasing number of publications on the biological aspects of human sexuality during the past 10 years in medical and behavioral science journals. There have been more articles published on the biology of human sexual function since 1966 than in all previous years combined.

Recent methodological advances in the measurement of male and female sexual responses have paralleled the establishment of sex therapy programs in institutional settings. Current investigations of psychophysiological aspects of human sexual arousal and

behavior have implications for the understanding of human sexual dysfunctions. Masters and Johnson (1) noted that their philosophy of rapid treatment for sexual dysfunctions evolved from the integration of knowledge on sexual physiology with methods designed to enhance communication between the couple. They have emphasized the need for thorough anatomical, physiological, and metabolic information on sexual function as a basis for the treatment of sexual disorders.

Masters and Johnson's physiological investigations have focused primarily on the correlates of sexual response at the peripheral organ level, with relatively little emphasis on its neural regulation. Their work has provided considerable information on changes in external and internal reproductive organs as well as extragenital physiological responses during the sexual response cycle. They also obtained data on cardiorespiratory reactions such as hyperventilation, tachycardia, and elevation in diastolic and systolic blood pressure during sexual activity. However, sexual arousal and sexual activity may be distinct conditions; there is currently no evidence to substantiate the assumption that the autonomic changes occurring during the early stages of sexual arousal differ only in intensity from the responses associated with overt sexual behavior. We know relatively little about the autonomic and neuroendocrine patterns associated with sexual arousal. Psychophysiological studies of sexual arousal may contribute to our understanding not only of sexual dysfunctions but also of low sexual drive in the face of unimpaired sexual performance.

In this paper, I will briefly discuss two areas of psychophysiological research that are of potential relevance to the diagnosis and treatment of sexual disorders. These areas are 1) the investigation of sexual arousal in men, and 2) studies of nocturnal penile tumescence.

RESEARCH ON SEXUAL AROUSAL

Assessment of sexual arousal is usually based on behavior and verbal reports. However, physiological changes associated with arousal may occur in the absence of conscious recognition. Physiological measures provide the advantages of objectivity, continuous monitoring, and quantitative evaluation. Unfortunately, a review of the studies using various measurements commonly employed in psychophysiological research (electrodermal measures, heart rate, blood pressure, respiration variables, temperature changes, and pupillary responses) revealed that most of these variables are not very sensitive measures of mild to moderate sexual arousal (2). Although there are marked increases in heart and breathing rate during coitus, these measurements do not seem to be sensitive indicators of mild arousal in the absence of sexual activity. Of all the cardiovascular measures evaluated, blood pressure is the only one that has shown a graded response relative to the intensity of sexual stimulation.

In this type of research, it is important to establish that the physiological reactions measured are specific to sexual arousal and do not reflect a response to novelty or to emotions evoked by the experimental situation. Several investigators (3, 4), using concurrent measurement of two or more physiological variables, have attempted to identify a characteristic pattern of autonomic responses that would distinguish sexual arousal from other emotional states such as anxiety or anger. As had previously been the case with other emotions, there has to date been no convincing evidence of a predictable autonomic response pattern to sexual excitation without significant individual differences.

Neuroendocrine Studies

Are neuroendocrine changes more specific and reliable indicators of sexual arousal? Levels of gonadal hormones are promising variables in the assessment of sexual arousal and function. There are data suggesting that testosterone levels may distinguish between two types of psychogenic impotence that have different developmental and behavioral characteristics (5). The significance of this finding is uncertain, however, because of our limited knowledge of the relationship between endocrine levels of testosterone and sexual arousal and behavior in men. This interrelationship has been the focus of considerable attention for many years, but only recently has the development of sensitive methods of hormonal determination made it possible to begin systematically studying relationships between psychological function and gonadal hormones. The interaction could be manifested in two ways: endogenous variations in circulating androgens may influence sexual arousal and activity and, in turn, sexual behavior may modify gonadal secretion. Clear evidence has been found in several laboratories (6–8), including our own (9), of marked minute-to-minute fluctuations of plasma testosterone superimposed on diurnal variations, with maximum levels usually occurring during the early morning. In addition, recent findings (10) suggest the existence of cyclic plasma testosterone variations, with periods ranging from 8 to 30 days. At present, there is no evidence of a correlation between endogenous fluctuations of plasma testosterone and sexual behavior in humans. There is evidence, however, that sexual arousal and activity can increase the circulating levels of testosterone (11, 12), although other researchers have not found this to be the case (13). Because chronic psychological stress has been found to inhibit testosterone secretion in men (14), it is important to take this factor into account in any experimental procedure designed to elucidate the relationship between sexual arousal and testosterone levels.

Penile Erection and Arousal

Increasing attention is being paid to the measurement of penile erection as an indicator of sexual arous-

al. Masters and Johnson indicated that penile tumescence is an early physiological response to sexual arousal in the male that sometimes precedes subjective recognition of arousal. Several devices have been developed to monitor changes in penile tumescence for experimental or diagnostic and therapeutic purposes (15, 16). One reliable method uses a thin elastic silicone tube filled with mercury and sealed at both ends to form a loop. This loop is placed around the shaft of the penis; changes in tumescence produce variations in electrical resistance, which are measured as the tube stretches during erection. Using this method, Bancroft and Mathews (17) investigated the relationship between penile erection and peripheral indicators of autonomic arousal. Erectile, electrodermal, and cardiovascular responses of normal males to sexual and neutral stimuli were recorded in a standardized procedure. Penile erections discriminated between sexual and neutral stimuli, whereas the other physiological parameters, including skin conductance, heart rate, and forearm blood flow, failed to do so. In addition, the correlation between erection and peripheral autonomic measures varied between individuals. Some subjects showed positive correlations between penile tumescence and at least one of the autonomic responses, while other subjects demonstrated no correlation or significant negative correlations.

While we await replication of these findings, it is tempting to speculate with Bancroft and Mathews (17) that erection and autonomic arousal, although usually associated, are independent responses. These two measures may or may not vary together depending upon the subject's appraisal of the sexual and nonsexual components of the situation. The possibility that individuals show specific patterns of autonomic response to sexual stimulation should also be considered. There may be characteristic differences across subjects, with some consistently showing a direct relationship between increased autonomic arousal and penile erection and others demonstrating an inverse relationship.

The existence of response specificity and particularly of a negative association between erection and autonomic arousal may provide a basis for the differential effect of an arousal state such as anxiety on an individual's sexual performance. (Fear of sexual performance is an important immediate causal determinant of erectile dysfunction in some men [1].) An individual who demonstrates a negative relationship between penile erection and autonomic activation may require an optimal low level of arousal for the development of tumescence. This individual may have an idiosyncratic vulnerability to react with erectile dysfunction in the presence of fear or anxiety.

In view of the relevance of these hypotheses to an understanding of the psychophysiology of impotence and the processes involved in the resolution of the dysfunction, it is of interest that there are, to my knowledge, no studies of men with erectile dysfunctions that have assessed the pattern of autonomic arousal and penile tumescence in response to erotic stimulation. This lack of information contrasts with the number of psychophysiological studies that used measures of penile erection to assess sexual preference in male homosexuals (18) and to monitor changes in sexual drive and orientation during their treatment (19).

STUDIES OF NOCTURNAL PENILE TUMESCENCE

Another approach to the study of psychophysiological processes related to sexual function and dysfunction is consideration of the physiological changes in genital structures and gonadal hormones during sleep. Fisher and associates (20) and Karacan and associates (21) were among the first investigators to systematically study the association between nocturnal penile tumescence and REM or dreaming sleep in normal men. They found that episodes of erection began and ended in close temporal relationship to the onset and completion of REM sleep.

The degree and frequency of erection are at their height in early adolescence, when an average of 5 to 7 episodes of tumescence occur during the night and an average of 32% of total sleep time is spent in erection. Although there is a progressive decline in frequency of nocturnal erections from adolescence to old age, the proportion of erection time in relation to total sleep time remains relatively constant between the ages of 20 and 69, ranging from 20% to 30%. Although there is a close temporal correlation between REM sleep and penile erection, this is by no means absolute. Furthermore, there is evidence that each process may be controlled by independent mechanisms that usually coincide temporally but may be unassociated (22–24). The functional and adaptive value of nocturnal cycles of penile erection remains unclear. It has been suggested that erections may be another aspect of the genital physiological activation associated with REM sleep (20).

Jovanovic (25) has indicated that there are definite parallels between disorders of potency, disturbances in the pattern of nocturnal erection, and sleep disorders. He is the first investigator to study the relationship between impotence and variations in the REM-erection cycle. He found that, compared to normal men of the same age group, men with psychogenic impotence had fewer and less intense erections and demonstrated a lack of temporal synchrony between the erection periods and REM phases, with markedly irregular cycles of tumescence. Jovanovic concluded that nocturnal REM erection objectively mirrors the state of a man's sexuality and may be useful in the differential diagnosis of sexual disorders. In view of the universality and regularity of the phenomenon of nocturnal penile tumescence, it is remarkable that there is so little information available on the patterns of nocturnal erection in men with either psychogenic or organic impotence.

In a recent paper on variations of nocturnal penile tumescence as a function of age, Karacan and asso-

ciates (26) summarized their findings on the relationship between nocturnal REM erections and sexual impotence. Based on their evaluation of 55 men with impotence of various etiologies, they concluded that the determination of nocturnal penile tumescence discriminates between organic and psychogenic impotence.

Nocturnal Penile Tumescence and Hormonal Levels

In collaboration with Dr. Charles Fisher, we have initiated in the human sexuality program at Mount Sinai School of Medicine a systematic investigation of patterns of penile tumescence and fluctuations in plasma luteinizing hormone (LH) and testosterone during nocturnal sleep in normal men and men suffering from psychogenic and organic impotence. The first stage of this program has involved gathering normative data on stages of sleep and penile tumescence in relation to levels of LH and testosterone in heterosexual men free from sexual dysfunctions. Frequent serial blood sampling during nocturnal sleep revealed abrupt fluctuations in both hormones, with no significant relationship to REM sleep. There was, however, a consistent tendency for mean testosterone levels to be higher during REM sleep with tumescence than during REM sleep without tumescence. The possibility of an interaction between REM sleep and penile tumescence relative to testosterone secretion deserves further consideration. It has been suggested (27) that changes in testicular blood flow as a result of sympathetic nervous system stimulation may be a determinant of short-term changes in plasma testosterone levels. It may be that the autonomic nervous system activation during REM-associated erections results in rapid increases in testosterone release. Further studies using frequent sampling in a greater number of subjects are needed to clarify the significance of nocturnal penile tumescence in relation to fluctuations in plasma testosterone.

CONCLUSIONS

Sleep studies offer a valuable approach to the investigation of hormonal secretion in terms of central nervous system activity. Concurrent assessment of gonadal hormonal levels and penile tumescence during sleep may provide a basis for needed psychophysiological research in sexual dysfunctions such as impotence. There have been few psychophysiological and endocrine studies of erectile disorders, and no sequential investigation of pituitary-gonadal activity in these patients has yet been reported. In addition to increasing our overall knowledge of the subject, the assessment of endocrine and erectile changes in men with psychogenic and organic impotence may have diagnostic as well as prognostic significance. Penile recording techniques may provide a means to differentiate patients who have similar symptoms but may have distinct etiological or developmental characteristics and may be differentially responsive to treatment. Karacan and associates' observations (26), as well as our own (28), seem to support this possibility.

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For further information write Jeanne Spurlock, M.D., Office of Minority Affairs, American Psychiatric Association, 1700 18th Street, N.W., Washington, D.C. 20009, or call (202) 232-7878.

Relationship Between Dosage and Response to Lithium Prophylaxis in Recurrent Depression

BY ROBERT F. PRIEN, PH.D., AND EUGENE M. CAFFEY, JR., M.D.

The authors analyzed the results of a multihospital collaborative study on the effectiveness of lithium prophylaxis in recurrent depression in terms of dosage and found that serum lithium levels between 0.5 and 0.7 mEq/liter and doses below 1000 mg/day were relatively ineffective in preventing recurrences. Serum lithium levels between 0.8 and 1.0 mEq/liter and doses above 1000 mg/day were associated with a relatively low failure rate. The authors discuss the relevance of these findings to current prescription guidelines for lithium carbonate.

THE MULTIHOSPITAL COOPERATIVE study on lithium carbonate therapy sponsored by the Veterans Administration and the National Institute of Mental Health between 1968 and 1972 (1, 2) helped establish lithium as an effective prophylactic treatment in recurrent depression; lithium was shown to be effective in preventing both depressive and manic episodes in unipolar and bipolar illness. The study also demonstrated that lithium is not a panacea: almost half of the lithium-treated patients relapsed over a two-year period. Attempts to relate treatment outcome to background variables such as age and psychiatric history were unsuccessful (3). However, subsequent analyses revealed that lithium dosage was significantly related to treatment outcome.

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When this work was done Dr. Prien was Assistant Chief, Central Neuropsychiatric Research Laboratory, Veterans Administration Hospital, Perry Point, Md.; he is now Research Psychologist, Psychopharmacology Research Branch, National Institute of Mental Health, 5600 Fishers Lane, Rockville, Md., 20852. Dr. Caffey is Deputy Assistant Chief Medical Director for Professional Services and Clinical Support Services, Department of Medicine and Surgery, Veterans Administration, Washington, D.C.

DOSAGE GUIDELINES

There appear to be no consistent guidelines regarding prophylactic lithium dosage. The lithium carbonate package insert (4) recommends serum lithium levels of 0.6–1.2 mEq/liter[1]; these levels are usually achieved with doses of 900–1200 mg/day. Some clinicians recommend more aggressive schedules. Schou (5) contends that full prophylactic effect in most patients is obtained only when serum lithium concentration is maintained at 0.8–1.2 mEq/liter. Other clinicians are more conservative and recommend levels between 0.4 and 0.8 mEq/liter (6–8). The American Medical Association Department of Drugs specifically cautions against maintenance levels exceeding 0.8 mEq/liter (9). A few clinicians argue for even lower levels of 0.3–0.6 mEq/liter (10–11).

It should be pointed out that the aforementioned recommendations are not substantiated by published dosage studies and appear to be based mainly on unsystematic clinical observation. There is, however, one point of agreement. All clinicians recommend that maintenance levels not exceed 1.5 mEq/liter because of the risk of toxicity and consider levels below 0.3 mEq/liter ineffective.

METHOD

The study was conducted at 12 Veterans Administration and 6 public or private hospitals. Patients were admitted to the study following hospitalization for acute depression. A complete description of background characteristics is provided elsewhere (1). Briefly, the sample consisted of 76 women and 46 men. All were

[1]Lithium is readily absorbed from the bloodstream into body fluids. As a result, serum lithium level (measured in milliequivalents of lithium per liter) serves as a reliable indicator of lithium concentration throughout the body, and is used to monitor dosage.

under 60 years of age and had experienced at least one affective episode requiring hospitalization during the preceding 2 years and two episodes requiring hospitalization during the preceding 5 years. Their diagnoses were manic-depressive psychosis, depressed type, and recurrent endogenous depression.

Following remission of the acute episode, each patient was stabilized on maintenance doses of lithium carbonate or imipramine. Lithium was administered in 250-mg capsules. Serum lithium levels were stabilized between 0.5 and 1.2 mEq/liter. At discharge, patients were randomly assigned to lithium, imipramine, or placebo. Patients assigned to active medication continued at the established maintenance level. Patients assigned to placebo had identical looking capsules substituted for active medication.

The lithium-treated group included 45 patients. Patients returned to the hospital every 4 weeks to be evaluated, obtain medication, and have their serum lithium levels monitored. Serum lithium levels were obtained from blood drawn by venipuncture before the first morning dose (8–12 hours after the previous dose). Serum lithium concentration was determined by emission flame photometry or atomic absorption spectroscopy.

Each patient was to receive his assigned treatment for 2 years. The treatment physician knew what medication the patient was taking, but the clinical raters operated under blind conditions.

Treatment outcome was evaluated primarily in terms of occurrence of affective episodes, defined as depressive or manic attacks requiring hospitalization or supplementary drugs (i.e., psychopharmacological agents other than the patient's assigned treatment). Patients were also evaluated on a variety of instruments including the Global Affective Scale (3) completed by a psychiatrist at each visit; depressive and manic symptoms were rated not present, mild, moderate, or severe.

In examining the relationship between treatment outcome and dosage, we excluded patients who were unable to remain out of the hospital or off supplementary medication for at least 6 consecutive months. This was to ensure that all patients had an adequate period of stabilization on lithium therapy. Six patients were excluded for this reason. We also excluded 7 patients who had no episodes but were unable to complete the study because of intercurrent illness or failure to keep appointments. The remaining 32 patients were classified as either treatment failures (N=12) or treatment successes (N=20). A patient was considered a treatment failure if he had an episode and a treatment success if he had no episode. The median time of treatment failure was the 17th month of the study. Eleven of the treatment failures resulted in hospitalization; the 12th required continuous supplementary medication.

Treatment failures and treatment successes were compared in terms of lithium dosage. The main purpose of this comparison was to determine whether treatment failure could be explained by insufficient dosage. Another goal was to determine whether dosage guidelines could be established for the treatment of recurrent depression. Three sets of doses were compared: 1) doses administered to treatment failures 6 months before their episode were compared with doses administered to treatment successes at the end of the first year; 2) the same comparison was conducted with doses administered 3 months before treatment failure; and 3) randomly selected doses for treatment failures were compared with randomly selected doses for treatment successes. Findings from the three sets of comparisons did not differ significantly; only the results from the first set are presented here.

RESULTS

Treatment successes received a significantly higher lithium dosage than treatment failures (means at 1275 and 979 mg/day; $p<.05$, two-tailed t test). Treatment successes also had higher serum lithium levels (means of 0.80 and 0.63 mEq/liter), although the difference between groups was not statistically significant.

Table 1 shows the proportion of failures and successes associated with various dosage levels. Fifty-three percent of the patients receiving daily doses of 1000 mg or less were failures compared with only 20% of the patients receiving doses exceeding 1000 mg ($p<.05$, Fisher's exact probability test). Similar results were obtained in comparisons of serum lithium levels (table 2). Fifty-five percent of the patients with levels of 0.7 mEq/liter or less were failures compared with only 14% of patients with higher levels ($p<.05$, Fisher's exact probability test). There was no major difference in treatment outcome between patients with levels of 0.8–1.0 mEq/liter and those with levels exceeding 1.0 mEq/liter.

There was a predictable positive relationship between dosage of lithium carbonate and serum lithium level. Patients with serum lithium levels exceeding 0.7 mEq/liter received an average daily dose of 1393 mg, (range, 1000–2250 mg). The average daily dose for patients with levels of 0.7 mEq/liter or less was 972 mg, (range, 750–1500 mg).

The Global Affective Scale showed no major difference in symptoms between failures and successes at

TABLE 1
Treatment Outcome by Dosage

Dosage (mg/day)	Treatment Failures		Treatment Successes	
	N	Percent	N	Percent
750	5	71	2	29
1000	4	40	6	60
1250	2	25	6	75
1500 and more	1	14	6	86

TABLE 2
Treatment Outcome by Serum Lithium Level

Serum Level (mEq/liter)	Treatment Failures		Treatment Successes	
	N	Percent	N	Percent
0.7 or less	10	55	8	45
0.8–1.0	1	11	8	89
Over 1.0	1	20	4	80

the time the analyzed doses were administered: 25% of the failures and 15% of the successes had mild symptoms. Moderate to severe symptoms were not apparent in the majority of treatment failures until the month preceding the episode. Dosage increases at this time obviously proved unsuccessful in keeping the patient out of the hospital.

Side effects were not a major problem, although 4 patients had moderate to severe gastrointestinal reactions early in the study that necessitated a decrease in dosage. A fifth patient experienced moderately severe hand tremors, and a sixth felt weak and lethargic. Three of these patients could not achieve serum lithium levels above 0.6 mEq/liter because of recurring side reactions.

The failure rate for 13 placebo patients who were able to remain episode free for 6 consecutive months was 69%. This rate is not significantly different from the 55% failure rate for lithium-treated patients with blood levels of 0.7 mEq/liter or less.

DISCUSSION

It should be stressed that this was not a dose-response study, i.e., patients were not assigned to various dose levels. The clinician selected what he considered to be the best dosage regimen for each patient. The only requirement was that serum lithium concentration be maintained at 0.5–1.2 mEq/liter.

Serum lithium levels between 0.5 and 0.7 mEq/liter were associated with a relatively high incidence of failure. This supports Schou's contention that levels below 0.8 mEq/liter are often inadequate to ensure full prophylactic effect (5). However, two factors should be considered before one regards these levels as ineffective. First, these levels may be necessary for elderly and debilitated individuals who cannot tolerate higher doses; it has been reported that some elderly patients respond well to levels as low as 0.3–0.4 mEq/liter (12). Second, the study sample represented a very high-risk population. All patients had been hospitalized at least once during the two years preceding the study; 60% had been hospitalized two or more times. It is possible that a less severely ill population would require lower doses.

The results suggest that levels exceeding 1.0 mEq/liter produce no advantage over levels of 0.8–1.0 mEq/liter. The latter levels are usually achieved with doses of 1250–1500 mg/day. As mentioned previously, not all patients can tolerate these levels. If a unipolar patient (i.e., a patient with no history of manic episodes) is unable to tolerate a level of 0.8 mEq/liter, a switch to maintenance doses of imipramine should be considered. Previously reported findings from this study (1) indicate that imipramine, in doses of 75–125 mg/day, is just as effective as lithium with the unipolar patient. Imipramine should be used cautiously with bipolar patients (i.e., patients with a history of mania) because of the risk of manic attacks, and its use in place of lithium is not recommended.

It is interesting to note that most of the study relapses occurred relatively quickly, often before the clinician had an opportunity to adjust dosage. Thus, the clinician who purposely maintains the patient on low levels with the expectation that dosage can be increased at the first sign of the emerging episode may not be providing the patient with adequate protection.

Finally, the three major sources for drug prescription information in the United States differ in the recommended serum lithium levels for long-term control. The lithium carbonate package insert (4) recommends levels of 0.6–1.2 mEq/liter, the American Medical Association Department on Drugs (9) recommends 0.5–0.8 mEq/liter, and the American Society of Hospital Pharmacists (13), levels of 0.5–1.0 mEq/liter. These varying recommendations, coupled with the findings from this study, emphasize the need for well-controlled studies on lithium dosage that use not only serum lithium levels but other promising monitors such as red blood cell lithium concentration (14). Until such studies are conducted, a significant number of patients may receive either ineffectively low or unnecessarily high doses.

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Pavor Nocturnus: A Complication of Single Daily Tricyclic or Neuroleptic Dosage

BY ABRAHAM FLEMENBAUM, M.D.

The author tested the hypothesis that a single bedtime dosage schedule of tricyclic or neuroleptic medication produces increased frequency of night terrors by administering a questionnaire to 30 medical patients who were not receiving such medications and 100 psychiatric patients on either multiple- or single-dosage schedules. Psychiatric patients on multiple-dosage schedules reported no more frightening dreams than the medical patients, whereas almost three-fourths of those receiving single bedtime doses had frightening dreams, a significant difference from the medical sample. This preliminary report is presented to call attention to the possible undesirable effects of a single dose schedule.

BECAUSE OF PATIENT noncompliance in taking multiple (divided) doses of tricyclic and/or neuroleptic medication and because of the lengthy half-life of these drugs, several authorities have suggested giving all of the medication in one dose at bedtime. In fact, one of the most frequently prescribed tricyclics has been marketed on the basis of a single daily (evening) dose. The advantages of this method include sedation to facilitate sleep, decreased side effects (which occur mostly while the patient is asleep), and better patient compliance.

In our clinic, this method has been beneficial in a number of cases, with little or no loss of efficacy. (There does, however, tend to be a lower steady state with longer periods of time between doses [1].) One problem has been that a number of patients have complained of terrifying nightmares, some of which have interrupted sleep, with symptoms of physiological activation accompanied by temporary confusion and disorganization. This description is fairly characteristic of night terrors (pavor nocturnus), with the exception of the amnesia frequently seen in those cases (2). Also, in six of our patients the pavor nocturnus disappeared when the dosage schedule was changed from once a day at bedtime to divided doses three or four times daily. These cases led me to test the hypothesis that single bedtime doses of these medications produce an increased frequency of night terrors.

The author is Associate Professor and Research Psychiatrist, Department of Psychiatry, Texas Tech University School of Medicine, P.O. Box 4269, Lubbock, Tex. 79409.

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METHOD

A simple one-page questionnaire was developed to obtain information on 1) the presence or absence of dreams and 2) whether dreams were accompanied by confusion, disorientation, and signs of physiological arousal.

The subjects were hospitalized psychiatric patients selected at random and given the questionnaire by nurses, who were instructed to give the questionnaire only to patients who were not senile, confused, or undergoing ECT. For control and comparison, the questionnaire was also given to 30 randomly selected medical outpatients at the Department of Family Practice, Texas Tech University School of Medicine, who were not taking any medication with central nervous system action. (Two of these patients were later found to be taking CNS-active medications such as hypnotics, and their responses have not been included in the data analysis.)

Inpatients at two private facilities (St. Mary of the Plains Hospital and Methodist Hospital) that serve as clinical bases for the Department of Psychiatry, Texas

Tech University School of Medicine, were given the same questionnaire at different times. A sample of 100 psychiatric patients was obtained in this manner. I discarded questionnaires of patients who had been in the hospital less than six days and had not reached steady state blood levels of medication and those who were undergoing treatment for alcohol, barbiturate, and/or other drug abuse. (Incidentally, all of the latter patients reported nightmares with physiological activation.) The final sample of psychiatric patients consisted of 76 individuals.

RESULTS

Approximately 50% of the medical patients (N=15) reported dreams of some kind, but only 4 (14.3% reported frightening dreams and only 2 of these 4 reported psychophysiological arousal.

Of the psychiatric patients, 61.8% (N=47) reported dreams of some kind. Although more psychiatric than medical patients reported dreams, this difference was not statistically significant. However, the psychiatric patients did report significantly more *frightening* dreams (N=21, or 28%) than the medical patients ($\chi^2=14.64$, df=1, $p<.001$, Yates' correction applied). This difference is even more marked when we compare only those psychiatric patients who received psychotropic medication (tricyclic antidepressants and/or antipsychotics) in a single daily dose at bedtime to the medical patients ($\chi^2=17.97$, df=1, $p<.001$, for the entire group given single bedtime doses, and $\chi^2=26.9$, df=1, $p<.001$, for those given tricyclics only) (see table 1). There was no significant difference in the frequency of frightening dreams when psychiatric patients given divided doses were compared with medical patients.

This indicates a clear tendency for more patients given single daily bedtime doses to report frightening dreams. Also, although some patients who received their medication in divided doses did report frightening dreams, most of the reports that included confusion and physiological arousal came from patients given single doses at bedtime: 8 of the 11 patients on tricyclic medication at bedtime who reported frightening dreams noted these effects. Furthermore, one of the single-dose patients reported a clear-cut episode of somnambulism for the first time in her life. Somnambulism, like night terrors, is a phenomenon of stage 4 (slow-wave) sleep (3).

To summarize, psychiatric patients receiving multiple doses of medications reported no more frightening dreams than did medical patients. On the other hand, almost three-fourths of the patients receiving single bedtime doses of psychotropic medication had frightening dreams. This suggests that we may be unintentionally inflicting additional miseries on patients who come to us for relief of their own suffering.

DISCUSSION

All psychotropic drugs seem to produce changes on EEG sleep records. Barbiturates and monoamine oxidase inhibitors produce virtually total REM suppression (4). Phenothiazines and butyrophenones tend to synchronize the EEG tracing (5). Given in small doses (e.g., 50 mg of imipramine orally at bedtime), tricyclics in general seem to produce a moderate reduction of REM sleep (6). However, administration of single doses of drugs like diazepam (10 mg) at bedtime seems to decrease stage 4 sleep (7). Stage 4 is the stage during which some of the most terrifying nightmares (pavor nocturnus) occur (2, 3, 8): for this reason, Kahn and associates (7) concluded that diazepam could reduce or abolish nightmares by suppressing stage 4 sleep. This group of investigators had already observed that chlorpromazine did not reduce the number of nightmares—in fact, two of the most severe nightmares occurred while the patients were on chlorpromazine.

The stage 4 nightmare is rare, with an incidence of only 3% to 4% in school children and less in adults. Its etiology is obscure. Although some nightmares are REM (and not stage 3 or 4) anxiety dreams of varying intensity, the most frightening dreams, causing arousal from sleep and a subjective experience of confusion and fear, usually occur during stages 3 and 4, with concomitant physiological arousal (2, 8).

It is interesting that Jus and associates (9) found that chronic schizophrenics receiving medication in a single bedtime dose had a significantly higher per-

TABLE 1
Frequency of Frightening Dreams for 76 Psychiatric Patients, by Type of Medication and Dosage Schedule

Dosage Schedule	Antidepressants		Antipsychotics		Total	
	Number	Percent	Number	Percent	Number	Percent
Single bedtime dose (N=19)						
Frightening dreams	11	78.6	3	60.0	14	73.7
No frightening dreams	3	21.4	2	40.0	5	26.3
Divided doses (N=57)						
Frightening dreams	3	11.5	4	12.9	7	10.5
No frightening dreams	23	88.5	27	87.1	50	89.5

centage of non-REM (stages 3 and 4, or slow-wave) sleep than did those given divided doses. This dosage also increased the mean percentage of REM sleep, whereas the mean percentages of stages 1 and 2 were significantly decreased.

I am not aware of any similar study on tricyclic antidepressants. However, because of the chemical similarity of neuroleptics and tricyclics, I suspect the results would be the same. Also, in studying enuresis, Kales and associates (6) described a slight decrease of REM and an increase of stage 4 with imipramine, changes that rebounded on withdrawal. Not surprisingly, lithium produces a similar significant increase in slow-wave sleep, although it decreases REM sleep (10).

The results I have reported in this preliminary study should be considered tentative, even though the statistical analysis showed marked effects. The hospitalized psychiatric subjects were a cross-section of patients present in private hospitals during four different weeks and were not preselected or followed in double-blind fashion. Most had other drugs prescribed for comfort and medical conditions. These other drugs included medications that affect the central nervous system (e.g., sleeping medications), although most of the patients received flurazepam HCl, which reportedly interferes little with sleep EEG patterns. Some patients received cardiovascular antihypertensives, antibiotics, and other medications. Furthermore, most patients on tricyclic antidepressants had either a tricyclic antidepressant alone or in combination with a neuroleptic (e.g., perphenazine-amitriptyline HCl) or other combinations with small doses of chlorpromazine, thioridazine, etc.

This preliminary report is presented to call attention to the potential ill effects of single-dose antidepressant or neuroleptic medication and to pinpoint this as a potential area of research. It is worthy of note that, as previously mentioned, single bedtime doses may lower the steady state blood levels of the medication. Another fact to remember is that a recent report (11) summarized previous findings supporting the idea that even though the fatal dose is approximately 30 mg/kg, single doses above 200 mg of imipramine or the equivalent come close to levels of toxicity for certain people (5 mg/kg). Reports indicate that heart blocks, convulsions, and death may occur at times in the absence of signs of adverse effects sufficient to prompt dose reduction. The Food and Drug Administration has refused to approve investigational use of imipramine in children in cases where the total daily dose is too close to the apparent danger point of 5 mg/kg. In our experience, it is not rare to find patients who require doses of 200 mg a day or more for the drug to be effective, and the above information clearly indicates potential dangers if this amount is given in a single dose.

I am currently involved in planning a blind prospective study to evaluate the effects of single-dose versus divided-dose schedules in relation to night terrors, but I feel that there is already sufficient reason to consider each case individually and to weigh the possible advantages of single doses against the disadvantages.

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Sexual Attitudes of Hospital Personnel: A Model for Continuing Education

BY DAVID J. WITHERSTY, M.D.

The author describes a program developed to help nurses and other health care professionals deal with the problem of sexual acting out by patients who have been hospitalized for a long time. The program involves group behavior rehearsal sessions with role playing. These sessions resulted in specific suggestions for coping with the problem and have been incorporated into the hospital's continuing education program.

THE DEMAND that the postgraduate health professional keep up in his or her area is increasing. The consumer demands it, professional organizations and institutions are beginning to require it, and, with the advent of such innovations as the medical audit and Professional Standards Review Organizations (PSROs), the government has indicated a strong interest in the maintenance of postgraduate standards. Many of the traditional ways of imparting knowledge, such as lectures, discussion groups, and panels, have been supplemented by such devices as closed-circuit videotape programs, programmed learning texts, and self-assessment evaluations. These educational methods all deal primarily with factual knowledge. How does one continue to grow professionally in areas in which factual knowledge is not needed but an opportunity to review, evaluate, and perhaps change attitudinal stances that might be interfering with one's professional functions is needed?

One approach to this difficult problem, which is used in the department of behavioral medicine and psychiatry at the West Virginia University Medical Center, is a type of role playing. In this paper I will present one of the problems that prompted the department to use this approach and discuss some of the advantages and disadvantages we have found in using this method.

THE PROBLEM

The staff of the behavioral medicine and psychiatry department were asked to assist in the presentation of an in-service training program for the nurses on the staff of West Virginia University Hospital. Nurses from several different units had indicated to their supervisors that they felt uncomfortable and inadequate when confronted with sexual acting out, particularly by patients who had been hospitalized for a prolonged period of time. They asked for a program to help them learn to deal with the problem.

To evaluate and better understand the problem, members of the department held several open discussions with the nurses who had requested help. We also sent out questionnaires to all of the nurses to find out if there were any who were concerned about the problem but embarrassed or reluctant to ask for help directly.

Through the open discussions and the answers to the anonymous questionnaires we found that a lack of factual knowledge was not an issue among the nurses, with the exception of questions related to sexual functioning of the paraplegic patient. Most of the participating nurses stated that they had sufficient knowledge of sexual physiology to discuss a problem with a patient but that their discomfort hampered them from doing so.

The nurses stated that they felt more uncomfortable when a patient of the opposite sex confronted them with a sexual problem. They also said they felt less comfortable about taking the initiative in discussing a sexual problem with a patient. However, some nurses stated that they were concerned that a refusal to take the initiative in discussing a patient's sexual frustrations or needs might result in sexual acting out in the form of masturbation or propositioning the nurses.

The nurses were asked, How comfortable would you be approaching and talking with another professional (doctor, nurse, etc.) about a sexual need or problem of a patient? Their answers were contradictory. In open discussion many of the nurses admitted that they would find it difficult to approach another professional about a patient's sexual problem. However, the answers to the anonymous questionnaires indicated a high degree of comfort in approaching and discussing a patient's sexual problem with another professional (according to a scale on which 5=100% comfort, the average response was 4.06).

Thus it appeared that the in-service training program should be geared toward alleviating the feelings of discomfort that interfered with the nurses' ability to impart factual information. With the agreement of the

Dr. Witherstry is Assistant Professor and Director, Inpatient Service, Department of Behavioral Medicine and Psychiatry, West Virginia University Medical Center, Morgantown, W.Va. 26506.

staff nurses we decided to use behavior rehearsal techniques to reach this goal.

BEHAVIOR REHEARSAL

Although there was expressed agreement in favor of role-playing techniques, it was obvious that there was some concern about trying new behaviors in front of a group of fellow professionals. We therefore decided to conduct several demonstrations with groups of between 25 and 40 people in order to alleviate anxiety. The nursing staff and faculty of our department initiated the sessions. Later, the opportunity to practice discussing sexual matters with another professional acting as a patient was offered on a voluntary basis to smaller groups of from 6 to 8 people. This ensured an opportunity for participation.

The demonstrations were well attended, not only by the staff nurses but by medical and nursing students, social workers, nurses' aides, and orderlies. A male psychology intern in our department, who was not well known throughout the rest of the hospital, took the role of a patient who had been hospitalized for many weeks. He was instructed to make verbal sexual advances toward the various female professionals, who were instructed to practice how they might cope with the situation. We pointed out to the participants that there was no one ideal way of dealing with this particular clinical situation, that mistakes and ineffective attempts were expected, and that we hoped everyone could learn from the mistakes. The only other ground rule was that the staff of the department as well as the spectators could interrupt the participants at any time to offer suggestions for dealing with the situation.

RESULTS

Even in the large-group setting, professionals in the group were willing to volunteer to practice new behaviors. Early in these sessions there was a lot of joking and laughing. The fact that humor used to detoxify a high level of anxiety might have the effect of detracting from the learning experience was openly discussed. The group then decided to try to play it straight.

The participants also discussed the fact that humor was used in a similar fashion on the wards to lessen their anxiety concerning the sexual needs of patients. The potential effect of this use of humor on the patients was discussed, and the danger of these behavioral patterns being viewed by the patient as a rejection became obvious.

As the various participants tried role playing (with suggestions and coaching from the sidelines), a common problem repeated itself with some regularity. Early in the encounter the nurse (or social worker, orderly, etc.) who was talking to the "patient" lost control of the situation when the issue of sex was approached. His or her discomfort was apparent, and we observed a variety of techniques for avoiding the issue—ignoring the reference to sex, changing the subject (often to a concrete clinical issue, such as laboratory test results), or actually leaving the patient's bedside. The psychology intern who played the role of the patient said that he would feel embarrassed or guilty if he were treated this way as a patient; the group as a whole concurred.

After a number of these role-playing situations had been observed, the group developed the following outline of suggested behaviors:

1. The professional should be aware of the first hint that a patient wishes to talk about his or her sexual needs and address the issue promptly.

2. The professional should make it clear to the patient that he or she understands that anyone who has been hospitalized for a prolonged period of time would have sexual needs and frustrations. The group emphasized empathy at this particular stage.

3. The relationship between the professional and the patient should be defined by the professional in a very clear, unambiguous manner. For example, the professional could say, "I can certainly understand your needs and frustrations and as a professional I would like to be able to help you, but in no way will I be willing to act on your suggestions." Some members of the group suggested that at this stage of the conversation it might also be appropriate to acknowledge verbal sexual advances as complimentary. However, they suggested that the professional stress the fact that he or she did not intend to act on the patient's suggestions. The major concern of the group was that the professional be firm in his or her approach and yet not put the patient down.

4. Professionals who find it embarrassing to discuss sexual matters with their patients even after practice sessions should admit their embarrassment to the patient and suggest that they would be willing to find another professional who might be willing to talk to him or her.

The large-group demonstrations were well received. The participants indicated that their anxiety about dealing with this particular clinical situation decreased as they learned and practiced new behaviors for dealing with their own discomfort. We decided to continue with behavior rehearsal sessions, using smaller groups, on the various nursing units. These sessions are now being conducted and are well attended; participation has been maximal. Similar learning experiences are now part of the training program for medical students and physician assistants.

DISCUSSION

Behavior rehearsal techniques are certainly not new. Although originally developed by Lazarus in 1966 as a therapeutic tool, the emphasis of this technique on "affective processes that inhibit or prevent the

implementation of effective social behavior" (1, p. 181) makes it ideal as an educational device:

> In addition to its disinhibiting function, behavior rehearsal also provides an opportunity for patient [or professional] to expand his repertoire of responses through modeling and imitation processes (role reversal) and through his own improvisation. (1, pp. 181–182)

Although it has been our experience that patients receive better care after hospital personnel have participated in the continuing education program, which includes behavior rehearsal sessions, the question has been raised as to what effect these sessions have on the personal lives of the staff members who attend them. Two comments are relevant to this issue.

First, the behavior rehearsal sessions are not designed as self-growth or therapy groups for the participants. The goal of the sessions is to evaluate staff attitudes in terms of effectiveness of patient care. Personal attitudes may lead to professional behaviors that interfere with adequate patient care. The sessions afford opportunities to explore and practice more effective behaviors. The quality of patient care improves when health professionals gain control over their emotions.

Second, although the staff's personal growth per se is not the goal of the program, one would expect that the sessions would result in their having less anxiety and better emotional control, thus allowing them to make more rational personal decisions concerning their own value systems.

In a time when the demand for continuing postgraduate health education is great, professionals must continue to be open to any technique that might help them maintain a high level of professional competency, either in the area of facts or in their emotional and attitudinal stances.

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Is Dying Being Worked to Death?

BY RONALD JAY COHEN, PH.D.

In recent years there has been a dramatic increase in the number of books and articles on death and dying. The ambivalence of some writers regarding the value of this proliferation of papers led the author to review and evaluate this literature; he found seven overlapping categories of death-related literature that were of both theoretical and practical value. He briefly explores possible reasons for the rise in this subject's popularity.

THE TREND IN THE popularity of death as an area of scientific inquiry could be described as having gone from "warmed-over" to "hot." In an editorial in the *Journal of the American Medical Association* questioning the utility and value of the burgeoning literature on death, Vaisrub noted that "we are now witnessing an extraordinary influx of essays, editorials, even books and specialized journals, on the subject of death and dying" (1, p. 1909). Others have observed that "death is one of the current fads" (2, p. 190) and that "it is incongruous to envisage death as a bandwagon but this is what it seems likely to become" (3, p. 468). A coup de grace is dealt to the would-be writer in this area by those who argue that everything that could possibly be said on the topic has been said already.

> It is much too easy to write about death. There is no need for statistical evidence and for lengthy references. And no one can claim to have more direct information on the subject from high-up. Perhaps because it is so easy to write about dying, so much is written about it without saying anything new. (1, p. 1910)

Dr. Cohen is Senior Psychologist, Department of Psychiatry, New York University—Bellevue Medical Center, Rm. C-410, Bellevue Psychiatric Hospital, 1st Avenue and 30th Street, New York, N.Y. 10016.

EVALUATION OF THE LITERATURE

Rather than indicting en masse the literature in this multifaceted area, it would seem reasonable to independently evaluate the contributions of the various approaches. Broadly speaking, seven overlapping categories can be distinguished. One is concerned with the care and treatment of the patient in extremis. Publications included here range from research reports and case histories (4–10) to "do-it-yourself" primers (11). Closely related is the category in which phenomena associated with death and dying are examined from a developmental/psychopathological perspective. Representative publications in this category deal with the death-related anxiety of various populations (12–18), the distinction between affective fear of and cognitive preoccupation with death (19), and the elucidation of bereavement processes (20–23), with special reference to the question of normal versus pathological grief (24).

Demographic and epidemiological approaches comprise a third category of papers. Amid the seeming plethora of statistical studies included here are many insightful and compelling findings such as those linking psychosocial variables with the occurrence of either death or suicidal attempts (25–30). Another classification includes publications that expound the numerous sociocultural aspects of death and dying (31–34). A fifth category is reserved for the discussion of legal, ethical, and philosophical issues. This literature ranges from the presentation of guidelines for research (35) to debate over current and increasingly complex clinical issues (e.g., "active" versus "passive" euthanasia and "Patients' Bill of Rights").

The sixth category is comprised of theses that are concerned only coincidentally with death-related issues. Descriptions of the testing of hypotheses derived from such notions as achievement motivation (36, 37), locus of control (38), and other formulations (39, 40) would be included here. A final category is reserved for those papers whose primary contribution is to supplement discussion in any of the other six areas by providing stimulating reports amenable to scientific inference. Nonevaluative transcripts of aged individuals' introspections (41) and heuristic experimental findings open to interpretation (42) are representative samples.

DISCUSSION

If the media accurately reflect popular concerns, then the wide appeal of Kübler-Ross's *On Death and Dying* (43), the extensive coverage given the Quinlan case, and the box-office success of sundry films (e.g., *Love and Death* and *Death Wish*) may be taken as evidence that death-related concerns are not confined to professional journals. How is this heightened general interest in death-related phenomena to be explained? Some may argue that it is the threat of world war and the specter of nuclear annihilation that have compelled individuals to intellectually confront the inevitable. Others de-emphasize international concerns to underscore intranational ones such as the recent rise in cancer-related deaths, the alarming increase in street crime, and the unprecedented increment of political bombings and other terrorist activities in this country. Theologians may point to the decline of the Church and the subsequent unrest stemming from ambivalence attendant on the relinquishment of sacred notions such as the belief in an afterlife. Humanists may argue that the growing literature on death is indicative of a rebellion against cold scientific inquiry and a reawakening of awareness that the dying person is, first of all, a person. There is no shortage of explanations, and those which are put forth are likely to vary within and between academic discipline as well as medical specialty.

Allowing for discussion of ethical and legal issues raised by medical advances, Vaisrub's question concerning the reason for the "current preoccupation" (1, p. 1909) is a cogent one. But whatever the reason for the apparent Zeitgeist, scholarly attempts to amplify, quantify, and reify phenomena related to death and dying have generally had the effect of richly enhancing psychological theorizing and complementing extant dynamic and behavioral intervention strategies. Even the lay media's treatment of this topic has been of educational and perhaps cathartic value.

Is dying being worked to death? I think not. It seems unfair to demean the proliferation of death-related literature as a "bandwagon" if this literature is useful in disseminating the raw data with which behavioral theories and life-supportive psychotherapies can be constructed and/or evaluated. The literature sampled in the seven categories conceptualized herein is only minutely representative of the many recent *novel* contributions to man's appreciation of his mortal dilemma. Untimely deaths have been defined as those that are premature, unexpected, and calamitous (44). In an era when the psychiatric community will increasingly be called upon to consult with moribund patients and their families, a call for a moratorium on death-related publishing would indeed be "untimely."

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Computerized Tomography in Patients with Tardive Dyskinesia

BY ALAN J. GELENBERG, M.D.

The author reviews the results of computerized tomography performed on eight patients with tardive dyskinesia to determine whether there was permanent structural damage. No significant abnormalities were found. Although it may not be useful in routine work-ups, this new noninvasive technique might be helpful in differential diagnosis, and its increasing sophistication may suggest further research on tardive dyskinesia.

TARDIVE DYSKINESIA is a syndrome of abnormal involuntary movements associated with neuroleptic therapy. Typically, it involves the tongue, mouth, jaws, and face, but it may also include the extremities and trunk (1). While the etiologic role of neuroleptic medication in this disorder continues to be questioned (2, 3), the syndrome is generally attributed to the long-term use of these drugs (1, 4), which is coming under increasing scrutiny (5).

The prolonged and frequently irreversible course of tardive dyskinesia, even after the discontinuance of neuroleptic medication, has suggested that permanent structural damage may underlie the disorder. Neuropathological studies have reported differing results. Phenothiazine administration has been reported to produce neuronal loss in the basal ganglia of rats (6, 7), and one study of human autopsies revealed greater cell degeneration in the substantia nigra of patients with tardive dyskinesia than in controls (8). Other autopsies, however, have found no pathology (9–11).

Autopsies of patients with Huntington's chorea, which resembles tardive dyskinesia (12), reveal degeneration of the caudate nucleus and putamen (13). Similarly, Duvoisin (14) has observed a woman with severe tardive dyskinesia whose pneumoencephalogram revealed ventricular dilatation consistent with caudate atrophy. The atrophy was greater on the side contralateral to her more severe dyskinesia.

Dr. Gelenberg is Co-Chief, Special Studies Clinic, Erich Lindemann Mental Health Center, Instructor in Psychiatry, Harvard Medical School, and Clinical Associate in Psychiatry, Massachusetts General Hospital, Fruit St., Boston, Mass. 02114.

The author wishes to thank Roger Duvoisin, M.D., James A. Schnur, M.D., and Paul F.J. New, M.D., for their suggestions and information, and Marlene Mieske, R.N., and Carolyn Widen, L.P.N., for their assistance with patients.

A new radiological technique, computerized axial tomography (CAT) scan, has provided a noninvasive view of the ventricles and surrounding tissue (15–18), an area formerly visible only via pneumoencephalography. The purpose of this paper is to report on eight patients with tardive dyskinesia who were given CAT scans as part of the clinical work-up.

METHOD

Subjects

Five men and three women with tardive dyskinesia were evaluated. The patients, whose ages ranged from 17 to 56 years, had been exposed to a variety of neuroleptic medications (chlorpromazine, trifluoperazine, perphenazine, fluphenazine, haloperidol, and thiothixene) for periods ranging from 10 months to 16 years. All had psychiatric diagnoses of chronic undifferentiated or paranoid schizophrenia. No patient had a prior history of neurologic disorder, and the only significant family history was the presence of Parkinson's disease in one patient's grandmother.

Diagnosis and ratings were made using the National Institute of Mental Health Abnormal Involuntary Movement Scale (AIMS), which uses a scale of 0–4 to rate facial and oral movements, upper and lower extremity movements, and trunk movements, and also rates global judgments about abnormal movements in terms of overall severity, incapacitation, and patient's awareness.[1] (This instrument will be described in detail in a future publication.)

Global serverity ratings for most patients were mild to moderate (2 to 3 on the 4-point scale). Perioral movements were present in seven patients, upper extremity movements in six, and tongue movements in five; mandibles, lower extremities, trunk, and head were involved less frequently.

Procedure

As part of the clinical diagnostic process, computerized axial tomography was performed using an EMI scanner. Transverse sections were made at a 25-degree angle to Reid's anatomical baseline, with sections at 2.5, 5.0, and 7.5 cm.

[1]Copies of the AIMS may be obtained from the Psychopharmacology Research Branch, National Institute of Mental Health, 5600 Fishers Lane, Rockville, Md. 20852.

RESULTS

Normal scans were reported in seven patients. One patient, a 40-year-old woman diagnosed as having chronic undifferentiated schizophrenia who had been taking phenothiazines for 16 years, showed mild, generalized cortical atrophy, but her lateral ventricles appeared normal and there was no suggestion of caudate atrophy.

DISCUSSION

No caudate atrophy was observed in any of these patients, and the mild, generalized cortical atrophy seen in one patient was not considered clinically significant. These essentially normal scans might be explained in any of several ways.

Limitations of the Technique

Menzer and associates (19) listed Huntington's chorea as a form of dementia diagnosable by CAT scan, but they did not state what degree of atrophy of the caudate nucleus is necessary to be visible on the scan. Paul F.J. New, M.D., of the Massachusetts General Hospital Department of Radiology, has informally estimated that a "moderate" degree of degeneration of the head of the caudate could be seen using the current computer reconstruction in the horizontal plane, but it is impossible at this time to give a more precise quantification. It is therefore not possible to state how small a degree of degeneration in the patients I have described may have escaped detection. A change in computer integration to reconstruct the coronal plane could conceivably result in the detection of degeneration of a lesser degree, and work is currently in progress to test this possibility.

Nature of the Lesion

It is plausible that if there is degeneration in the caudate in tardive dyskinesia, it is of a much finer degree than the gross changes observed in Huntington's chorea and possibly not visible even with a more precise radiological technique. The caudate degeneration Duvoisin (14) observed on pneumoencephalography may have represented an incidental finding or another illness. Alternatively, there may be no morphological lesion underlying the syndrome, but rather a biochemical one, such as neuronal supersensitivity to dopamine (4).

Nature of the Sample

The small number of patients studied raises the question of sampling, but the absence of positive findings in any of these patients suggests that changes that might be found using a larger sample would have a low incidence. The group was heterogeneous, with a wide age range and a large variety of medications. All had dyskinesia that had persisted for at least 4 months, suggesting the truly tardive or persistent form of the disorder, rather than withdrawal dyskinesia (4).

CONCLUSIONS

The technique of computerized tomography is an exciting innovation in the neurosciences. The procedure takes approximately 20 minutes, causes no discomfort to the patient, and provides an in vivo view of structures formerly visible only via the very uncomfortable and potentially hazardous pneumoencephalogram. At the Mayo Clinic, the use of pneumography decreased by 60% after the introduction of the CAT scan (18). On the basis of the data I have presented, the CAT scan in its current program does not seem useful as a part of the routine work-up of patients with tardive dyskinesia. However, it may be valuable when the differential diagnosis of this or other neuropsychiatric syndromes is unclear. The sophistication of the technique is growing, and further investigation into its value in tardive dyskinesia is planned.

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Hypothalamic Tumor Presenting as Anorexia Nervosa

BY GEOFFREY B. HERON, M.D., AND DONALD A. JOHNSTON, M.D.

The authors describe a patient with symptoms of anorexia nervosa who was initially treated on a psychiatric ward but was later found to have a hypothalamic tumor. They recommend repeated neurological and endocrine evaluations for patients with anorexia nervosa who are unresponsive or only partially responsive to psychiatric treatment. Initially negative medical evaluations, obvious psychopathology, and even some response to treatment may all prove insufficient to rule out organic disease.

THIS PAPER describes a patient with an appetite disorder initially diagnosed as anorexia nervosa but later ascribed to a pinealoma affecting the hypothalamus. Despite theoretical and experimental evidence relating hypothalamic function to control of appetite (1), few cases have been reported in which hypothalamic dysfunction first presented behaviorally. Our patient illustrated the difficulty of discriminating between emotional and organic pathology early in the evolution of a syndrome. He had behavior abnormalities without endocrine or neurological deficits. Although this patient's tumor, an ectopic pinealoma, is rare, the presenting symptoms parallel some features of anorexia nervosa and may lead to a misdiagnosis.

CASE REPORT

First Admission

Mr. A was a 24-year-old self-employed single man with persistent weight loss unexplained by repeated medical evaluations. A diagnosis of anorexia nervosa had been made when he entered a state psychiatric hospital for treatment. Weight loss continued there for 3 weeks, and he was transferred to a psychiatric ward in a Veterans Administration hospital with a recommendation for hyperalimentation.

Mr. A had anorexia, nausea, and vomiting after an honorable discharge from the Army 2 years before admission. Nausea and vomiting ceased after 5 months, but his weight gradually fell from 132 pounds to 72 pounds at admission; he was 6 feet tall. He had progressively lost interest in previously enjoyed activities and sexuality.

The patient had not dated in high school, but while in the Army had courted a girl for several months. Although he wanted to marry her, the relationship ended when she refused to leave her native country. The broken engagement upset him, but he felt that after 2 years he had "gotten over it."

Mr. A, the second of four boys, was the only child remaining at home with his parents. His father was a mechanic and his mother a housewife. The mother's obesity was the sole instance of family weight disturbance. The parents confirmed that the patient had always been thin but without marked fluctuations in weight, and that he was a "finicky eater." The mother became increasingly distraught during her son's prolonged weight loss and devoted extraordinary attention to feeding him; she regularly cooked separate menus for husband and son, had stocked a large pantry with excessive quantities of food, and cajoled her son to eat more. She seemed personally injured by his continued wasting. The patient's father tried to remain calm and objective, and he rarely expressed his frustration in the patient's presence. He seemed to feel stuck between his son's bewildering illness and his wife's rather overwhelming anxiety about it.

The patient's early development and history were unremarkable. Although he had been myopic since childhood, his vision had not changed recently. He had occasional headaches, but there were no other neurological symptoms. He had not been hyperactive, nor did he use excessive activity to lose weight.

The initial physical examination revealed little except the striking cachexia. Gross testing of visual fields and funduscopic examinations were within normal limits, as were the patient's hair distribution and the size of his testicles. His lack of concern about his illness was striking, but he did not insist that he was overweight or trying to diet, as patients with anorexia nervosa often do. His affect was rather flat, and he seemed somewhat withdrawn and listless, but he did not appear psychotic or display signs of organic impairment. He stated he would rather go home than to another hospital, but agreed to admission.

In the hospital his evaluation continued in consultation with the medical service. His completed work-up included thyroid function tests, plasma cortisol levels,

The authors are with the University of Colorado Medical Center, 4200 East Ninth Ave., Denver, Colo. 80220, where Dr. Heron is a resident in psychiatry and Dr. Johnston is Assistant Professor of Psychiatry.

urine specific gravities, D-xylose excretion, brain scan, skull X rays, total bowel X rays, and a small bowel biopsy. All tests were within normal limits.

After evaluation Mr. A was started on a behavior modification regimen in which his ward privileges and time for socialization were dependent upon a daily weight gain. He reluctantly agreed to this program, but insisted that he had no control over his weight. He began to sabotage the treatment by visiting outside allowed times, sneaking candy while refusing a high calorie diet, and protesting that the food cooked by the hospital was inedible. Even after direct instructions his mother refused to stop bringing home-baked snacks to him.

After repeated adjustments were made in the treatment program to reward the patient for weight gain and to minimize his unusual eating habits, he slowly began to gain weight. He did not recognize any progress, however, and grew increasingly angry at the staff for imposing what he considered unfair treatment. After gaining 15 pounds in 2 months, he signed out against medical advice with his family's approval.

The psychiatric staff concluded that this family could not tolerate interference with their shared psychopathology, in which conflicted feeding was a prominent feature. Although his weight gain had been limited, the patient left the hospital convinced he could do better at home. Follow-up telephone contact 2 months after discharge showed that he felt better and had continued gaining weight; he then weighed 99 pounds.

Second Admission

Five months following discharge Mr. A returned to the hospital as an emergency medical admission. He was semicomatose, dehydrated, had a serum sodium of 180 mEq/liter, and was almost totally blind. Further studies revealed a full-blown syndrome of pituitary and hypothalamic destruction, with diabetes insipidus, decreased thirst sensation, abnormal thermal regulation, decreased adrenal, thyroid, and gonadal hormones, and optic atrophy. A pneumoencephalogram showed a mass lesion in the area of the hypothalamus, and a subsequent biopsy provided the tissue diagnosis of an ectopic pinealoma. Following these diagnostic studies and careful medical management of hormonal replacement, he began a course of radiation therapy, a treatment to which pinealomas have proven sensitive (2).

DISCUSSION

Anorexia nervosa is a psychiatric syndrome characterized by excessive weight loss and malnutrition resulting from reduced caloric intake, which may be due to irrational self-imposed dieting through willful control of appetite or to the experiential loss of appetite. Although the disorder occurs in men, it is most typically seen in adolescent girls ranging in age from prepuberty to young adulthood (3, 4). Its most typical feature is thought to be a dread of obesity, frequently associated with a distorted body image and the patient's insistence on continued weight control or loss in the face of externally obvious wasting (3, 5). The patient's premorbid personality frequently includes constricted compulsive rigidity and schizoid traits (6). Anorexia nervosa patients usually have an ambivalent relationship with one or both parents; often one parent is overly concerned and solicitous and attempts to coerce or inveigle the patient to eat (4).

Anorexia caused by infections, extracranial malignancies, or other conditions may be ruled out by the usual physical work-up. Special diagnostic attention should be given to the possible presence of tumors affecting the pituitary-hypothalamic axis, which are generally manifested by nonspecific neurological symptoms such as headaches and visual field changes, and by endocrine abnormalities. Ectopic pinealomas are usually seen in adolescent or young adult males, and present with a characteristic triad of diabetes insipidus, pituitary dysfunction, and neurological deficits (7). Puschett and Goldberg (8) suggested that the first two components may appear well before any neurological involvement. The diagnostic indicators that are most helpful are skull films, which reveal the calcified pineal, abnormal urine specific gravities, and decreased levels of serum cortisol and gonadal and thyroid hormones. Pneumoencephalography revealing a hypothalamic mass is usually definitive. Electroencephalograms and brain scans do not contribute to diagnosis (8).

Although Mr. A was a man slightly outside the usual expected age range of anorexia nervosa, he otherwise fit many criteria for diagnosis of this syndrome. For 2 years he had maintained a low weight and suffered progressive anorexia, which by the time of admission was profound. His personality pattern included social isolation, rigidity, aloofness, ambivalence, and a nearly symbiotic relationship with his mother. This relationship between mother and son was highlighted by her attitude and behavior about his eating. His extensive physical work-up revealed normal endocrine function, his pineal was not calcified, and his urine specific gravities were within normal limits. It is interesting that only 5 months prior to the development of diabetes insipidus and panhypopituitarism his endocrine studies were normal. We speculate that his 2-year period of anorexia resulted from the insidious growth of the ectopic pinealoma and was the first symptom of this tumor.

This case makes clear several points relevant to anorexia nervosa. Progressive brain pathology may not be revealed in one examination, and a slow response to treatment should prompt reexamination. It should also be kept in mind that obvious individual or family psychopathology may co-exist with and does not rule out a brain lesion. Finally, although psychiatric treatment may alter organically induced failure to eat, successful psychological intervention does not rule out brain pathology.

LETTERS TO THE EDITOR

Auditory Hallucinations

SIR: Hallucinations observed during the early phase of psychotic episodes are usually auditory, even though man uses vision as his principal means of perception after the fourth postnatal month (1). In order to gain some insight into this phenomenon, the development of auditory and visual perceptions in newborn kittens and human infants under various conditions was studied by the best available method—the behavior of the auditory and visual evoked potentials. According to current concepts the P_3-N_4 wave complexes are closely related to intracerebral elaborations such as cognitive processes and decision making (2). The behaviors of auditory and visual evoked potentials of newborn kittens exposed to various recurrent conditions were compared to those of kittens reared under uneventful conditions, and human infants subject to recurrent crying spells of unidentified etiology (absence of detectable pathological changes) were compared to infants whose lives were uneventful.

Randomly delivered pleasant auditory stimuli resulted in earlier maturation (decreased latency, increased amplitude, and changes in wave shape) of auditory evoked potentials in the kittens. Visual deprivation (taped eyes during the first three postnatal weeks) delayed maturation of the visual P_3-N_4 wave complex, with concurrent earlier maturation of the auditory P_3-N_4 wave complex. Recurrent early exposure to stressful experiences (disturbing noise, hunger, cold, etc.) resulted in a minor but statistically significant delay in maturation of the visual P_3-N_4 wave complex and a statistically significant increase in maturation of the auditory P_3-N_4 wave complex.

The human infants with recurrent crying spells had a statistically significant delay in development of visual evoked potentials compared to controls with comparable IQ and attention span.

These observations suggest that the genetically preprogrammed maturation of sensory evoked potentials may be altered through postnatal experiences, resulting in a tendency toward delayed maturation of visual evoked potentials and a prolonged prevalence of hearing as the means for perception of environmental events. Perception through a sensory modality proceeds by registering incoming stimuli through a peripheral sensory receptor, transmitting this signal to central receptors, comparing it with preexisting engrams of the same sensory modality, evaluation, decision making, and (if possible) execution of the decision. This process requires both functioning peripheral and central anatomical systems and a minimum memory bank of engrams of the same sensory modality.

Morphological maturation of the auditory system is faster in mammals. Also, intrauterine life seems to permit accumulation of auditory engrams but not visual ones. The observations on the development of the P_3-N_4 wave complex of auditory and visual evoked potentials suggest that coping with recurrent early postnatal stressful situations consumes time and energy and works at the expense of lack of accumulation of the minimum visual memory bank necessary for efficient use of visual perception. Prolonged use of auditory perception results in reinforcement and in accumulation of auditory engrams containing unusual elements (e.g., pain due to stresses). Therefore, recall by similar painful (anxiety) experiences in future life of reinforced auditory memory traces is facilitated. This is one of the potential sources of auditory hallucinations.

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CLARA TORDA, PH.D., M.D.
New York, N.Y.

An Expanded Acknowledgment

SIR: I am writing to amplify the acknowledgment given to Ronald S. Poze, M.D., in the footnote to my article, "The Immediate Effects of Chlorpromazine in Newly Admitted Schizophrenic Patients" (October 1975 issue). I feel that my original acknowledgment did not do justice to Dr. Poze's contribution, which consisted of suggesting the idea for the research and participating in the development of the research design.

WILLIAM T. HAMILL, M.D.
Palo Alto, Calif.

Still More on the Semantics of Hysteria

SIR: I would like to respond briefly to Dr. Edward Margetts' letter "The Semantics of Hysteria Continued" (January 1976 issue). I understand his concern about the use of the term "Briquet's disorder"; this concern has been expressed for some time by a number of psychiatrists and for various reasons.

As a member of the St. Louis group in which much of the validating research has been done (begun by Drs. Robins and Guze), I can report that the eponym was chosen with the hope of minimizing controversy. The term "hysteria" has never been precise, referring to myriad symptoms and syndromes. Briquet's disorder, on the other hand, *is* precisely defined. If one is familiar with the literature on the subject, there is little question what is meant. Briquet's disorder is

thought by many of us to be the heart of hysteria, the nuclear disturbance, with a set of surrounding phenomena that are more or less related.

As a member of the APA Task Force on Nomenclature and Statistics, I can comment that we have chosen to speak in a generic sense of hysterical disorders. These disorders include a number of fragmentary clinical states, many of which are poorly understood. Briquet's disorder, identified by the eponym and by a lengthy literature, is central.

There was considerable discussion within the task force about what Briquet's disorder should be called. The matter was decided by vote, with Dr. Spitzer, the chairman, voting against adopting the eponym. Dr. Spitzer's view may have failed to carry because no acceptable alternative to "Briquet's disorder" was found. "Polysomatizing disorder" was suggested, but it was rejected because of its confusing implications.

Admittedly, the history of the term "hysteria" is the history of a problem, although the major disorder has been clearly defined. Dr. Margetts suggests that we all know what hysteria means. That may be true. Each of us knows; unfortunately, few of us agree.

ROBERT A. WOODRUFF, JR., M.D.
St. Louis, Mo.

SIR: I happen to agree with the view that all efforts should be made to find a noneponymic term for the category that is now referred to as Briquet's disorder. I believe that "polysomatizing disorder" conveys the essential notion, namely, that Briquet's disorder involves somatizing, the expression of emotional conflict or problems by medically unexplained physical symptoms. Since the essence of the condition is that somatizing involves multiple organ systems, the prefix "poly" is necessary to avoid confusion with other conditions in which somatizing may be involved.

I was unable to convince the other members of the task force of the wisdom of this view. However, at a forthcoming conference to critically evaluate *DSM-III*, we will put the issue to a group of distinguished nosologists for their assessment. Then, who knows?

ROBERT L. SPITZER, M.D.
New York, N.Y.

CAT Scans in Children with Behavior Disorders

SIR: Computerized axial tomography (CAT scan) is a remarkably versatile and accurate new method of examining intracranial contents. The technique is rapid, entirely noninvasive, and can detect a wide variety of lesions, (neoplasm, hemorrhage, edema, cysts, calcifications, and other abnormalities), often with greater sensitivity than either angiography or pneumoencephalography. Moreover the CAT scan affords an opportunity to evaluate the central nervous system (CNS) in individuals who may have brain disease when there is too little likelihood of positive findings to justify diagnostic procedures that entail any risks. Such a situation preyails in children with learning and behavior problems that are often ascribed to underlying brain damage, although the evidence of CNS impairment is mostly presumptive. The high cost of CAT scans prohibits widespread screening of these children, but the method might have some potential in increasing scientific understanding of these disorders.

As a pilot approach, we selected 4 children with the greatest evidence of CNS impairment from a population of 35 children hospitalized for the treatment of severe emotional disorders. There were 3 boys and 1 girl between the ages of 6 and 12 years. Judgments of "organicity" were based on independent psychiatric, psychological, and EEG assessments. Each child had a history compatible with some kind of CNS damage and at times displayed hyperactivity, aggressive outbursts, and/or autistic behavior. They had significant learning disabilities and tended to show large discrepancies between verbal and performance WISC scores and impairment in measures of visuomotor integration. EEGs were markedly or severely abnormal, with both generalized and focal electrical abnormalities. None of the children had positive neurological findings, although "soft signs" such as awkward gait and poor coordination were present. A control group of 4 children was chosen from the same population as having the least likelihood of CNS pathology because of the relative absence of the previously described characteristics. All children were referred for CAT scans; the radiologist did not know which children were presumably brain damaged and which were not (the brain-damaged group, however, received the procedure first). Results of the scan in 3 of these children were interpreted as entirely within normal limits. Before further studies could be accomplished, the fourth child in the brain-damaged group developed an acute myasthenic crisis that established the neurological diagnosis without need for further investigation. At this point, the study was terminated without scanning the children assumed to have no CNS disorder because of the negative results in the other group as well as the expense of the procedure.

This pilot experience suggests that the CAT scan is unlikely to provide additional evidence about a neuropathological basis for severe learning and behavior problems of childhood.

JOYCE G. SMALL, M.D.
DONALD A. DIAN, M.D.
VICTOR MILSTEIN, PH.D.
TAKUYA SATO, M.D.
Indianapolis, Ind.

Art Is Not Mad

SIR: The *Journal* is to be commended for using as a lead article an English professor's work, "The Madness of Art" by Leon Edel, D. Litt. (October 1975 issue). We have long known that artists have their own road to the unconscious, and we too often have ignored the contributions they make to our understanding of man. The poets know the sense and beauty of man's unconscious. There is a kinship between us, and it is not our mutual interest in what Dr. Edel calls a "tristimania," Dr. Benjamin Rush's term for agitated depression.

In his tribute to Dr. Rush, Dr. Edel makes herculean but procrustean efforts to wed our field to his; as if that were not strained enough, he gives us the bicentennial squeeze play. Dr. Edel gives a respectable interpretation of Dr. Rush's dream, although it is obviously and naturally retrospective. It is a dream of Dr. Rush's impotence. Fine. Freud would go along with that. It is hard to ignore the symbols of the tri-

dent, steeple, ball, and the inability to control the elements. But Dr. Edel goes too far when he says, "It was a dream of revolutionary America . . . or of a Britannia that claimed to rule the waves. . . . The dream was prophetic in its warning against national megalomania. It said that neither man nor nation alters natural forces." Not only is this a squeeze play, but don't we alter natural forces?

The title of the article is another attempt, perhaps unconscious, to join poetry and psychiatry. In a tribute to Dr. Rush, we are given the dangerous impression that art is mad. It isn't. Dr. Edel knows this and I await with anticipation his further insights about poets and their miraculous awareness of life.

MAX SPITAL, M.D.
White Plains, N.Y.

Dr. Edel Replies

SIR: Dr. Spital's letter seems ambivalent and wanting in precision of phrasing. He uses such words as "herculean" and "procrustean" to characterize a paper that simply assumed that the material of the psyche, whether thrown up as symptom or as poetry, can legitimately concern my humanistic discipline as well as his.

That Dr. Spital did not notice my allusion to Lombroso I can only regard as a bit of "selective inattention." My title for the paper was ironic. I was not suggesting that artists are madmen, as should be obvious to anyone who reads the paper carefully—particularly my synopsis of Henry James's "The Middle Years."

I did not attempt to interpret Dr. Rush's dreams. I treated them as poems or fables, which is the only way a humanist can treat dreams, since he is not an analyst and is without license to deal with such material therapeutically. Dr. Rush's fables spoke to me as a symbol of our inability to change history, even when we plan to assassinate presidents of other countries or bid earthquakes to go away. We bring slight modifications by staging wars, but history has a way of determining its own fateful course in spite of wars. Finally, I do not think I "strained" to bring in the bicentenary in a "squeeze play," as Dr. Spital says. Dr. Rush did after all sign the Declaration of Independence.

LEON EDEL, D.LITT.
Honolulu, Hawaii

Reducing Oral Medication with Depot Fluphenazine

SIR: There are several other explanations for the observations reported in "Fluphenazine with Other Antipsychotic Drugs" by Guy Chouinard, M.D., and Rejean Fontaine, M.D. (letter to the editor, July 1975 issue). Drug monitoring combined with vigorous attempts to reduce all other medications in the past 4 years at the Psychiatric Hospital, Royal Park, Victoria, Australia, has been successful (1). Fluphenazine decanoate has fewer side effects and a longer duration of action than fluphenazine enanthate. This partially explains why 95.7% of the authors' patients who were given the latter drug required an antiparkinsonian drug. A survey in February 1975 of 379 patients in our facility revealed that only 30.3% were on these anticholinergic drugs. Previous surveys had revealed a much higher percentage (e.g., 57.4% in 1973). The almost 30% reduction resulted from a determined attempt to cease all oral medication and from reeducation of hospital staff and standardization of the principles involved in managing these complex depot phenothiazines.

Another sample on injections for a mean of two years (2) indicated that 45.1% were still on antiparkinsonian drugs and 4.3% had not been on these drugs at all. We then looked at the time taken to cease administration of antiparkinsonians. The 49.6% of patients for whom these drugs had been discontinued were assessed for mean duration of time spent on antiparkinsonians: in females it was 20.07 months and in males, 15.83 months. Thus we believe there is a difference between oral and intramuscular phenothiazines in terms of time that antiparkinsonian medication is required. Other contributions to the reduction include increasing the interval between injections and reducing the dosage.

The February 1975 survey showed that 24.9% of the patients received oral medication other than an anticholinergic antiparkinsonian (mainly benzodiazepines and tricyclic antidepressants), but only 8.7% received other phenothiazines. The benzodiazepines have a definite role. Nitrazepam is useful as a hypnotic and diazepam is useful for the side effects such as akathisia.

As well as potentiating the anticholinergic side effects of depot fluphenazine, concurrent oral medications can have nontherapeutic drug interactions and lead to other problems such as drug dependency, insomnia (3), and potentiation or worsening of tardive dyskinesia (4). The physician should attempt to reduce all oral medication as soon as possible.

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PETER MARRIOTT, M.A.N.Z.C.P.
Melbourne, Australia

Dr. Chouinard Replies

SIR: The main point is to try to avoid polypharmacy in the treatment of schizophrenic patients. Dr. Marriott is not only reporting that 8.7% of the patients treated with fluphenazine were also receiving other phenothiazines, but that 16.2% were receiving benzodiazepines or tricyclic antidepressants. Tricyclic antidepressants have not been demonstrated to be of therapeutic advantage in combination with neuroleptics (1). As far as benzodiazepines are concerned, double-blind controlled studies have not shown them to help schizophrenic patients. Recently it has been reported that two schizophrenic patients out of six with high levels of anxiety treated with a dose of 150–300 mg/day of chlordiazepoxide were significantly helped (2). Thus Dr. Marriott's suggestion to reduce oral medication with depot fluphenazine does not

seem to apply to his use of benzodiazepines and tricyclic antidepressants, medications for which there is no rationale in the treatment of schizophrenia.

The use of antiparkinsonian drugs in the treatment of drug-induced pseudoparkinsonism is a more complex issue. The difference of incidence in the percentage of patients taking antiparkinsonian drugs could be explained by the doctor's tolerance or nonrecognition of extrapyramidal side effects. It would be interesting to know the percentage of patients presenting bradykinesia or akinesia when treated with fluphenazine alone. Unfortunately, bradykinesia is very often confused with psychomotor retardation in schizophrenic patients and considered as a residual schizophrenic defect (3).

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GUY CHOUINARD, M.D.
Montreal, Que., Canada

Another Group Heard From

SIR: I read "History and Analysis of a Leaderless Group of Professional Therapists" by Winslow Hunt, M.D., and Amnon Issacharoff, M.D. (November 1975 issue) with interest since I have participated in and reported on a leaderless group (1–3). Our group seemed satisfactory to the participants, and it certainly satisfied me. The report by Drs. Hunt and Issacharoff describes a less satisfactory experience, which leads me to ask the following questions:

1. Was the group too large? Insight groups usually have five to eight members. Many group therapists think that therapy is difficult with more than eight patients.
2. What was the average age of group members? In our group the youngest member was 45.
3. Did the mixture of professionals from different disciplines contribute to fragmentations?

Finally, I hope that Drs. Hunt and Issacharoff will comment on the following point. Our group was recruited and started with a clear understanding that it was a therapy group. Individuals who were not interested in therapy did not come. In this sense our membership was carefully selected. The group described by Drs. Hunt and Issacharoff started for one purpose (supervision) and then tried to change to another (therapy). I suspect this is difficult to do.

In a panel on leaderless groups at the annual meeting of the Los Angeles Group Psychotherapy Society, all the participants were in, or had been in, a leaderless group. The dynamics described in that meeting seemed similar. There were two groups that began as case discussion. Both of them had difficulty when they tried to change to therapy groups, while a group of Jungian analysts that started with a clear understanding that it was a therapy group did not experience such difficulties.

The authors' comments about the midlife crisis and the dynamics of their group seem accurate and very similar to our group.

From our experience I have a strong impression that the leader is not necessary when the group is composed of experienced therapists. In fact, the leader becomes a restrictive solution. I wondered if the authors' group had difficulty because some members wanted to hide behind case presentations or a leader? It takes great courage, good luck, and the right circumstances to even begin to be honest. Our group was relatively successful because members were able to be open, honest, and direct with one another. Do leaders maintain safety by inadequately helping the group to maintain restrictive solutions?

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FRANK KLINE, M.D.
Los Angeles, Calif.

Drs. Hunt and Issacharoff Reply

SIR: We appreciate Dr. Kline's thoughtful letter and wish to respond to his questions point by point.

Size did not seem a crucial or limiting factor in our group. When the group engaged in peer supervision or discussion of countertransference issues, it functioned very well despite its relatively large size. If it had become a true insight therapy group, we might have noticed a constraint imposed by size.

Our average age was about 37, making ours more of a midlife group than Dr. Kline's. I would expect that the members of his group had probably, for better or worse, made some peace with those conflicts characteristic of the midlife crisis and gone on to struggle with other issues.

Differences of professional background were irrelevant in our group. Polarization occurred on many issues, but never along that dimension.

Dr. Kline's comment about changing the group purpose in midstream contains an astute observation. In an as yet unpublished paper we have discussed group dynamic issues in more detail, including a long section on the difficulty or impossibility of changing the group contract or goals once the group is already in progress. Dr. Kline's group may have a happier history than ours largely because of a clear initial contract that was not changed.

Dr. Kline's suggestion that a leader is not necessary in a group of experienced therapists requires more extensive comment. In our opinion the fact that the group members were experienced therapists affected the language and content of group interaction but had little effect on the overall history of the group. The relationships between groups and their leaders involve very fundamental and strong group processes against which the psychological sophistication of the members is but a fragile shield. The laws of individual psychology and of group process are binding for all of us. The deeper our knowledge of those principles, the more we

can make use of them, but we are always subject to their force.

I can well imagine groups in which the members avoid self-revelation by hiding behind case presentations or other discussions of subject matter. With our group, it was the other way around (this may not be unusual). We tended to avoid exposing material relative to work competence by diversionary self-revelation.

We believe that it is in the nature of groups to create leaders and that it is in the nature of groups and leaders to be in conflict. The very existence of a group creates security in its members that sets the stage for their regression to a more childlike state. This activates dependency needs that result in a wish for a leader, which causes the group to create a leader. Once a leader is differentiated from the group, his very role makes him separate from the group and in some way antagonistic to it. Every group experiences its leader as restrictive and every leader experiences his group as recalcitrant. Leaders are judged (rightly so) by the nature of the restrictions they place on the group, but every leader must do this to some extent.

Without a leader who can keep his orientation to the group task and maintain the basic culture of the group and the conditions that support its cohesiveness, a group would lose its purposefulness and become a homeostatic structure satisfying the needs of group members. In order to function as a leader, an individual needs some base outside the group itself so that he is not dependent on the group for his sense of self or purpose. In this sense, every group with a task needs a leader or, as we would prefer to say, a leadership function. The leadership function need not be embodied in one individual. It can move from individual to individual, it can be contained in the relationship of the group to some outside institution or society or in some commonly held group ideal. There may be still other ways of embodying the leadership function.

We suspect that Dr. Kline's group contains a leader or leadership function in the sense discussed above. It is probably covert and functioning well, but could be discerned through careful observation of the group process.

WINSLOW HUNT, M.D.
AMNON ISSACHAROFF, M.D.
New York, N.Y.

Dignity, Dilemmas, Diagnosis

SIR: I would like to comment on "A Case of Petit-Mal Status: A Diagnostic Dilemma" by Michael Weissberg, M.D. (November 1975 issue). It seems that petit-mal status is not only a diagnostic problem but a problematic diagnosis. As Dr. Weissberg noted, "the independence of the clinical and electrical manifestations of the disease is well-documented." This brings into serious question the clinical significance of the EEG findings in the case he described. If biofeedback work shows nothing else, it does show that mental change can alter basic EEG patterns. Was electrical activity causing the mental status or vice versa?

It is striking that during the supposed epileptic impairment the patient was able to do the idiosyncratic and difficult task of skipping every other letter as she recited the alphabet backward. Are mental faculties curiously augmented in petit-mal status?

Another interesting aspect of the case is the winking or "irregular twitching." While I tentatively accept the possibly unwarranted assumption that the resident could not distinguish the two, I am nevertheless unwilling to consider irregular twitching of the eyes as always "organic." I have seen nonepileptic patients spontaneously demonstrate eye twitching during hypnosis. Further discouragement with the neurologic diagnosis in this case comes from the history of poor response to seizure medication.

How has Dr. Weissberg's diagnosis helped this patient? With the stigma of epilepsy substituted for the stigma of psychiatric disorder, this patient may go uncured for another 17 years on seizure medication, when perhaps hypnosis or biofeedback might help. I do not know whether to suggest psychodynamic psychotherapy also, since Dr. Weissberg gave no clues as to what lay behind the patient's description of "emotional upheaval," which she related to her seizures.

Finally, it is ironic that the paper expresses concern about the stigma of psychiatric hospitalization since the patient achieves no dignity in the case report until the positive EEG. The differential diagnosis seems to be: petit-mal status versus "crazy stripper." Thus we support the prejudices we decry.

KENNETH NAKDIMEN, M.D.
Jamaica, N.Y.

Dr. Weissberg Replies

SIR: The purpose of my report was to remind my colleagues of the existence and various presentations of petit-mal status. I hope my reply will satisfy Dr. Nakdimen's diverse and seemingly technical objections.

The EEG is an imperfect tool. Used in conjunction with clinical and historical evidence, however, it can verify diagnostic impressions. I caution to add that one can rarely confirm or deny the existence of an epileptic disorder with an EEG alone. It is unlikely that the disordered electrical activity by itself was "causing the mental status"; the neurophysiology of consciousness and cognition is incompletely understood. It is known that "psychological" events do alter brain processes, but the linkage between body and mind—the leap from soma to psyche—is not understood (1, 2). It is widely accepted that generalized seizures involve the centroencephalic systems (3), and I know of no instance where biofeedback techniques or mental activity provoked two to three cycles per second spike and wave discharges typical of petit-mal.

Response to seizure medication is not a test of neurologic diagnosis, and I am certain that Dr. Nakdimen is aware that there are many diseases in medicine that we diagnose but only imperfectly treat. I share his discouragement when faced with chronic diseases. I am glad to say, however, that petit-mal status usually responds to diazepam, as it did in the patient I described. Furthermore, the patient exhibited quite a good response to her medications after our first contact with her and, although not "cured," she was well controlled. Psychodynamic psychotherapy also seemed to be of benefit.

A careful rereading of the case will reveal that the patient was able to recite the alphabet backward skipping every other letter only after her EEG normalized. Therefore her "augmented" mental faculties were a result of the cessation of her seizures. Some patients (not the one reported) with

"markedly limited intellectual performance associated with severe paroxysmal abnormalities in the EEG, but with little else to indicate a continuing epileptic state" have improved markedly in cognitive abilities following medical or surgical treatment (4).

I am surprised that Dr. Nakdimen finds my concern about the stigma of psychiatric hospitalization ironic; I am sure he is aware that some patients suffering from epilepsy are also uneasy about their illness. Finally, I am glad to note that I share Dr. Nakdimen's conviction that irregular twitching of the eyes or winking is not always organic. Otherwise, we would find ourselves in not only diagnostic but also social dilemmas.

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MICHAEL WEISSBERG, M.D.
Denver, Colo.

A Positive View of the Chief Resident's Role

SIR: Having recently completed two years as a chief resident in psychiatry at the University of California, Los Angeles, Neuropsychiatric Institute, I was very interested in "The Chief Resident as Reluctant Staff Therapist" by Stephen A. Green, M.D. (October 1975 issue). Although I am no stranger to the quaint vocabulary of Kleinian and object-relations analytic schools, I found its use in a description of the functioning of a chief resident an academic exercise that did injustice to human interactions.

Like Dr. Green, I was often delayed in my hectic schedule by people seeking answers to difficult clinical problems, but not once did I feel the bite of "unconscious oral sadism." I was often called upon, as is any leader, to make decisions that affected people's work loads. Not infrequently, these people acted to protect their interests. Again, I managed to function without an "investigation of the entire staff's unconscious motivations." I discovered that people who work together often have frictions and fail to communicate. I do not recall having to "soothe and balance narcissism of staff members" or to interpret "unrealistic transference distortions" and "resistances." Unlike Dr. Green's obsession with unconscious fantasies, I found dealing with reality an excellent antidote for "becoming embroiled in struggles that seemingly came from nowhere."

Dr. Green is honest enough to describe his initial approach to his position as filled with "a flurry of obsessions and intellectualized activity" and expectations of the staff performing with "frenetic efficiency." He also bemoans his "progressive exclusion" from the unit, which he sees as a consequence of the "withdrawal" required by the job of chief resident in order to "preserve an effective therapeutic stance." I never experienced this progressive exclusion, and rather than the loneliness and isolation he described, I felt I worked in a warm, family-like atmosphere. I developed and maintain close friendships with many of my former colleagues. Rather than reawakening the anxieties of the first year of residency, I found my years as chief resident the most rewarding and satisfying in my training.

I suspect Dr. Green's findings are the result of his personal style of leadership and are not representative of the role of a chief resident. I believe that adherence to any dogma that turns people into "good" and "bad" objects is likely to create an image of a distant, aloof, uncaring leader. A Tavistock group model that was designed to intensify anxiety and transference might serve a particular group of patients well, but I see a strong argument against its application to the role of a chief resident in a setting already overwhelmed with anxieties.

The requirement that an effective leader be pragmatic seems ill served by slavish devotion to cutting and stretching reality to *any* theoretical model—and the more unsound the model, the poorer the fit.

JOSEPH R. SICIGNANO, M.D.
San Fernando, Calif.

Dr. Green Replies

SIR: It is surprising that Dr. Sicignano, a former leader of a therapeutic milieu, denies the existence of unconscious motivations in a psychiatric ward staff. His inability to recall the need to interpret staff resistances or transference distortions does not mean they were not present.

Dr. Sicignano feels I am "obsessed with unconscious fantasies" in suggesting that staff interactions do not always occur on the level of conscious awareness and criticizes my "slavish devotion" to a theoretical model. If he is referring to my belief that unacknowledged feelings, wishes, and needs derive from both patients and staff in a therapeutic milieu, he is correct. Unless this fact is recognized in psychiatric training and administration, the patient will ultimately suffer.

STEPHEN A. GREEN, M.D.
Bethesda, Md.

BOOK REVIEWS

Psychiatrists and Their Patients: A National Survey of Private Office Practice, *text by Judd Marmor, M.D.; based on a survey conducted by Patricia Scheidemandel and Charles Kanno; with commentaries by Gerridee Wheeler, James N. Sussex, Edward M. Kennedy, Zigmond Lebensohn, and Raymond Fink. Washington, D.C., Joint Information Service of the American Psychiatric Association and the National Association for Mental Health, 1975, 181 pp., $7.50.*

A 181-page book that contains 98 tables of statistics is difficult to read. I found myself wishing that Dr. Marmor's comments had been uninterrupted by the tables, that they had been relegated to an appendix. However, the material presented in the analyses perhaps justifies the rather ponderous method of presentation.

Stimulated by the current interest in national health insurance and the praiseworthy insistence that such plans provide adequate coverage for psychiatric care, the Joint Information Service of APA and the National Association for Mental Health (NAMH) set out to investigate the private practice of psychiatry, the patients receiving help from this sector, and the economics of the operation. With great persistence, Ms. Scheidemandel and Mr. Kanno were able to achieve a 73% return on questionnaires sent to 10% of the psychiatrists who reported in APA's 1970 census that they spent more than 15 hours a week in private office practice.

The data gathered in this way seemed so important that the Joint Information Service requested and (again, perhaps through admirable persistence) obtained an in-depth analysis by Judd Marmor and general comments from the president of NAMH, a U.S. Senator, an official of the Health Insurance Plan of Greater New York, and two distinguished psychiatrists. The result is a thought-provoking and informative survey of psychiatric practice that will be immensely useful to promoters of insurance plans and directors of training programs. Others will find themselves enlightened and at times surprised by the data presented.

Dr. Marmor notes the rather large proportion (29%) of psychoanalysts in the respondent group. Because psychoanalysts tend to see their patients more frequently and for longer periods of time, the use of psychoanalytic therapy as the treatment of choice raises some questions concerning the type of insurance coverage that could reasonably be provided. Full coverage would mean that a disproportionate amount of benefits would be used by comparatively few patients. Psychoanalysis as the treatment of choice should therefore be monitored by effective peer review systems. It would be equally unfair to deny patients a treatment modality that is optimum for them and to burden the majority of insured patients with an economic burden applicable to a few.

The survey also reveals that the "typical" patient of the private psychiatrist is not a bored, rich, idle housewife but a seriously ill person and that more men are represented in the patient population than is popularly believed. A surprising finding is the lack of children patients seen by the psychiatrists in the sample: only 2% of the patients seen by these psychiatrists were under the age of 10, and only 3% were between the ages of 10 and 14.

The commentaries following Dr. Marmor's extensive discussion provide additional dimensions to the picture. Both Ms. Wheeler and Senator Kennedy are concerned that insurance principles apply to the entire range of people who need psychiatric care and that the roles and responsibilities of psychiatrists in private practice working toward this goal be clarified. They agree that truly effective accountability is essential to avoid inequities.

Drs. Sussex and Lebensohn express their concern that quality of care be considered in any attempt to relate the practice of psychiatry to an economic system. In response to Dr. Marmor's stated surprise that ECT was administered to only .5% of the patients (a finding explained by the fact that these were outpatients and ECT therapy is usually an in-hospital procedure), Dr. Sussex and Dr. Lebensohn comment on the usefulness of this treatment modality in selected cases.

The insurance side of the picture is briefly presented by Raymond Fink, Ph.D. He points out that it is doubtful that national health insurance legislation could support the fiscal requirements of mental health insurance on a par with all other health services if a substantial proportion of these services were provided by private psychiatrists using their present modalities of care. It is this necessity for review of the current practices of private psychiatrists that inspired my comment about the importance of this survey to the training program director. Psychiatrists must learn to select treatment modalities in terms of patient needs, not in terms of preference or their own orientation. Dr. Lebensohn feels that general psychiatrists in private practice, "deeply steeped in the tradition of medicine," are best equipped to deal with the wide variety of psychiatric disorders.

This survey will help us all to evaluate the situation more accurately than has been possible in the past. The Joint Information Service is to be commended.

C.H. HARDIN BRANCH, M.D.
Santa Barbara, Calif.

The Psychological Birth of the Human Infant, *by Margaret S. Mahler, Fred Pine, and Anni Bergman. New York, N.Y., Basic Books, 1975, 293 pp., $15.00.*

The authors describe the infant as he proceeds from biological birth to psychological individuation. The senior author, a well-known psychoanalyst and theorist, has been dealing with the question of the preverbal development of the infant for many years. This work first began in the study of symbiotic psychosis and emerged into a study of the developmental process. The first time I heard Dr. Mahler speak, a discussant of her paper noted that the vocabulary had changed so considerably that it would be difficult for Freud

to understand it. The second discussant remarked that if this were true, perhaps the author should translate the language into terms that *even* Freud could understand.

Although the text is at times ponderous, the hypotheses are clear and the concepts sharply defined. To understand the intrapsychic development of preverbal children one must depend upon inferences from observations, and there will always be argument about the legitimacy of such inferences. The authors recognize this difficulty: "Although we cannot ultimately prove the correctness of such constructions, we nonetheless believe that they can be useful and we are committed to attempting their formulation" (p. 14).

The volume describes the research setting, providing floor plans and observational methods. There is a concluding section of summary and reflections with suggestions for research directions and inferences about possible further ways of proceeding with the research.

The main body of the volume consists of an exposition of the phases of the separation and individuation process:

> We see each phase as a time when a qualitatively different contribution is made to the individual's psychological growth. The normal autistic phase serves postnatal consolidation of extrauterine physiological growth. It promotes postfetal homeostasis. The normal symbiotic phase marks the all-important phylogenetic capacity of the human being to invest the mother within a vague dual unity that forms the primal soil from which all subsequent human relationships form. The separation-individuation phase is characterized by a steady increase in awareness of the separateness of the self and the "other" which coincides with the origins of a sense of self, of true object relationship, and of awareness of a reality in the outside world. (p. 48)

The authors identify the subphases of separation and individuation as follows: 1) differentiation in the development of the body image, 2) practicing, 3) rapprochement, and 4) consolidation of individuality and the beginnings of emotional object constancy. They recognize that individuals have their own styles and that there is no definitive timetable for this process, which takes place during the first three years of life. They also recognize the importance of normal mothering as a catalytic phenomenon:

> It is the specific unconscious need of the mother that activates out of the infant's infinite potentialities, those in particular that create for each mother "the child" who reflects her own *unique* and individual needs. This process takes place, of course, within the range of the child's innate endowments. (p. 60)

The fact that the authors indicate vulnerabilities in the developmental process and their consequent preventive implications is an important contribution. They also wisely include five detailed descriptions of children's subphase development so that clinicians may correlate their own experience with them. Although they make an effort to adapt biological principles and terminology (e.g., structure, epigenesis, and homeostasis) to metapsychology, they do not fully achieve the law of parsimony in biology. They also fail to relate to their discussion the work of others, such as that of Bowlby, Ainsworth, and the ethologists as well as the recent findings of competency in infancy.

It will be necessary to retest and correlate the authors' findings with current work, and the volume will provoke much discussion. Nonetheless, it will stand by itself as a classic of psychoanalytic research and investigation, shedding new light on the vicissitudes of child development. For those of us who have followed this work over the years and have taught these concepts, it is a welcome addition, and further contributions will be eagerly anticipated.

IRVING PHILIPS, M.D.
San Francisco, Calif.

Coverage and Utilization of Care for Mental Conditions Under Health Insurance—Various Studies, 1973–1974, *by Louis S. Reed, Ph.D. Washington, D.C., American Psychiatric Association, 1975, 80 pp., $4.00 (paper).*

This book represents Dr. Reed's final contribution to the collection and analysis of data demonstrating the feasibility of covering mental disorders under health insurance. It was published just a few days before his death in December 1975. To this work, as to all his many other writings in the field of health economics, Louis Reed brought great gifts: high intelligence, an ability to organize and make sense out of complex material, an unparalleled background in health insurance, and a remarkable zest for accumulating new knowledge.

Dr. Reed came to work for APA in 1970 as director of a project on the utilization and cost of psychiatric care under health insurance. He had just retired after a distinguished career in government and teaching in which he carried out in-depth research in the field of medical care with a view toward the eventual enactment of national health insurance. I do not think it an exaggeration to suggest that the book that resulted from the APA project (1) was given special attention by the insurance industry and the drafters of health insurance legislation because of Louis Reed's reputation for integrity and for rigorous, scholarly research. As one who was involved in that book as a coauthor, I can testify to Dr. Reed's skill in digging out material from myriad sources (some of which were less than eager to share their information), organizing it in an intelligent and coherent way, and analyzing it so it was understandable to those whose knowledge of health economics was not very deep.

This new book may be considered in one sense a supplement to the earlier one because it updates some of the information in that book, but all of the data are new. Six discrete studies were made by Dr. Reed (five of them under a contract with NIMH), and no effort was made to fit them into a cohesive package. However, together they make an impressive case for the inclusion of substantial mental illness benefits in health insurance plans.

Probably the most dramatic piece of evidence leading to this conclusion is the study of utilization of the high option of the Blue Cross and Blue Shield plan for federal employees, which is the largest private health insurance plan in the world and has essentially nondiscriminatory coverage of mental illness (although there are some special restrictions on care in private and public mental hospitals). The upward trend in utilization of benefits for mental disorders that has characterized this plan since its beginning in 1960 seems to have ended: utilization leveled off in 1973 and was slightly lower in 1974. In 1972 benefits for mental disorders constituted 7.3% of benefits for all conditions; in 1973 they constituted 7.4%; and in 1974 they were back down to 7.3%. This

study also gives detailed information on utilization patterns, with geographical, age, and sex breakdowns, duration of hospital stay, and distribution of persons undergoing office care for mental conditions by amount of physicians' charges covered.

Also of special interest in this book is the study describing the coverage of mental illness in employee health benefit plans. Nearly 150 plans, representing a broad cross section of American industry and including such giants as IBM, General Motors, Exxon, U.S. Steel, and American Airlines, were surveyed in regard to their coverage of mental illness. Almost all of the plans had some coverage of hospital care for mental conditions, and about two-thirds had the same coverage for mental as for other conditions when the care is given in general hospitals. In addition, about 90% of the plans covered some office care for general and/or mental conditions, and nearly 50% of these had nondiscriminatory mental illness coverage. The existence of this very substantial coverage makes the framers of most of the health insurance bills introduced in Congress appear unduly timid; one wonders if they are aware of how many Americans already have relatively good coverage of mental illness through the health insurance plans where they work.

The four other studies described in Dr. Reed's book cover the following areas: coverage of mental illness under the collective bargaining agreements of selected major unions; utilization of care for mental conditions under the Canadian health insurance program, which covers mental illness on the same basis as other illnesses; current benefits for hospital care of mental illness in Blue Cross plans; and data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

The six studies described in this book should prove extremely useful in winning improved mental health benefits in health insurance: through employer-employee-union plans, through state-mandated coverage, and eventually through national health insurance. If through research efforts such as this more patients who need mental health care are helped to pay for their treatment, the book will serve as a worthy memorial to a man who was not only an able health economist but a warm humanitarian.

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EVELYN S. MYERS
Washington, D.C.

Emergencies in Child Psychiatry: Emotional Crises of Children, Youth and Their Families, *edited by Gilbert C. Morrison, M.D. Springfield, Ill., Charles C Thomas, 1975, 456 pp., $24.50.*

This book is a collection of 30 articles, 23 of which have been previously published. Most of the 22 contributing authors are highly respected teachers of child psychiatry.

Following a captivating essay that illustrates views of childhood portrayed in literature, the introductory section begins with a discussion of crisis intervention versus long-term intensive treatment. The hope is expressed that someday the clinicians dedicated to each ideological position will stop debating maximum versus minimum in favor of optimum treatment. May the planners and funders please take note.

Defining emergencies in child psychiatry is likely to be a matter of some interest to readers familiar with this subject, who will already have found many less than satisfactory attempts at definition. These authors point out that most emergencies present as intolerable behavioral problems but that developmental and emotional disturbances were present much earlier. At any age, homicidal and suicidal threats and acute decompensating psychotic reactions constitute emergencies. They constitute a threat to biological integrity or life, a condition commonly cited in defining a medical or psychiatric emergency.

Acute school phobia, generally considered an emergency by child psychiatrists, does not constitute such a threat. It interferes in a major way with consistent ego development, however, and that theme is developed by the editor to reach the following definition of an emergency in child psychiatry: "that situation in which the significant adults around the child can no longer help him master his anxiety and can no longer provide temporary ego support and controls."

The second section of the book, Crisis Intervention, Brief Treatment, and Early Prevention, contains 7 chapters on these subjects. The third section, Clinical Experience and Research, includes discussions of depression, suicidal behaviors, aggressive behaviors, school phobia, and emergency consultations on pediatric wards. The fourth section, Treatment Planning, has more chapters on crisis intervention as well as reports on drug abuse and drug therapy.

A common criticism of volumes of collected papers by several authors is that the papers may not all be related to the central theme. That must be said of this volume, but it is the only disappointment I found. The papers on primary prevention and intensive diagnosis and treatment, although laudable, are only peripherally pertinent to a discussion of emergencies. A discussion of psychosocial factors in adolescent pregnancies is even less so, particularly because this one was written long before the Supreme Court decision on abortion of January 1973.

Perhaps the subtitle of this book, *Emotional Crises of Children, Youth and their Families*, more accurately describes its content than the title. As is pointed out in the book, " 'Crisis' is not the same as 'emergency.' " If taken as a volume on crises, it will be of value and interest to all mental health professionals—a book in which one reads a chapter or two at a time but which is frequently taken from the shelf to reread.

JOSEPH M. GREEN, M.D.
Tucson, Ariz.

The Future Role of the State Hospital, *edited by Jack Zusman and Elmer F. Bertsch. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1975, 390 pp., no price listed.*

The words "state hospital" often conjure up an image of things—massive buildings or isolated grounds—rather than an image of people—patients and personnel doing things and currently in the throes of role change. The role change is complex because of the great variation among state hospitals in leadership, location, professional practices, patient populations, political relationships, community images, and economic realities. This book, which scans the many issues related to change in state hospitals and successfully serves to

emphasize function over structure, reports on a conference held under the leadership of the department of psychiatry of the State University of New York at Buffalo. The editors take the rich content of the individual contributions and give them a meaningful continuity and a balanced perspective.

Demone and Schulberg provide a clear view of the new human services network as the context for the future role of the state hospital. Because community-based mental health services are emerging as viable cores for these networks, state hospitals that are linked to community centers are likely to emerge with new vitality and different functions. On the other hand, state hospitals that are isolated from community programs are assigned the kind of bleak future they may have earned through their rigid adherence to mental status, labels, and pills as the only or primary methods for human problem solving.

The stance that state hospitals must either change or close is continued in the next section of the book, which considers outside pressures. Here the increasing impact of constitutional requirements in regard to the management of the mentally ill are made lucid by Judge Flashner. One's constitutional right to liberty is shown to override such other legal issues as treatability and dangerousness. The impact of litigation on the state hospital is neatly packaged by Bruce Ennis, who sees the state hospital as on its way out. However, Peszke demands that laws be ultimately workable at the level of clinical practice.

A review of the newer treatment modalities presents the constructive impact of behavioral approaches in dealing with regressed patients. Cannon stresses the need to prepare patients as well as staff members of both state hospitals and halfway houses for the transitional crises that emerge when patients are placed in the community.

A major section of the book deals with internal pressures for change by presenting programs at state hospitals that have humanized their patients and staff, established new links with programs in the community, established their own programs in the community, and used empty space in the hospital for other human services agencies. This section includes a rich sampling of experience from rural and urban programs. For example, Mesmer's description of the community program of Warren State Hospital in Pennsylvania demonstrates how outreach programs represent roads to survival. From the standpoint of urban state hospital services, Talbott is worth quoting:

> The state hospitals' long standing commitment to the severely mentally ill, coupled with the widespread lack of interest by others in dealing with such impoverished people, militates against an early demise.

Cole's personal and colorful account of his experience at Boston State Hospital describes what happens when a superintendent juggles demands from every direction. Another chapter deals with recent developments in public mental health care in France and concepts of the state hospital in the U.S.S.R. These enlighten the reader to the fact that complex bureaucratic binds that serve to perpetuate the status quo in mental health service delivery systems exist everywhere.

The opinions given in this book are generously supported by statistical tables, many of them from the Biometry Branch of NIMH, which through the years has helped to make planning of state hospitals less speculative and rhetorical.

Conferences and books about conferences are successful only if their information generates new concepts and methods. This book succeeds because it highlights the need to find and solve problems relating to the future role of the state hospital. As Erickson asked (1), "Is neglecting the traditional solution [the state hospital] leading to neglect of the problem . . . that makes entry into an institution necessary?" In agreement with Arnhoff's recent warning concerning abandonment of state hospital programs (2), this book conveys the message that a soaring interest in closing state hospitals is not necessarily paralleled by deeper concern or clear plans for the future of the people the hospital serves. New programs are needed to assist in problem solving with the regressed mentally ill, the public offender, the chemically disabled, the deviant, the alienated, and the socially dependent person.

When the walls of state hospitals built with the zeal of reform in the 1840s have crumbled and the dust of the 1970s' reform movement has settled, we may still have failed to deal effectively with the severely regressed or damaged person whose psychosocial functioning is fragile.

To me, this book conveys the message that state hospitals will close, but their trappings may be hung in other halls and their old rituals may be performed in the community without really accomplishing a great deal for the citizen. It urges all those who are concerned with the role of the state hospital to focus on programs for people. Unless this is done now, we may look back from some point in the future and remember with dismay the warning best expressed by the French: "Plus ça change, plus c'est la même chose."

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JAMES W. DYKENS, M.D.
Concord, N.H.

The Care and Training of the Mentally Handicapped: A Manual for the Caring Professions, *by Charles H. Hallas, S.R.N., William I. Fraser, M.D., and Ronald C. MacGillivray, M.B., D.P.M. Bristol, England, John Wright and Sons (Baltimore, Md., Williams & Wilkins, exclusive U.S. distributor), 1974, 269 pp., $18.75.*

This is the fifth edition of a textbook and manual first published in 1958. It is written for nurses and child-care personnel and concerns the care and management of mentally handicapped individuals. This edition is important because it is the first in which the senior author, a nurse, has had the assistance of two psychiatrists as coauthors. As a result, this edition has an up-to-date emphasis on multidisciplinary and community approaches. The legacy of the four previous editions provides the reader with a perspective that combines the new and the traditional.

Although the book is addressed to professionals in England, Scotland, and Wales, many of its principles and discussions are applicable in other nations as well. In Great Britain the diagnosis of mental handicap is applied to persons who would be classified under the diagnosis of mental retardation in *DSM-II* (1).

The authors provide historical comments on the changes of opinions concerning concepts of care for the mentally handicapped. They endorse the modern emphasis on non-

institutionalization of the mentally retarded, but they caution that this trend may become overemphasized to the point that children who need to be separated from their families for a period of time may not be given this opportunity because of the tendency of some professionals to follow the latest ideas to the extreme.

The chapter outlining diagnostic categories of mental handicap is written with competence. The chapters on patient and family management, written specifically for the staffs of institutions, are also of particular value. For those interested, the description of the recent health acts in Great Britain and Scotland provide a detailed review of current developments there.

In general, this volume would make a good text for nurses, aides, and child-care workers in training as well as a good manual for in-service programs. It provides adequate references for particular problems. The reader in the United States will sometimes be amused by parochialisms on the part of the British authors, including occasional uncomplimentary references to American culture. Also, the old and the new are sometimes strikingly juxtaposed. For example, a section giving detailed instructions for the nursing sisters on the making and stoking of coal fires on the wards is followed by a description of the latest techniques of behavior modification as applied to patient management.

The style of writing varies from chapter to chapter, a result, one would guess, of the difficult process of revision that had to take into account the changes in professional and cultural attitudes toward the mentally retarded in the last decade.

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PAUL N. GRAFFAGNINO, M.D.
Hartford, Conn.

Frames and Cages: The Repertory Grid Approach to Human Understanding, *by Anthony Ryle. New York, N.Y., International Universities Press, 1975, 146 pp., $11.50.*

The repertory grid technique stems from the late George Kelly's theory of personal constructs. For Kelly, the key to understanding the personality of an individual lies in the representations or models of the world that the individual has adopted as a result of his experience with it. Access to this personal structure of constructs and values is attained by asking the person to rate what Kelly termed "elements," such as significant others, the self, objects, situations, or concepts, on a series of "constructs," which consist of bipolar scales, such as timid/bold, attractive/unattractive, and desirable/undesirable. These ratings may then be related on a grid that offers a two-dimensional spatial representation or projection of the interrelationship between elements and constructs. The grid may result from a simple empirical analysis of the ratings or from the application of highly sophisticated mathematical procedures.

The technique is a flexible one because it calls for the clinician to select those elements and constructs which he feels are pertinent to the patient's (or client's) problems. Thus a patient with disturbed family relations might be asked to rate significant family members and himself on such dimensions as friendly/hostile, dominant/submissive, and pleasing/disturbing. The repertory grid as a technique is independent of the theoretical framework within which it is applied. Thus, although Ryle's orientation is psychoanalytic, his systematic position does not alter the basic repertory grid methodology. Rather, it expresses itself in the choice of elements and constructs to be used and in the clinical interpretation of findings.

Ryle begins with a critical assessment of Kelly's philosophy, followed by an exposition of the repertory grid technique. He provides numerous case examples and cites the experimental literature, albeit not extensively. He then integrates all of this with his personal analytic position. Thus the book essentially consists of two intermingled parts: a clear exposition of the repertory grid technique and an equally clear development of the author's psychoanalytic approach.

There can be little disagreement with the explanation of the technique. Anyone should understand and be able to use the grid technique after reading this book. However, some disagreement may inevitably arise with Ryle's psychoanalytic interpretation. As a theoretician he is stimulating, but he writes with a definitely authoritative stance that makes few concessions to opposing views. One wonders whether his strong criticism of Kelly's thinking as sometimes "naive, overstated, and overpolemical" may not boomerang.

The book is addressed primarily to those who are professionally concerned with the assisting and measuring of change in individuals with personal difficulties. It is clearly written but at a professional level that demands careful, attentive reading. It merits the interested clinician's attention, both as an introduction to the repertory grid technique and as a thoughtful integration of Kelly's position with the psychoanalytic orientation of the author.

WILLIAM A. HUNT, PH.D.
Chicago, Ill.

LSD—A Total Study, *edited by D.V. Siva Sankar. Westbury, N.Y., PJD Publications, 1975, 922 pp., $29.75.*

In the eighteenth century it was fashionable for men of letters to write compendia on natural history. These scrapbooks often contained a miscellany of observation, experience, hearsay, and fantasy. Because the authors were well grounded in religious learning, the volumes also included religious allegory and citation. *LSD—A Total Study* is such an effort for our century; it combines biochemical, pharmacological, physiological, and historical information with anecdotes, opinions, and popular statements.

This compilation is large (922 pages); 73% of it was written by Dr. Sankar and the rest by such students of hallucinogens as Harold Abramson, Roland Fischer, Leonide Goldstein, and John Smythies. In addition to the technical sections, there are chapters devoted to social and legal issues. The appendices include a classification of psychotropic drugs, a list of legal penalties in New York State and California, and an index of slang terminology. Reference lists follow each chapter; these are in the style of chemical journals with authors, journal, volume, and first page number given but without the article's title.

The volume's strength is in its chemical details, particularly in the formulas for congeners of hallucinogens and identifi-

cation of LSD in fluids. There is also value in the miscellany that appears in the figures (many delightful drawings of opium use, for example) and in the citations concerning a wide range of drugs of abuse. One finds such tidbits as "Mimosa Hastibus: a hallucinogen used by Brazilian Indians; contains dimethyltryptamine" and "Fly Agaric: consumed in Siberia especially by Koryaks. During carousing men stayed intoxicated for several days at a time, even drinking their own urine to prolong the effects! Used in Shamanistic rituals."

It is not clear what posture Dr. Sankar wishes to take with regard to the use of LSD in psychiatry. In his epilogue he warns against LSD use ("LSD is a very potent drug and as such its use could certainly result in damage that cannot be adequately estimated at this time"); suggests uses for which the text shows little support ("LSD has enhanced appreciation of sight and sound, including music, on occasions . . . [and] increased religious consciousness and mystic abilities"); and exhorts ("With all the evidence indicating the undesirable effects of LSD, I wish to emphasize . . . it has opened the eyes of our society to many problems. In so doing, the users themselves became victims of the use"). There are references to Oriental and Hindu philosophy in the text, suggesting that LSD was selected as the topic for this volume more for its association with mysticism than its salience for modern psychiatry.

The volume is commendable as a source of miscellany concerning LSD and other hallucinogens. It is a useful addition to resource libraries, where it would best be located among volumes dedicated to the chemistry, pharmacology, and ethnopharmacognosy of drugs.

MAX FINK, M.D.
Stony Brook, N.Y.

The Theory and Practice of Group Psychotherapy, 2nd ed., *by Irvin D. Yalom. New York, N.Y., Basic Books, 1975, 522 pp., $12.50.*

Dr. Yalom sets the tone of the second edition of his book by stating that psychotherapists who examine their basic view of man soon discover they are and always have been existentialists. Presumptions such as this abound in the volume, and, although the author states that this book is not the place for a treatise on the existential approach in psychiatry, he offers a good deal more about existentialism than the reader of a book on group psychotherapy may have bargained for.

Dr. Yalom displays a tendency toward overstatement that is confusing. One wonders why it is necessary to describe the entire body of knowledge regarding psychiatric diagnosis as "not only errorful but offensive"—a view with which most psychiatrists would disagree. Is Freudian psychology truly "materialistic" and "antivitalistic"? Is concurrent individual and group therapy "neither necessary nor helpful"? Are structured exercises such as those used in behavioral group therapy "undesirable and antitherapeutic"? And so on. The author shows a strong urge to separate himself from any psychoanalytic contamination, seriously questions genetic causality, minimizes early experience and its effects on adult behavior and character structure, and believes that groups should be primarily ahistoric and should not "water down the tragedy of life," as they too often do, in his opinion.

In addition to speaking out on theoretical issues, Dr. Yalom incorporates into this book many of his research findings on encounter groups in an effort to update the volume. He measured outcome and the relationship of outcome to leader technique and found, among other things, that most encounter group participants are generally positively inclined toward the experience and believe it to have been useful. A word of caution to the beginning group therapist is in order here. Many of the clinical vignettes and conclusions scattered throughout the book are drawn from this research and other short-term encounter group experiences in which the groups were composed of normal people who required a type of education. The therapy group, on the other hand, is composed of persons who are mentally ill and who seek to have their disorders cured or, at least, ameliorated. Although the two types of groups have some things in common, there are important differences. A technique used or an outcome realized in one type of group is not necessarily valid for the other.

A variety of group therapy approaches is available to the clinician, and no one has been able to demonstrate the superiority of one method over another. Dr. Yalom favors the here-and-now approach and does not pay enough attention to the many alternative methods of group treatment. Indeed, he shows a tendency to be hypercritical of other methods, as in his description of transactional group psychotherapy as simplistic and nonproductive. One wonders if the time is not past when a single author can discuss the variety of group approaches with sufficient depth and objectivity to do justice to this rapidly expanding field.

In this book's first edition, which I also reviewed, Dr. Yalom's discussion of the curative factors at work in every therapy group—irrespective of the therapist's technique or theoretical orientation—was of value, as were chapters on the selection and preparation of patients for a group therapy experience, on the composition and organization of a group, and on problem patients and group therapy dropouts. Fortunately, these chapters, which constitute a large part of the second edition, remain virtually intact. Those who are thinking of buying the new edition should be aware that they will get a great deal of the author's personal views of psychiatry in addition to a book on group psychotherapy.

BENJAMIN J. SADOCK, M.D.
New York, N.Y.

The Dream in Psychoanalysis, revised ed., *by Leon L. Altman, M.D. New York, N.Y., International Universities Press, 1975, 272 pp., $12.50.*

Dr. Altman has written a short, clear description of the clinical and theoretical use of the dream in psychoanalysis. He thinks analysts and analytic students do not currently use the dream as fully and as effectively as they might. The author carefully selects clinical examples to illustrate the dream's use in analyzing resistance, transference, underlying impulses, homosexual libido, and oedipal conflict. There is a chapter on how the dream can be used to measure adaptive ego and therapeutic progress as well as a description of how Dr. Altman uses his own dreams to understand countertransference and to better understand his patients.

The author carefully describes the dream, the associative material, the analyst's unspoken thoughts, and finally his interpretation. He carefully explains that not all dreams can be analyzed; he presents only carefully selected dreams that

were understood. In psychoanalysis, dreams that cannot be understood outnumber those which can be analyzed by far.

This book belongs in the hands of the analyst and the budding analyst. It is also clinically useful and can be profitably read by therapists with a marginal interest in psychoanalysis.

FRANK KLINE, M.D.
Los Angeles, Calif.

Chemical Coping: A Report on Legal Drug Use in the United States, *by Carl D. Chambers, James A. Inciardi, and Harvey A. Siegal. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1975, 154 pp., no price listed.*

The goals of ascertaining the extent of use of legal psychoactive drugs in this country and learning something about the people who use them are to be applauded. This book is divided into four parts: Prescriptive Drugs, Over the Counter (OTC) Psychoactive Drugs, Alcohol, and a Summary that includes excellent abstracts of 15 recent publications. However, there is no bibliography, which limits the usefulness of the book for professionals. Each section contains a description of the psychological and physiological effects of the drug under consideration. Although this is not of sufficient depth for the knowledgeable professional, it will interest lay readers.

The information in the book represents the results of personal interviews of unspecified length and format with 30,000 people from the age of 13 to over 50 in 16 states, including Washington, D.C. No western coastal state was included. The authors assure us that the higher rates of drug use reported in the states excluded would not be "so high as to significantly alter the projections we are proposing." No information about the selection of sample or the numbers that came from any area of the country are included. It is impossible to tell whether the sample of 30,000 included any or all of the 7,500 people surveyed in New York State. The latter survey is the basis of a "special analysis concerning the rate of regular [drug] use within various worker categories [which makes] it possible to rank the regular users of the drugs."

The statistical analysis is presented by projecting survey data for the total population older than 13 in terms of age, sex, socioeconomic status, race, and occupation. The analysis highlights the fact that the majority of the drug using population are women; the exception is that men younger than 25 use more prescription and OTC stimulants. There appears to be little difference between races with regard to use of barbiturates, major tranquilizers, amphetamines, and OTC stimulants and tranquilizers. Blacks are significantly overrepresented among users of nonbarbiturate sedatives, and whites are overrepresented among users of minor tranquilizers, OTC tranquilizers, and antidepressants. Housewives, who are major drug users according to the data for the whole sample, do not rank as high in drug use in the more detailed study of occupation and drug use performed for the 7,500 respondents of New York State.

The number of typographical errors in this book is considerable. Therefore, questions have to be raised about the care with which tabulations were presented. Obvious errors exist on page 124 (4.9% should read 49%) and on page 122 (the legend for a bar graph is inaccurate). In the course of outlining relevant points to present in this review, I prepared a table from the data given in the text. When I compared my table with those presented on pages 51–53 and page 87, I noted 5 discrepancies. In addition, there were 14 areas in which data presented in one section of the book were not described in other, comparable sections.

The book would be of more interest if it moved from emphasizing the demographic to considering a broader range of topics, e.g., speculating on reasons why people who use one drug choose to use another as well; why women seek more medication than men; or why major tranquilizers were associated with a higher rate of alcoholic use than minor tranquilizers or sedatives. Critical consideration of the respondents' answers is also missing in at least one area: 71.4% of regular users of amphetamines for diet control stated they never used the drug while on the job; it is conceivable that the respondents took sustained release capsules early in the day so that they had "drug activity" even though additional pills were not taken.

Because the authors had an opportunity to gather data on a national rather than a local level, it would have been of interest to learn about the drug use habits of families, the relationship between legal and illicit drug use, and the personality patterns of drug users (1–3).

The importance of this book lies in its provision of basic epidemiological data. The simple straightforward style with which it is written makes it possible to skim it quickly and gain a general understanding of the subject. One only wishes that the authors had included better data as to the representativeness of their sample and given more thought to the implications of their findings, rather than conclude, "We suggest that social planners . . . begin to determine what 'the problem' really is."

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EDWIN ROBBINS, M.D.
New York, N.Y.

Stone Age Crisis: A Psychiatric Appraisal, *by B.G. Burton-Bradley. Nashville, Tenn., Vanderbilt University Press, 1975, 121 pp., $8.95.*

Dr. Burton-Bradley draws on his 15 years of experience as the only psychiatrist in Papua New Guinea for this book. The original papers were presented at Vanderbilt University while he was the Abraham Flexner lecturer in 1973.

The initial chapter describes the transformation of a culture from the stone age to the twentieth century in the brief span of one generation. The author subsequently focuses on specific psychiatric entities, including the cargo cult, suicide, and amok. He then proceeds to examine the people, the problems of mixed races and mixed blood, and the type of psychiatry practiced in Papua New Guinea.

Burton-Bradley describes the cargo cult as a pathological group process in which the leader is usually a psychotic indi-

vidual who promises to deliver a cargo that will change the status and life of his followers. The author shows through case examples that this behavior often leads to psychotic homicide or suicide.

Dr. Burton-Bradley notes the difficulty of defining and diagnosing the phenomenon of suicide. He contrasts the means of suicide in New Guinea to those in more developed nations; popular modes include poisoning by derris root and running amok. He theorizes that the explosive reaction referred to as amok is seriously influenced by group expectancies and discusses the psychogenic versus the organic as causes of amok, concluding that the organic explanation is a red herring with no scientific validity.

This book is easily read and provides a transcultural view of psychiatric illnesses. I found it to be concise and free of the ambivalence typical of many psychiatric textbooks. I would recommend it to psychiatrists for both its informative and its transcultural content.

CLAYTON PETTIPIECE, M.D.
Omaha, Neb.

The Role of Diagnosis in Psychiatry, *by R.E. Kendell, M.D. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1975, 166 pp., $14.00 (paper).*

This short book undertakes the task of discussing the pros and cons of diagnosis in psychiatry. On the disadvantage side of the controversy the author discusses the disease concept, the harmful effects of labels (including physicians' treating diseases instead of people), and other related issues. The major point made on the advantage side of the ledger is that diagnosis improves communication.

In the middle of the book the author discusses reliability and validity, disease entities in psychiatry, and *ICD-8* (1). The final three chapters broach the subject of defining criteria for different disease entities; the last chapter is titled "Diagnosis by Computer."

While reading this book one has a response similar to that one gets from having to fill out the Medicare-Medicaid forms to justify admission of a patient. After reading this book one has a better understanding of the principles behind checklist diagnoses. With the increasing demand for accountability in medicine, this book offers some assistance in the task of formulating a means of objective diagnosis. It allows the reader to formulate his or her thinking in an active rather than a reactive manner.

Overall, this is an interesting book that may prove timely in that it may assist the reader to make suggestions to APA's Committee on Nomenclature and Statistics, which is working on the third edition of *DSM* (2) (planned for completion in 1978).

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JAMES A. DAVIS, M.D.
Omaha, Neb.

Medical Sociology: A General Systems Approach, *by Leon S. Robertson, Ph.D., and Margaret C. Heagarty, M.D. Chicago, Ill., Nelson-Hall, 1975, 208 pp., $11.00.*

This textbook of medical sociology devotes eight chapters to a rather detached but comprehensive view of various aspects of medicine: the making of a patient, the making of a physician, the multiple relationships of the patient and physician, and the relationship of both to the family, the community, the hospital, the local and central government, and, finally, the international scene.

As an addition to this rather standard sociological approach, the first chapter is devoted to a good, concise description of general systems theory, and the final chapter is a generalized application of systems methods to the sociological material.

My overall reaction to this book is positive. Although it contains no specifically psychiatric material, it has a great deal that will interest the psychiatrist. It is carefully written and has a good list of references, a name index, and a subject index. One hopes that the authors will bring a closer integration of systems theory with their sociological material in a later book.

EDWARD J. CARROLL, M.D.
Miami, Fla.

The Innovative Psychological Therapies: Critical and Creative Contributions, *edited by Richard M. Suinn and Richard G. Weigel. New York, N.Y., Harper & Row, 1975, 242 pp., $7.95 (paper).*

This book consists of a group of articles selected by two college professors as representative of the newer forms of psychotherapy. The editors' goal was to provide college students with samples of similar size and style. Most of these are garnished with scientific vocabulary, but the result is rather bland. One will find pieces on behavior therapy, operant conditioning, coverant control, aversive control, anxiety management, Gestalt groups, nude marathons, touching, saturation, drug addiction therapy, group exercises, the use of videotape in group therapy, free clinics, campus crisis centers, psychodrama, milieu therapy, an analysis of hospital discharge rates, and paraprofessional counseling.

At least four of the articles deserve special mention: Ayllon and Azrin on a token economy, Mintz on touching, Bassin on Daytop Village, and Russell on group exercises. The first is a good example of clinical description and explication of treatment goals; the second is a reflective essay about intimacy phrased in psychoanalytic terms; the third is a warm, candid account of drug addiction treatment; and the fourth enumerates the many techniques group leaders use along with the objective of each maneuver.

The overall flavor of this sampler can be seen in the following statement of Kanfer and Phillips:

> But whenever undue emphasis is placed on a particular technique, be it hypnosis, psychoanalysis, psychosurgery, or use of tranquilizers, there is a serious danger that faith instead of reason becomes the guiding criterion.

The bias reflected in this collection appears to be toward activism. Notably absent, although "innovative," are Trans-

cendental Meditation, transactional analysis, and biofeedback. This text does not pretend to be an exhaustive and definitive college survey course, but it should provide some nourishment without dulling the taste of its readers for heavier fare and full digestion.

ROBERT VOSBURG, M.D.
Hanover, N.H.

Re-Visioning Psychology, *by James Hillman. New York, N.Y., Harper & Row, 1975, 260 pp., $12.50.*

This book, the most comprehensive expression of Hillman's thought to date, was created from his 1972 Terry Lectures given at Yale University. Carl Jung had given the Terry Lectures 35 years earlier on the subject of psychology and religion. Jung documented the reality of the unconscious and the power of the natural symbols emerging. Hillman tackles somewhat the same subject from the vantage point that time has given and ranges farther afield, from Heraclitus to Freud, Jung, and beyond. His book is so rich in content and imaginative possibilities that a review cannot do it justice.

Hillman describes his aim as "an attempt at a psychology of soul, an essay in re-visioning psychology from the point of view of soul." His concern is "soul making," and he uses the word "soul" rather synonymously with "anima," the archetype that relates one to the deep unconscious and its wealth of images. As the soul provides a connection to the God-image, we can understand the author's statement, "A re-vision of psychology means recognizing that psychology does not take place without religion, because there is always a God in what we are doing." For Hillman there are many gods affecting the soul, a polytheistic position, and man's abode is a Jamesian pluralistic universe.

The author delineates four functions of the soul: personifying, pathologizing, psychologizing, and dehumanizing.

To describe the personifying function Hillman goes back to the Greeks, who personified the psychic powers and gave them names, shrines, and altars. He chooses this word to keep close to the image per se and its impact. He would say trust and have faith in the imagery rather than interpret, discipline, or form defenses against it.

On pathologizing Hillman states, "Without psychopathology there is no wholeness." Soul, archetype, and myth insist on pathologizing, i.e., forming images of sickness. Hillman searches for the psychological necessity in this. Ultimately it is toward death that the soul leads, but there can be symbolic deaths and new beginnings on the road toward building one's own unique death. Again Hillman advocates going with the imagery rather than treating, correcting, or medicating it away.

In his chapter on psychologizing Hillman searches for the gods in our ideas and how the soul provides the tools to have insight into its own purpose. He argues against literalism, which blocks understanding of the soul's symbolic expression.

In the final chapter, titled "Dehumanizing or Soul Making," Hillman points out that modern psychology "has been obsessed by one overvalued idea: Man." He states that "we refuse to admit . . . that human reality is dependent on the realities that take place in the soul." Therefore, he strikes out against the humanistic psychologies, which he says have softened "our sensitivity to archetypal realities and keeps our perceptions too shortsighted, focused only on ourselves and neighbor."

This book is austere, painful, provocative, and challenging. It is concerned with the depths of eternal verities. It is a good book for any therapist of any school because it encourages self-reflection regarding one's own life and how one works with people.

On the questionable side there seems to be a certain imbalance in its content. Although there is a timely and compensatory emphasis on soul, there is a de-emphasis of ego and of the central "self archetype." The relative neglect of the self archetype suggests that Hillman sees this archetype differently from many of his colleagues, who do not see it as a "monotheistic model" but as a basic structure of the psyche that presides over the birth of the ego and regulates its development from cradle to grave.

This book may well be controversial even within the Jungian community from which it originates. However, if it incites reflection and open-eyed review of our condition and options, its purpose will be achieved.

ROSS L. HAINLINE, M.D.
New York, N.Y.

This Month's Reviewers

C.H. HARDIN BRANCH, M.D, is Program Chief and Training Officer, Santa Barbara County Mental Health Services, Santa Barbara, Calif.

IRVING PHILIPS, M.D., is Director of Child and Adolescent Psychiatry and Professor of Psychiatry in Residence, Langley Porter Neuropsychiatric Institute, University of California, San Francisco, Calif.

EVELYN S. MYERS is Managing Editor of the *Journal* and Coordinator, Psychiatric Care Insurance Coverage, APA, Washington, D.C.

JOSEPH M. GREEN, M.D., is Medical Director for Children's Services, La Frontera Center, Tucson, Ariz.

JAMES W. DYKENS, M.D., is Director, Division of Mental Health, New Hampshire Department of Health and Welfare, Concord, N.H.

PAUL N. GRAFFAGNINO, M.D., is Director, Child Psychiatric Services, Institute of Living, Hartford, Conn.

WILLIAM A. HUNT, Ph.D., is Professor Emeritus of Psychology, Loyola University of Chicago, Ill.

MAX FINK, M.D., is Professor of Psychiatry, Health Sciences Center, State University of New York at Stony Brook, N.Y.

BENJAMIN J. SADOCK, M.D., is Professor of Psychiatry, Director of the Division of Group Process, and Director of Continuing Education in Psychiatry, New York Medical College, New York, N.Y.

FRANK KLINE, M.D., is Associate Director, Adult Psychiatric Outpatient Clinic, Los Angeles County-University of Southern California Medical Center, Los Angeles, Calif.

EDWIN ROBBINS, M.D., is Clinical Professor of Psychiatry, New York University School of Medicine, New York, N.Y.

CLAYTON PETTIPIECE, M.D., is Director, Immanuel Community Mental Health Center, Omaha, Neb.

JAMES A. DAVIS, M.D., is Assistant Professor of Psychiatry, Nebraska Psychiatric Institute, University of Nebraska Medical Center, Omaha, Neb.

EDWARD J. CARROLL, M.D., is Clinical Professor of Psychiatry, University of Miami, Fla.

ROBERT VOSBURG, M.D., is Associate Professor of Psychiatry, Dartmouth Medical School, Hanover, N.H.

ROSS L. HAINLINE, M.D., is in private practice in New York, N.Y.

Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Specialty Board Review: Psychiatry, *by Robert W. Atkins, M.D. New York, N.Y., Arco Publishing Co., 1976, 144 pp., $12.00 (paper).*

Black Child Care: How To Bring Up a Healthy Black Child in America, *by James P. Comer, M.D., and Alvin F. Poussaint, M.D. New York, N.Y., Simon and Schuster, 1975, 394 pp., $9.95.*

Clinical Hypnosis: Principles and Applications, *by Harold B. Crasilneck, Ph.D., and James A. Hall, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1975, 324 pp., $19.50.*

A Study of Abortion in Primitive Societies: A Typological, Distributional, and Dynamic Analysis of the Prevention of Birth in 400 Preindustrial Societies, revised ed., *by George Devereux. New York, N.Y., International Universities Press, 1976, 397 pp., $15.00.*

Psychopharmacogenetics, *edited by Basil E. Eleftheriou. New York, N.Y., Plenum Press, 1975, 468 pp., $28.00.*

Current Developments in Psychopharmacology, Vol. 2, *edited by Walter B. Essman and L. Valzelli. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1976, 248 pp., no price listed.*

New Perspectives in Schizophrenia, *edited by Alistair Forrest, M.D., and James Affleck, M.B. Edinburgh, Scotland, Churchill Livingstone (New York, N.Y., Longman, distributor), 1975, 251 pp., $18.00.*

Childhood Psychopathology and Adult Psychoses, *by Thomas Freeman. New York, N.Y., International Universities Press, 1976, 286 pp., $16.50.*

Emotional Problems of Adolescents, 3rd ed., *by J. Roswell Gallagher, M.D., and Herbert I. Harris, M.D. New York, N.Y., Oxford University Press, 1976, 199 pp., $8.95.*

Depression: Behavioral, Biochemical, Diagnostic and Treatment Concepts, *edited by Donald M. Gallant, M.D., and George M. Simpson, M.D. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1976, 334 pp., no price listed.*

Psychology Versus Metapsychology: Psychoanalytic Essays in Memory of George S. Klein. Psychological Issues Monograph 36, *edited by Merton M. Gill and Philip S. Holzman. New York, N.Y., International Universities Press, 1976, 370 pp., $17.50.*

Clinical Behavior Therapy, *by Marvin R. Goldfried and Gerald C. Davison. New York, N.Y., Holt, Rinehart and Winston, 1976, 292 pp., no price listed.*

How To Understand and Analyze Your Own Dreams, *by Louis S. Gottschalk, M.D. New York, N.Y., Vantage Press, 1975, 93 pp., $5.95.*

Drugs in Combination with Other Therapies, *edited by Milton Greenblatt, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1975, 188 pp., $14.00.*

Alcohol Intoxication and Withdrawal: Experimental Studies II. Advances in Experimental Medicine and Biology, Vol. 59, *edited by Milton M. Gross. New York, N.Y., Plenum Press, 1975, 653 pp., $37.50.*

Contemporary Marriage: Structure, Dynamics, and Therapy, *edited by Henry Grunebaum, M.D., and Jacob Christ, M.D. Boston, Mass., Little, Brown and Co., 1976, 485 pp., no price listed (paper).*

The Family. Fourth International Congress of Psychosomatic Obstetrics and Gynecology, Tel Aviv, October 27–November 2, 1974, *edited by Herman Hirsch, M.D. New York, N.Y., S. Karger, 1975, 560 pp., $71.25.*

Stress Response Syndromes, *by Mardi Jon Horowitz, M.D. New York, N.Y., Jason Aronson, 1976, 362 pp., $15.00.*

Primal Man: The New Consciousness, *by Arthur Janov, Ph.D., and E. Michael Holden, M.D. New York, N.Y., Thomas Y. Crowell Co., 1975, 522 pp., $10.00.*

Letters, Vol. 2: 1951–1961. Bollingen Series XCV, *by C.G. Jung; selected and edited by Gerhard Adler with Aniela Jaffé; translated by R.F.C. Hull. Princeton, N.J., Princeton University Press, 1976, 638 pp., $20.00.*

No Single Thread: Psychological Health in Family Systems, *by Jerry M. Lewis, M.D., W. Robert Beavers, M.D., John T. Gossett, Ph.D., and Virginia Austin Phillips. New York, N.Y., Brunner/Mazel, 1976, 260 pp., $13.95.*

Current Psychiatric Therapies, Vol. 15—1975, *edited by Jules H. Masserman, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1975, 300 pp., $25.00.*

Assessment of Organic Intellectual Impairment, *by John McFie. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1975, 161 pp., $12.00.*

Open Family Living: A New Approach for Enriching Your Life Together, *by Dr. Thomas C. McGinnis and John W. Ayres. Garden City, N.Y., Doubleday & Co., 1976, 366 pp., $8.95.*

Vulnerability, Coping, and Growth from Infancy to Adolescence, *by Lois Barclay Murphy and Alice E. Moriarty. New Haven, Conn., Yale University Press, 1976, 452 pp., $20.00.*

College Programs for Paraprofessionals, *compiled by the New Human Services Institute, Queens College. New York, N.Y., Human Sciences Press (Behavioral Publications), 1975, 135 pp., $9.95.*

Diet Away Your Stress, Tension, and Anxiety: The Fructose Diet Book, *by J. Daniel Palm, Ph.D. Garden City, N.Y., Doubleday & Co., 1976, 213 pp. $6.95.*

The Modernization of Sex: Havelock Ellis, Alfred Kinsey, William Masters and Virginia Johnson, *by Paul Robinson. New York, N.Y., Harper & Row, 1976, 195 pp., $8.95.*

Understanding Human Behavior for Effective Police Work, *by Harold E. Russell and Allan Beigel. New York, N.Y., Basic Books, 1976, 293 pp., $13.95.*

A New Language for Psychoanalysis, *by Roy Schafer. New Haven, Conn., Yale University Press, 1976, 385 pp., $20.00.*

Manual of Psychiatric Therapeutics: Practical Psychopharmacology and Psychiatry, *edited by Richard I. Shader, M.D. Boston, Mass., Little, Brown and Co., 1975, 321 pp., no price listed (spiral bound).*

The Impaired Physician: An Interpretive Summary of the AMA Conference on the Disabled Doctor: Challenge to the Profession, April 11–12, 1975, *by E.M. Steindler. Chicago, Ill., American Medical Association Department of Mental Health, 1976, 54 pp., no price listed (paper).*

Religion and the Unconscious, *by Ann and Barry Ulanov. Philadelphia, Pa., Westminster Press, 1975, 280 pp., $12.00.*

Brain, Behaviour and Drugs: Introduction to the Neurochemistry of Behaviour, *by David M. Warburton. New York, N.Y., John Wiley & Sons, 1975, 252 pp., $18.50.*

Guiding the Psychological and Educational Growth of Children, *by Jerry W. Willis, Ph.D., Jeane Crowder, Ph.D., and Joan Willis, M.S.W. Springfield, Ill., Charles C Thomas, 1976, 208 pp., $11.50.*

Between Survival and Suicide, *edited by Benjamin B. Wolman and Herbert H. Krauss. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1976, 185 pp., $15.95.*

Schizophrenia: A Multinational Study. A Summary of the Initial Evaluation Phase of the International Pilot Study of Schizophrenia. Public Health Papers 63, *by the World Health Organization. Geneva, Switzerland, WHO (New York, N.Y., Q Corp., distributor), 1975, 150 pp., 56 Swiss francs (paper).*

The Crisis of the Community College: Second Best, *by L. Steven Zwerling. New York, N.Y., McGraw-Hill Book Co., 1976, 373 pp., $10.00.*

Position Statement on Mental Health Representation in Governance and Staffing of Health Systems Agencies

This statement was approved by the Assembly of District Branches at its November 4–5, 1975, meeting and by the Board of Trustees at its December 5–6, 1975, meeting, upon recommendation of the Council on National Affairs. It was prepared by the Task Force on Comprehensive Health Planning.[1]

THE NATIONAL HEALTH PLANNING and Resources Development Act of 1974 (Public Law 93-641) establishes at the local, state, and national levels organizational entities that will govern the planning, development, and allocation of all health resources to come. The objectives of the legislation are to develop a more rational system that can equitably, efficiently, and effectively utilize scarce health resources. The legislative guidelines call for increased services to underserved populations, consolidation of multilevel services in order to improve service accessibility and availability, training and redistribution of health manpower, development of mechanisms for cost containment and quality assurance, development of preventive services, and control over capital construction costs.

The House Interstate and Foreign Commerce Committee report on the health planning legislation (1) establishes legislative intent that health planning include activities such as mental health planning and that expertise in this area be represented to the maximum possible extent. It is essential that mental health not be regarded, for the purposes of this legislation, as a special interest. It must be viewed as a primary component of total health planning and service delivery and thus of central importance and focus to the development of Health Systems Agencies and the other organizational elements called for under this legislation. It is vital that persons with mental health knowledge, experience, and capability be represented on the governing bodies of Health Systems Agencies and the State Health Coordinating Councils that will govern the State Health Planning and Development Agencies. In addition, staff with experience and capability in the field of mental health planning must be part of the total staff of each organizational element required by this legislation.

The field of mental health, broadly defined, includes the areas of emotional disturbance and stress, mental disabilities, substance abuse, and developmental disabilities. Since 1963, with the passage of the community mental health centers legislation, mental health systems at local, state, and national levels have concerned themselves with developing organized systems for health care delivery and planning that have been directed at the very objectives now enunciated and espoused under the present legislation, i.e., increased services to the underserved, development of primary prevention programs, improved availability and accessibility of services through consolidation of programs, expanded utilization and training of manpower, utilization review and cost containment, and the integral involvement of consumers in planning and development of mental health programs. Thus the mental health system in the United States probably has the greatest degree of experience of any organized health delivery system in actually dealing with the issues keyed to the success of this legislation.

Implicit in the development of mental health programs has been the attempt to approach the individual as well as the development of health care services in a holistic manner—which includes consideration of essential environmental factors. Thus mental health programs have been intimately involved and intertwined in the delivery of physical health services. It is estimated that anywhere from 50% to 70% of individuals presenting with an initial presumptive diagnosis of physical illness are, in fact, suffering from primary or secondary emotional problems. The development of a mental health component generic to the delivery of all health services has become recognized as an essential element of any health care delivery system. Studies conducted in organized group health programs have shown the positive cost benefits as well as the patient benefits that can be achieved in incorporating a mental health orientation into the overall health planning and delivery process (2, 3).

On the basis of cost of services and numbers of persons receiving health services, it is essential that there be mental health representation both in governance and staffing. One of every four persons in the United States receives health services for the treatment of a mental health problem (4). If we consider physical health services in which there are primary or secondary mental health problems, this figure would be doubled or tripled. Thirty percent of hospital beds utilized in the United States are for mental health conditions (4–6).

The cost of direct health services for the fiscal year 1975 was $118 billion. This represents a figure approaching one-tenth of the gross national product (7). Fourteen percent was expended for direct mental health services (4). If the cost of treatment services for substance abuse and developmental

[1]The Task Force on Comprehensive Health Planning included Robert L. Williams, M.D., chairperson, James Harris, M.D., Marvin E. Perkins, M.D., Edward G. Long, M.D., George F. Wilson, M.D., Howard Gurevitz, M.D., and Falk Fellows S. Charles Schulz, M.D., and John O'Neal, M.D.

disabilities were added, this figure would be doubled. In addition, indirect costs due to factors such as loss of productivity and income, increased social dependency, and crime and delinquency have been estimated to increase direct service expenditures by 100%. Thus in 1975, it can be conservatively surmised that the United States spent approximately $100 billion for direct services and indirect costs related to mental disabilities, emotional disturbances, alcoholism, drug abuse, and developmental disabilities.

The field of mental health has taken a position and orientation by interfacing with other human services such as education, corrections, and social welfare, which provide vital opportunities for early detection and intervention in situations that can lead to mental, physical, and emotional disturbances. These interfaces place the mental health system in a very advantageous position to deal knowledgeably with the planning of health services and programs that are community oriented, geared toward prevention and early intervention of disabilities, emphasize ambulatory care and deinstitutionalization, and have the capacity to provide services to the chronically ill.

For the foregoing reasons, we urge that there be mental health representation in the governance of each jurisdictional level called for under this legislation. Such representation on boards of governance must reflect the essential quality of mental health planning to the entire field of health planning and service delivery. It must also be adequate and in proportion to the time, money, and experience invested in these mental health efforts.

Furthermore, staff with demonstrated capability and expertise in dealing with matters related to mental health planning should be available to these bodies. It is recommended that at least one staff person for each health service area be specially qualified through experience and training in the field of mental health planning and program development.

These recommendations are forwarded in light of Congressional intent that both mental health planning activities and expertise be an integral part of total health planning and service delivery.

REFERENCES

1. US House of Representatives, Committee on Interstate and Foreign Commerce: Report on National Health Policy, Planning, and Resources Development Act of 1974. Washington, DC, Committee on Interstate and Foreign Commerce, Sept 26, 1974
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3. Follette W, Cummings NA: Psychiatric services and medical utilization in a prepaid health plan setting. Med Care 5:25–35, 1967
4. National Institute of Mental Health, Work Group on Health Insurance: Insurance for mental health: trends in the delivery and financing of mental illness services in the United States (draft document). Rockville, Md, NIMH, November 1974
5. Reed LS, Myers ES, Scheidemandel PL: Health Insurance and Psychiatric Care: Utilization and Cost. Washington, DC, American Psychiatric Association, 1972
6. American Psychiatric Association, Committee on Financing Mental Health Care: Adequate mental health coverage under national health insurance: an APA position statement with supporting data (draft document). Washington, DC, APA, 1975
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Position Statement on the Quality of Life

This statement was approved by the Board of Trustees at its December 5–6, 1975, meeting upon recommendation of the Council on National Affairs. It was prepared by the Task Force on Ecopsychiatric Data Base.[1]

THE MEDICAL specialty of psychiatry is concerned with psychosocial issues as they impinge on individual growth and development and the achievement of positive mental health. Accordingly, psychiatry cannot be relieved of sharing responsibility with other human services for advancing understanding and formulating plans to eradicate biological, psychosocial, economic, and environmental conditions that are detrimental to achieving an optimal quality of life for all individuals.

[1]The Task Force on Ecopsychiatric Data Base included Aristide H. Esser, M.D., acting chairperson, Ronald Chew, M.D., Alfonso Paredes, M.D., Edward Norman, M.D., and Marshall Belaga, M.D., Falk Fellow. (Jay Shurley, M.D., chairperson, was on sabbatical leave at the time this statement was formulated.)

Position Statement on Desegregation of Hospitals for the Mentally Ill and Retarded

This statement, a revision of a statement approved in December 1963, was approved by the Assembly of District Branches at its November 4–5, 1975, meeting and by the Board of Trustees at its December 5–6, 1975, meeting. The revision was recommended by the Council on National Affairs.[1].

THE AMERICAN PSYCHIATRIC ASSOCIATION is in favor of desegregation of all hospitals for the mentally ill and retarded. This statement is offered as contributory to the national will to eliminate legal and social impediments to the extension of all services to all citizens. The acceptance of this principle and its translation into practice would remove the need to duplicate facilities to accommodate segregation. It would release all available resources in support of a wider range of treatment services for the benefit of all mentally ill citizens.

[1]The Council on National Affairs included Harold M. Visotsky, M.D., chairperson, James M. Bell, M.D., vice-chairperson, Hiawatha Harris, M.D., Jean Shioda Bolen, M.D., Frank M. Ochberg, M.D., Hector Jaso, M.D., Assembly of District Branches liaison, Esther P. Roberts, M.D., observer-consultant, and Marshall Belaga, M.D., Ezra Griffith, M.D., and Russell Phillips, M.D., Falk Fellows.

Position Statement on Administration of Psychiatric Facilities

This statement was approved by the Assembly of District Branches at its November 4–5, 1975, meeting and by the Board of Trustees at its December 5–6, 1975, meeting, upon recommendation of the Council on Mental Health Services. It was prepared by the Council on Mental Health Services.[1]

STANDARD 15 of the *Standards for Psychiatric Facilities*, as amended in 1974, is revised by adding the following at the end of the first paragraph at the top of page 11:

> In a psychiatric facility where the chief administrative officer is a non-medical administrator, conflicts which develop between the administrator and the physician regarding the medical-psychiatric management of patients shall be adjudicated by appropriate peer review mechanisms described in the Bylaws of the facility.

It is recognized that there are facilities with an orientation toward prevention of mental illness and disease as well as maintenance of mental health. In such settings, mental health professionals other than psychiatrists may retain the primary responsibility for programs, using psychiatrists for consultation as needed, always taking into consideration the welfare of the patient.

In any psychiatric facility where treatment of mental illness or disease is offered, the physician-psychiatrist shall retain the ultimate responsibility for the admission, diagnosis, treatment, rehabilitation, and discharge of patients under his or her care.

Regular review and evaluation of the mental health programs, policies, and services provided the public should be accomplished by professionals from the disciplines of psychiatry, psychology, social work, nursing, and other qualified mental health professions, with adequate representation from the public consumers of such services. The professionals engaging in such review and evaluation shall have recognized stature in their own professions and have no fiduciary relationship with the system being evaluated.

APA, through existing mechanisms, will provide the drafters of the regulations accompanying Public Law 94-63 with these concepts for inclusion in such regulations.

[1]The Council on Mental Health services included James C. Johnson, M.D., chairperson, Mildred Mitchell-Bateman, M.D., vice-chairperson, Israel Zwerling, M.D., Bruce W. Alspach, M.D., J.M. Stubblebine, M.D., Aubrey Dent, M.D., observer-consultant, and William B. Spriegel, M.D., Assembly of District Branches liaison.

Position Statement on the Training of Minority Psychiatrists

This statement, a revision of a previously approved statement (published in the August 1971 issue), was approved by the Assembly of District Branches at its November 4–5, 1975, meeting and by the Board of Trustees at its December 5–6, 1975, meeting. The revision was recommended by the Council on National Affairs.[1] *The original statement was recommended by the Committee on Medical Education.*[2]

ALL RESIDENCIES must provide treatment facilities; in these facilities the proportion of minority patients should reflect that of the surrounding community and/or represent its ethnic, socioeconomic, and cultural balance.

The future of stipends for psychiatric residents is uncertain, but if they are to be continued, minority residents should be favored. If there are to be loans for psychiatric residents, the lowest possible interest rate should be granted to minority residents, and the interest rate should be lower for those with greater need.

The Assembly of District Branches should study the situation in the district branches to see what effort they are making to deal with minority problems. The Assembly of District Branches may recommend that a specific committee on minority problems be set up within each district branch.

An annotated bibliography of the literature on the treatment of minorities, including papers by both minority and other psychiatrists, should be developed by APA, and the Association should determine how such a bibliography might best be distributed to its membership.

APA must powerfully encourage and support systematic research into such issues as the treatment, demography, sociology, etc., of minorities. These are the same questions that have been studied extensively in whites. The Council on Research and Development should determine if private or public funds are available for such research, because the problem of funding both minority and other research workers is crucial for the future of the Association. While most minority psychiatrists are currently on the "firing line," a few must be protected so that they can do research. This might be done by the National Institute of Mental Health and/or foundations developing career research and teacher funds for minority psychiatrists.

The American Board of Psychiatry and Neurology should investigate residency training programs to see if they are deficient in training minorities. If deficiencies are found, the Residency Review Committee should recommend specific changes, with further certification of the programs dependent upon the implementation of the changes.

The written examination of the Board should include a significant number of questions regarding minority psychiatry, and all oral examiners for the Board should include such questions in their examinations.

An effective residency training program should have an ethnically balanced faculty. Certification should be dependent upon proof of an ethnic balance or upon the presentation of a schedule for the program's achieving such a balance.

It is essential that APA express its sense of urgency in this matter. No unnecessary bureaucratic machinery should slow the process of implementing the recommendations as they are accepted.

[1]The Council on National Affairs included Harold M. Visotsky, M.D., chairperson, James M. Bell, M.D., vice-chairperson, Hiawatha Harris, M.D., Jean Shioda Bolen, M.D., Frank M. Ochberg, M.D., Hector Jaso, M.D., Assembly of District Branches liaison, Esther P. Roberts, M.D., observer-consultant, and Marshall Belaga, M.D., Ezra Griffith, M.D., and Russell Phillips, M.D., Falk Fellows.

[2]The Committee on Medical Education then included Robert J. Stoller, M.D., chairperson, Jeanne Spurlock, M.D., James I. Mathis, M.D., S. Mouchly Small, M.D., Henry Coppolillo, M.D., Manuel Pearson, M.D., and Robin C.A. Hunter, M.D.

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in New Orleans, La., Jan. 19–20, 1976.

PSYCHIATRY

Abbas, Mahmoud Fouad, M.D., Baltimore, Md.
Abernethy, Robert Sidney III, M.D., Dover, Mass.
Abrams, Richard Steven, M.D., Chicago, Ill.
Allen, Robert Edwin, M.D., Long Beach, Calif.
Anderson, Hans Pierce, M.D., LaCrosse, Wis.
Asher, John David, M.D., Palo Alto, Calif.
Asnes, Daniel Paul, M.D., Belmont, Mass.
Atwood, Sue C., M.D., Memphis, Tenn.

Barquist, Walter Eric, M.D., Washington, D.C.
Barry, Carole Joyce, M.D., Rochester, N.Y.
Barry, Ronald Makepeace, M.D., Baltimore, Md.
Beall, James Harvey, M.D., Athens, Ga.
Bender, Estelle P., M.D., New York, N.Y.
Berger, Dirk Marc, M.D., New York, N.Y.
Berger, Joseph, M.D., Willowdale, Ont., Canada
Berkowitz, David V., M.D., Cincinnati, Ohio
Blake, Carlton H., M.D., Roosevelt, N.Y.
Bloom, Lee Benjamin Brown, M.D., Beverly Hills, Calif.
Brown, Edward M., M.D., Providence, R.I.

Carino, Aurora L., M.D., Wingdale, N.Y.
Cavanaugh, Gary Lynn, M.D., Stockton, Calif.
Cerruti, Piero John, M.D., San Mateo, Calif.
Colligan, Joseph F., M.D., Chestnut Hill, Mass.

Darsey, Susan B., M.D., Houston, Tex.
Dinaburg, Daniel, M.D., San Francisco, Calif.
Dolgoff, Robert Kenneth, M.D., Berkeley, Calif.
Dresnin, Sandor George, M.D., Woodland Hills, Calif.

Evans, Jerome Edward, M.D., West Hartford, Conn.
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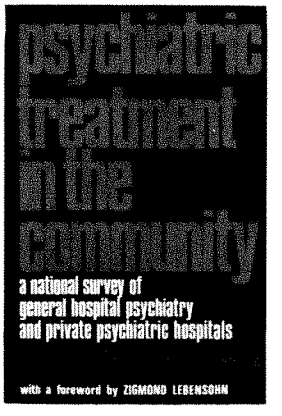
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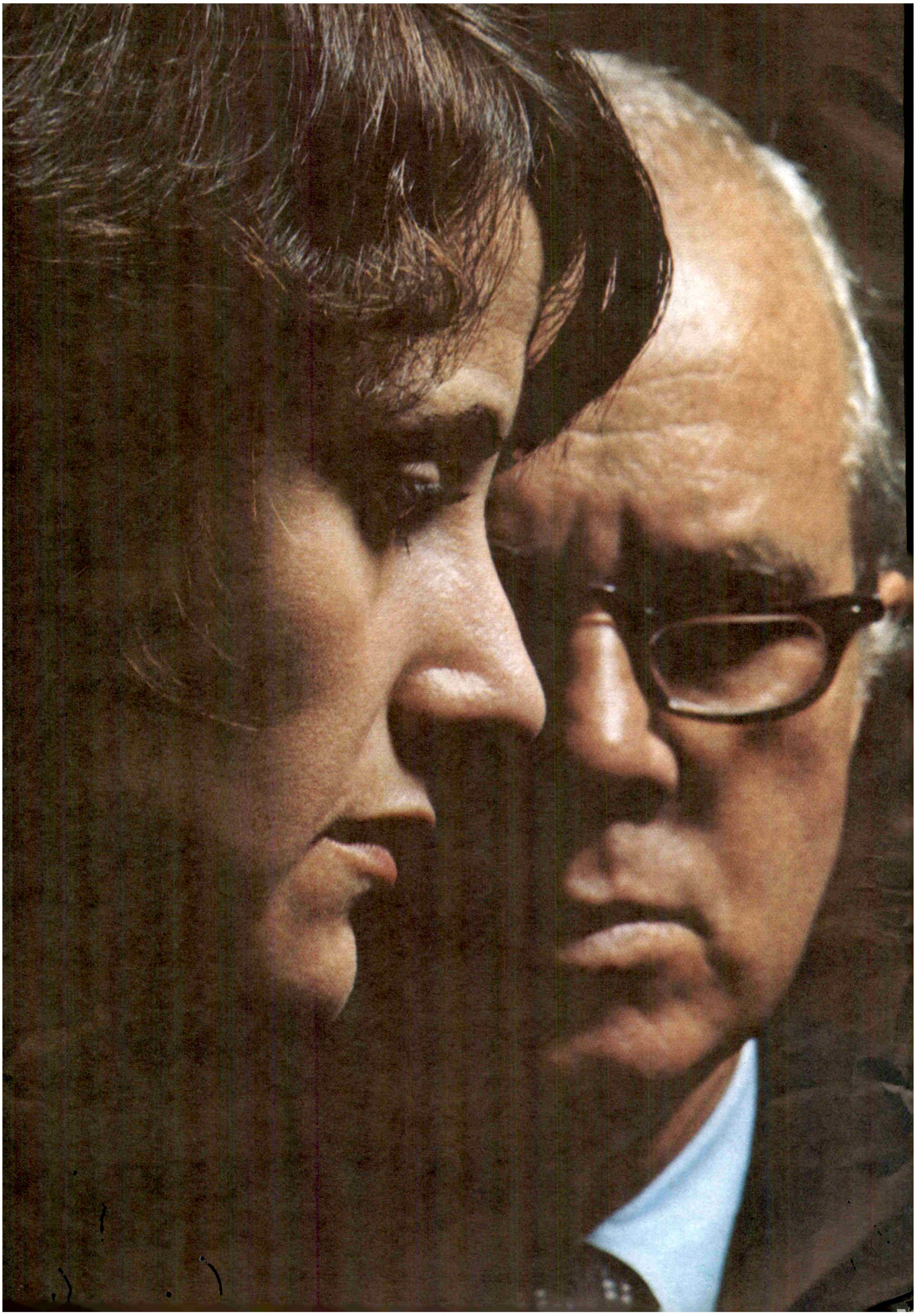
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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Do not give concomitantly with MAOI drugs because hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. Allow minimum of 14 days between therapies, then initiate therapy with TRIAVIL cautiously, with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given with guanethidine or similarly acting compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, particularly in high doses, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. Caution patients performing hazardous tasks, such as operating machinery or driving motor vehicles, that drug may impair mental and/or physical abilities. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Side effects may be any of those reported with phenothiazine drugs: extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and that the full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; hypnotic effects; pigmentary retinopathy; corneal and lenticular pigmentation; occasional lassitude, muscle weakness, mild insomnia. Other adverse reactions reported with various phenothiazine compounds include blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); liver damage (jaundice, biliary stasis); grand mal convulsions; cerebral edema; polyphagia; photophobia; skin pigmentation; and failure of ejaculation.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness; weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

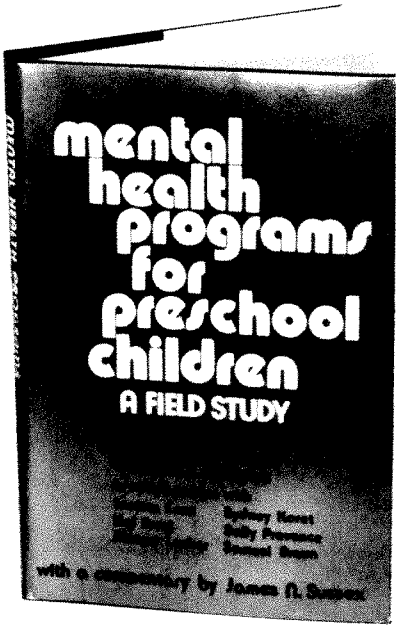
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Valium® (diazepam)

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can relieve psychoneurotic anxiety and its associated depressive symptoms

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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Mysoline® (primidone)

may be the start of a better life for the epileptic

initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

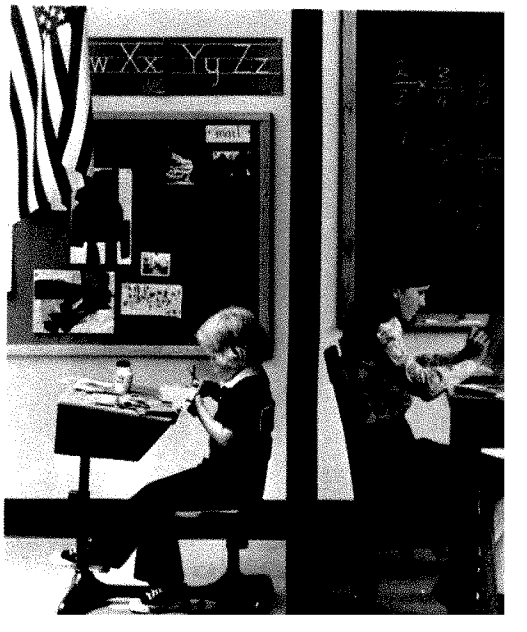
In patients already receiving other anticonvulsants, MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (teaspoonful) contains 250 mg. of primidone, in bottles of 8 fluid-ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. *37*:49 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. *47*:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. *68*:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline [primidone] for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards — used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate—As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail—A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

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Mysoline®
[primidone]

Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

New APA Task Force Reports

Behavior Therapy in Psychiatry

This comprehensive report reviews the historical development of behavior therapy, its efficacy for the treatment of psychiatric problems, its current forms and uses as well as potential abuses, and its relationship to dynamic psychiatry. Approved for publication by the APA Council on Research and Development, the Report was prepared by a Task Force comprising Lee Birk, M.D., John Paul Brady, M.D., Alan J. Rosenthal, M.D., W. Douglas Skelton, M.D., Joseph B. Stevens, M.D., and Consultants Stephanie B. Stolz, Ph.D., Joseph V. Brady, Ph.D., Arnold A. Lazarus, Ph.D., James J. Lynch, Ph.D., and Edwin J. Thomas, Ph.D.

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The Present and Future Importance of Patterns of Private Psychiatric Practice in the Delivery of Mental Health Services

This report marks the first formal statement the Association has ever issued delineating the vital role of its private practitioner members in the delivery of mental health services in the U.S. Contains sections on patterns of private practice, the economics of it, the impact of third party payments on it, its relation to the public sector, and an assessment of the overall contribution of the private sector to the treatment of mental illness. Stresses the theme that any national system for the delivery of mental health services must be a "balanced mix" of both the private and public sectors, each reinforcing the other. Approved for publication by the Council on Mental Health Services, the Report was prepared by a Task Force comprising Drs. Ewald Busse, Rogers J. Smith, Reed S. Andrus, Winston Cochran, Albert A. Lorenz, Robert L. Leopold, Louis W. Nie, and Consultants Alan I. Levenson, Zigmond M. Lebensohn, Walter E. Barton, and Mr. Robert L. Robinson.

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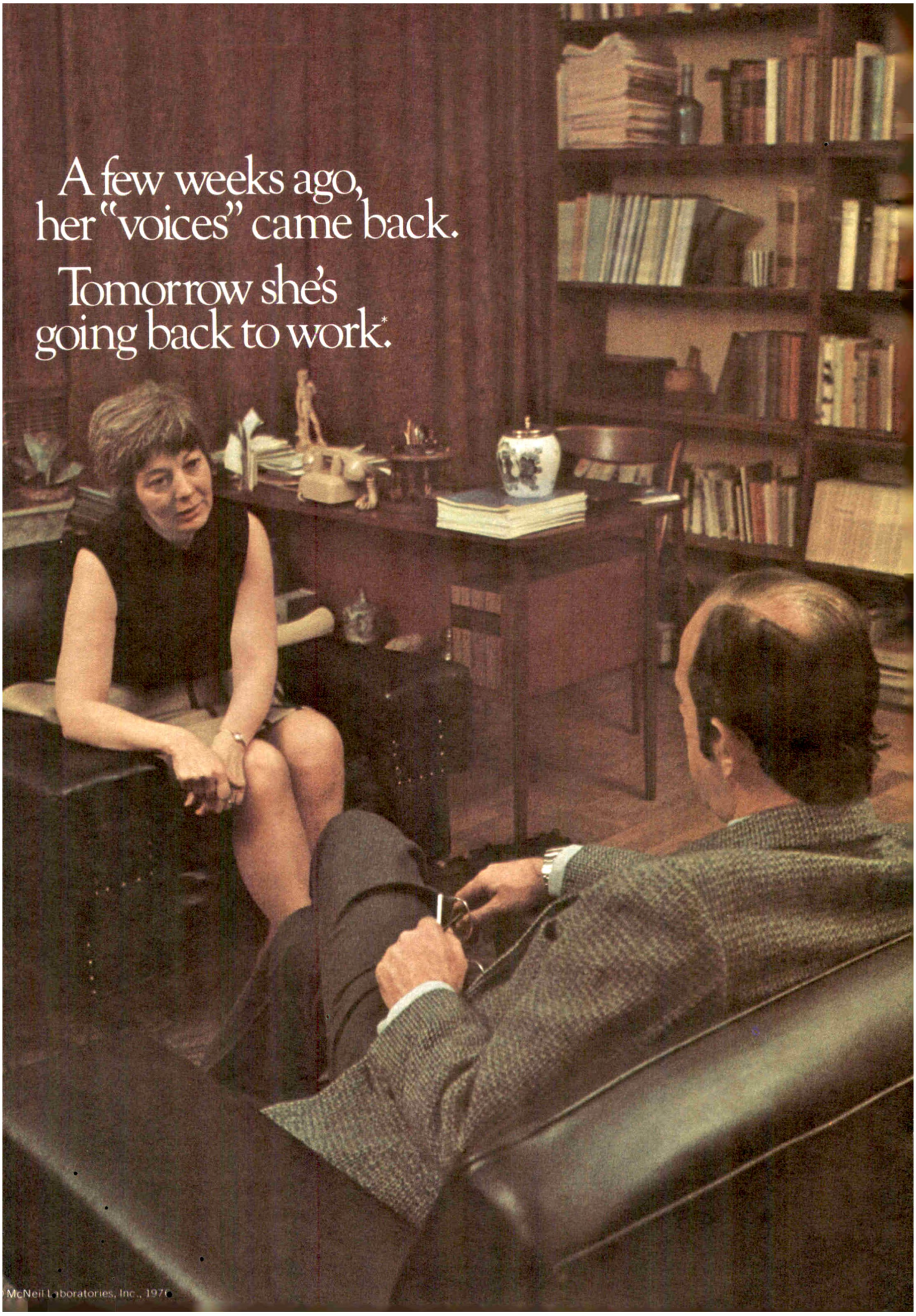
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Haldol®
(haloperidol)
tablets/concentrate/injection

For prompt control with minimal risk of hypotension or oversedation

Highly effective in a wide range of psychotic symptoms[1-7]
such as hallucinations, delusions, suspiciousness, hostility, mania, psychomotor agitation, etc., in both acute and chronic disorders.

Helps keep patients functioning and avoid hospitalization.[1-3]
HALDOL haloperidol usually leaves most patients alert and responsive, able to function at work and at home without impairment, and more amenable to other therapeutic measures.

Permits aggressive titration to effective dosage levels[1,4]
up to 100 mg/day orally (with minimal risk of usual troublesome reactions) to achieve optimal response when patient is inadequately controlled at lower dosage...to help you rapidly control and stabilize new patients, promptly regain control during periods of exacerbation.

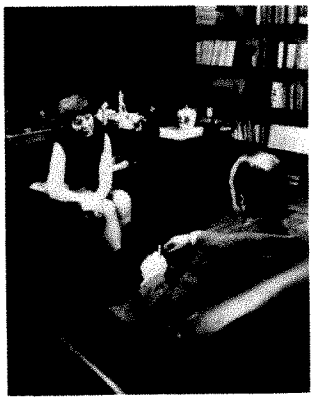
Common side effects easily controlled.[1,2,4]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Reduces risk of certain troublesome reactions.[1,2,4-6]
Transient hypotension occurs rarely and severe orthostatic hypotension has not been reported. Marked sedation is rare, although some instances of drowsiness have been reported. In addition, with chronic use, it is unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes...and has minimal effect on renal function.

Please turn page for information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions.

Important: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.



Haldol® (haloperidol)

tablets/concentrate/injection

For prompt control of psychotic symptoms with minimal risk of hypotension or oversedation

A Dosage Form for Every Need:

5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.

A tasteless, odorless, colorless liquid concentrate for better patient acceptability: 2 mg. per ml.

A rapid-acting injection for psychiatric emergencies: 5 mg. per ml., with 1.8 mg. methylparaben and 0.2 mg. propylparaben per ml., and lactic acid for pH adjustment to 3.4±0.2.

Indications: HALDOL haloperidol is indicated for use in the management of manifestations of psychotic disorders.

It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9/74

References:

1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. **2.** Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. **3.** Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. **4.** Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. **5.** Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. **6.** Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. **7.** Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973.

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In clinically significant depression



TABLETS, 5 mg and 10 mg

Vivactil®*

(Protriptyline HCl|MSD)

*** When you want an antidepressant that is characteristically nonsedating for the patient under close medical supervision (Symptoms such as anxiety or agitation may be aggravated.)**

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information please see following page.

In clinically significant depression

TABLETS, 5 mg and 10 mg

Vivactil®

(Protriptyline HCl | MSD)

Contraindications: Known hypersensitivity; acute recovery phase following myocardial infarction. Should not be given concomitantly with an MAOI; hyperpyretic crises, severe convulsions, and deaths have occurred in patients receiving tricyclic antidepressant and MAOI drugs simultaneously. When it is desired to substitute protriptyline HCl for an MAOI, a minimum of 14 days should be allowed to elapse after the latter is discontinued. Protriptyline HCl should then be initiated cautiously with gradual increase in dosage until optimum response is achieved.

Warnings: May block the antihypertensive effect of guanethidine or similarly acting compounds. May impair mental and/or physical abilities required for the performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Should be used with caution in patients with a history of seizures and, because of its autonomic activity, in patients with a tendency to urinary retention or increased intraocular tension.

Tachycardia and postural hypotension may occur more frequently than with other antidepressant drugs. Should be used with caution in elderly patients and patients with cardiovascular disorders; such patients should be observed closely because of the tendency of the drug to produce tachycardia, hypotension, arrhythmias, and prolongation of the conduction time. Myocardial infarction and stroke have occurred with drugs of this class. On rare occasions, hyperthyroid patients or those receiving thyroid medication may develop arrhythmias when this drug is given.

Usage in Children: Not recommended for use in children because safety and effectiveness in the pediatric age group have not been established.

Usage in Pregnancy: Safe use in pregnancy and lactation has not been established; therefore, use in pregnant women, nursing mothers, or women who may become pregnant requires that possible benefits be weighed against possible hazards to mother and child.

Precautions: When protriptyline HCl is used to treat the depressive component of schizophrenia, psychotic symptoms may be aggravated; likewise, in manic-depressive psychosis, depressed patients may experience a shift toward the manic phase; paranoid delusions, with or without associated hostility, may be exaggerated. In any of these circumstances, it may be advisable to reduce the dose of protriptyline HCl or to use a major tranquilizing drug concurrently. Symptoms, such as anxiety or agitation, may be aggravated in overactive or agitated patients.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. May enhance response to alcohol and effects of barbiturates and other CNS depressants. Possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent administration with electroshock therapy may increase hazards of therapy; such treatment should be limited to patients for whom it is essential. Discontinue drug several days before elective surgery, if possible. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions which have not been reported with this specific drug. However, the pharmacologic similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when protriptyline HCl is administered. Protriptyline HCl is more likely to aggravate agitation and anxiety and produce cardiovascular reactions such as tachycardia and hypotension.

Cardiovascular: hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: confusional states (especially in the elderly) with hallucinations, disorientation, delusions, anxiety, restlessness, agitation; insomnia, panic, and nightmares; hypomania; exacerbation of psychosis.

Neurological: numbness, tingling, and paresthesias of extremities; incoordination, ataxia, tremors, peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns, tinnitus.

Anticholinergic: dry mouth and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilatation of the urinary tract.

Allergic: skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight), edema (general, or of face and tongue), drug fever.

Hematologic: bone marrow depression; agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of other tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets, containing 5 mg and 10 mg protriptyline HCl each, in single-unit packages of 100 and bottles of 100 and 1000.

For more detailed information, consult your MSD representative or see full prescribing information.

Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

MSD
MERCK SHARP & DOHME

PSYCHIATRISTS

Full-Time Director for the Psychosomatic-Psychiatric Liaison Service in the University of California School of Medicine, San Francisco. Will be responsible for consultative services, training of medical students, interns, residents in emotional factors of illness, postgraduate education for staff and community physicians. Will hold faculty title of Associate Professor or Professor of Psychiatry. Need administrative skills in coordinating all existing relationships between psychiatry and other clinical services, establishing training, service and research where such activities would be appropriate and do not yet exist. Qualified candidates representing minorities and women encouraged to apply.

Direct inquiries to:
Ms. Mary Asahina, Dept. Admin. Assist.
Langley Porter Neuropsychiatric Inst.
401 Parnassus Avenue
San Francisco, CA 94143

PSYCHIATRIC IN-PATIENT SERVICES HEAD

University Hospital, Indiana University School of Medicine; Indiana licensed M.D. with residency training in Psychiatry and prior academic experience in a related basic Psychiatric science and evidence of research. Salary range $22,500-$28,000 as Associate Professor of Indiana University School of Medicine with many fringe benefits including TIAA policy paid by the University; 12 month year. Teaching duties include supervision of residents of Psychiatry, medical students and other professional groups retained through the In-patient Department; in addition to responsibility for procedures, medical records, referrals, etc. for the over-all management of the Psychiatric in-patient service involving individual patient care. Qualified and interested individuals may send Vita to Dr. Hugh C. Hendrie, Chairman, Department of Psychiatry, University Hospital, 6 North, Indiana University School of Medicine, Indianapolis, Indiana 46202.

AN EQUAL OPPORTUNITY EMPLOYER
AN AFFIRMATIVE ACTION POLICY

Three Treatment Centers

Three treatment centers provide individually prescribed programs of care, education, treatment and training for children, adolescents and adults with learning disabilities, neurological impairment, mental retardation or emotional disturbance.

Advantage: the availability of a **large, trained and experienced staff,** exchanging ideas and insights in a comprehensive program of supportive environment and individualized treatment.

Advantage: **geographically separated** to provide the proper residential milieu . . . yet near enough to benefit from the combined training and experience of a wide range of staff members.

The Oaks — A residential psychiatric treatment center for emotionally disturbed children and adolescents between the ages of six and 18. Co-educational, Austin. In addition, the Center provides a Short Term Adolescent Center and a six bed intensive care unit.

The San Marcos Treatment Center — a facility with three campuses designed for individuals with psychiatric, neurological, and intellectual difficulties who have evidenced a need for a 24-hour therapeutic milieu.

The Center offers a broad spectrum of services to both mentally retarded individuals and those with psychiatric disorders. In addition to a structured therapeutic environment, individual and group psychotherapy services are available to each resident. Clinical supervision and support services are provided by the Center psychiatrists.

The Ranch Treatment Center — located in the suburbs of Austin the Center provides both long-term and intermediate tenure services to multi-handicapped male adolescents and adults whose primary diagnostic classifications are in areas of mental retardation and mental illness. All residents receive program services which may permit their early discharge to the less restrictive settings of community-based programs.

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Jackson R. Day, M.D./Medical and Psychiatric Director
James L. Boynton, M.D./Director, The Oaks Residential Treatment Center
John L. Carrick, M.D./Staff Psychiatrist
Orrie L. Forbis, Jr., M.D./Staff Psychiatrist
Other Medical Staff: Thomas F. Caldwell, D.D.S./ Patrick A. Cato, M.D./Willis M. Thorstad, M.D.



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THE BROWN SCHOOLS

ROCHE

Introducing

The unforgettable antidepressant

Endep™ 75 mg

amitriptyline HCl / Roche

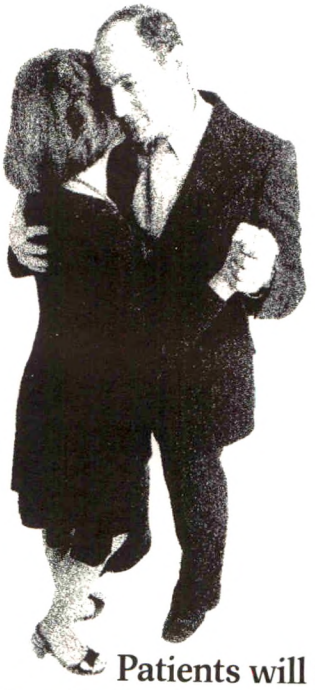
Here's what makes it unforgettable



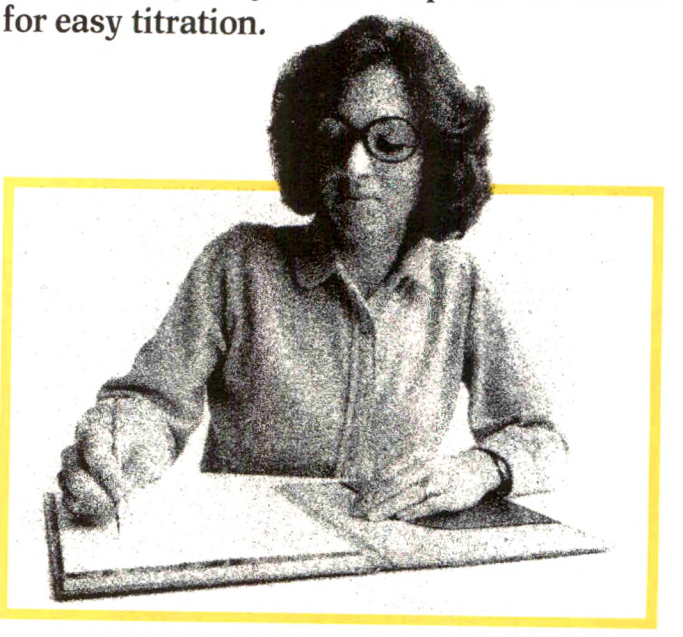
Patients are more likely to remember the single bedtime dosage of Endep (amitriptyline HCl).



You'll remember 1 or 2 tablets *h.s.*, because fewer forgotten doses produce a more consistent clinical effect, and because it is the only tricyclic antidepressant scored for easy titration.



Patients will also remember family/interpersonal relationships that they have forgotten or neglected.



In three or four weeks patients will usually remember how well they used to feel.

Endep™ 75 mg

amitriptyline HCl/Roche

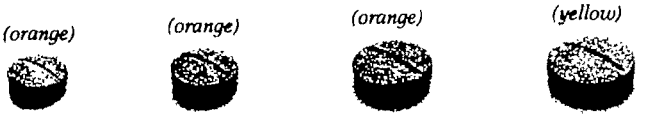
The antidepressant you can prescribe at night to work all day

Before prescribing, please consult complete product information, a summary of which appears on the following page.

Introducing Endep™

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day



10-mg scored tablets | **25-mg** scored tablets | **50-mg** scored tablets | **75-mg** scored tablets

■ Endep (amitriptyline HCl), in responsive patients, lifts mood and relieves symptoms such as anorexia, loss of interest, inability to concentrate, decreased libido or GI disturbances, often within three or four weeks after starting therapy.

■ Endep usually relieves accompanying sleep disturbances, particularly early in the treatment regimen.

■ All four dosage strengths are scored for greater flexibility and convenience in titration.

■ The 75-mg *h.s.* dose minimizes skipped doses, maximizes patient acceptance and compliance.

■ Specifying Endep/Roche assures quality plus potential economy.

Important Considerations

Should not be given concomitantly with an MAO inhibitor.

Not recommended for use during the acute recovery phase following myocardial infarction.

Contraindicated in patients with hypersensitivity to amitriptyline.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh potential benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia. purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels; increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier. *Initial Adult Dosage:* Outpatients—25 mg t.i.d.; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate—50 to 100 mg h.s., gradually increasing h.s. dose up to 150 mg/day. Hospitalized Patients—Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day. *Adolescent and Elderly Patients:* In general, 10 mg t.i.d. with 20 mg h.s. may be satisfactory for those who do not tolerate higher doses. *Maintenance Dosage:* With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg b.i.d. to q.i.d., or 10 mg q.i.d. Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg and 75-mg scored tablets—bottles of 100 and 500; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg), available singly and in trays of 10.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

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May 1976

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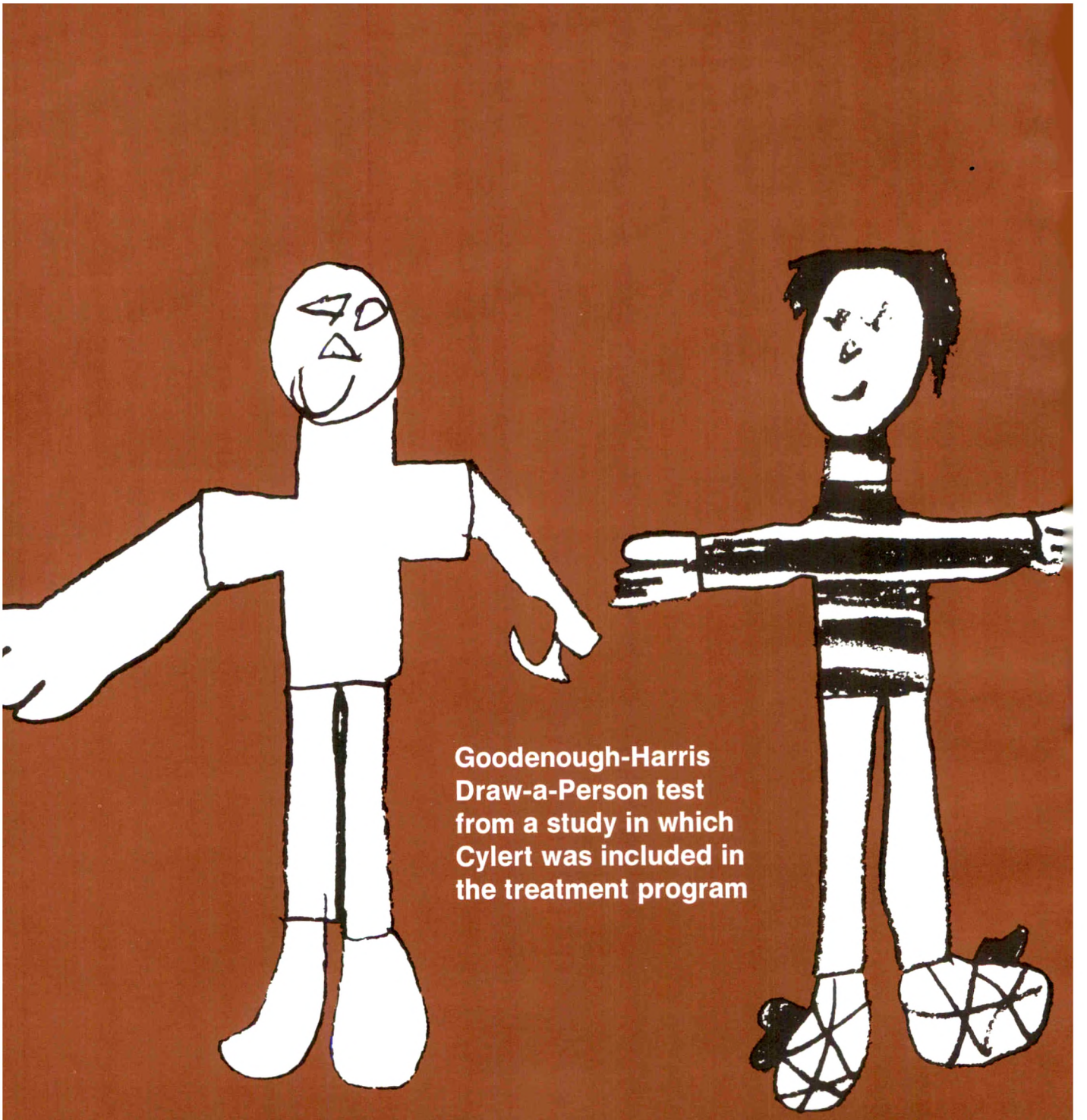
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Goodenough-Harris Draw-a-Person test from a study in which Cylert was included in the treatment program

Drawing made prior to treatment

Drawing made at week 8 during treatment

Cylert (pemoline) will not in itself "enhance learning" or resolve difficult behavioral problems. But it can increase attention span in the hyperkinetic child and reduce the impulsivity that often interferes with the learning process.

Cylert® (pemoline)

offers these benefits in a treatment program for MBD

- **Single daily dose administration**
- **Minimal cardiovascular effects**
- **Mean dosage in long-term studies remained remarkably constant**

EFFICACY

Multi-clinic study[1,2]

21 investigators from 10 states and two provinces in Canada took part in the clinical studies.

Double-blind, placebo control

413 patients were randomly assigned to Cylert or placebo groups. 238 patients met all criteria for evaluation of efficacy.

Psychological test results

Children on Cylert had significantly higher scores statistically than those on placebo on these psychological tests:

- The Wechsler Intelligence Scale for Children (WISC) and its performance IQ Sub-Component
- The Wide Range Achievement Test (WRAT) (reading and arithmetic)
- The Lincoln-Oseretsky Motor Performance Test Factor II

Overall results

Approximately two out of three patients were significantly improved by treatment with Cylert as reflected by global ratings.

1. Conners, C. K., ed., *Clinical Use of Stimulant Drugs in Children,* Excerpta Medica, 1974, p. 98.
2. Page, J. G., et al., J. Learning Disabilities, 7:498, Oct., 1974.

SAFETY

Multi-clinic study (9 weeks); safety data analyzed on 407 patients

There was no significant difference between Cylert and placebo groups in:

- Blood pressure
- Laboratory tests
- Pulse
- Neurological status

Insomnia and anorexia were the most frequently seen side effects and often improved with continuation of treatment or reduction of dosage.

Mean weight loss of 1.1 lbs. was demonstrated in the Cylert group during early weeks of treatment; long-term studies have shown that by 3-6 months, most children return to the normal rate of weight gain for their age group.

Long-term study on Cylert; up to 3 years and continuing

Mean dosage . . . remained remarkably constant.
Blood pressure . . no significant changes attributed to Cylert.
Pulse rate . . . . . no significant changes attributed to Cylert.

Laboratory examination—mild to moderate increase in transaminase (SGOT and SGPT) levels in 1-2% of patients (no clinical symptoms); levels returned to normal on withdrawal of medication.

No clinically significant abnormalities in the other tests.

Please see last page of this advertisement for Prescribing Information.

Importance of single daily dosage to the child, the parents and the teacher

For the child		For the adults
No drug in child's possession while at school	⟷	Control of medication remains with parents
Avoids situation in which child is repeatedly singled out as being "different"	⟷	Obviates need for nurse or teacher to supervise taking of mid-day doses
Helps prevent possible variations in effect caused by missed, forgotten or delayed doses	⟷	Helps assure that the prescribed dosage is being given each day



Cylert (pemoline), alone among CNS stimulants used to treat MBD, is inherently long-acting, permitting once-daily dosage.

Cylert can be taken with meals

You can prescribe Cylert a.c., p.c., or with meals. Although the speed of absorption is slightly slowed by food, the total absorption is not affected.

Dosage and administration

Cylert is given as a single oral dose each morning.

The recommended starting dose is 37.5 mg. per day. This daily dosage should be gradually increased at one-week intervals using increments of 18.75 mg. until the desired clinical response is obtained.

The mean daily effective dose ranges from 56.25 to 75 mg. per day. The maximum recommended daily dose of Cylert is 112.5 mg.

Using the recommended schedule of dose titration, significant benefits may not be seen until the third or fourth week of drug therapy. Side effects may be seen prior to optimum clinical results.

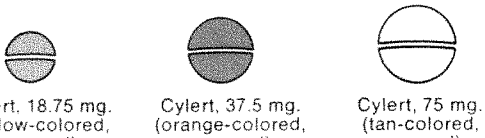
When not to use Cylert

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school-age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

The three dosage strengths of Cylert (pemoline)



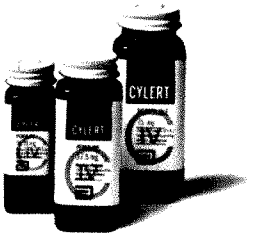
Cylert, 18.75 mg. (yellow-colored, grooved)

Cylert, 37.5 mg. (orange-colored, grooved)

Cylert, 75 mg. (tan-colored, grooved)

Tablets are actual size.

Cylert
(pemoline) C IV



Prescribing Information

Description: Cylert (pemoline) is a white, tasteless, odorless powder which is relatively insoluble (less than 1 mg/ml) in water, chloroform, ether, acetone, and benzene. In 95% ethyl alcohol, the solubility of pemoline is 2.2 mg/ml.

Actions: Cylert (pemoline) is a central nervous system stimulant. The pharmacologic activity of pemoline is similar to that of other known stimulants but with minimal sympathomimetic effects. Pemoline is structurally dissimilar from the amphetamines and methylphenidate. Although the exact mode of pharmacodynamic action is undetermined in man, pemoline has been reported to increase the rate of synthesis of dopamine in rat brain.

In human subjects, Cylert produces peak blood levels within 2-4 hours. The serum half-life is approximately 12 hours. Multiple dose studies in adults at several dose levels indicate that serum levels plateau in approximately three days. Cylert and its metabolites are primarily excreted by the kidneys with approximately 75% of an oral dose appearing in the urine within a 24-hour period. Approximately 43% of pemoline is excreted unchanged. Metabolites include pemoline dione, conjugated pemoline and mandelic acid.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: MINIMAL BRAIN DYSFUNCTION IN CHILDREN—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: Specific etiology of minimal brain dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis includes the use not only of medical but of psychological, educational, and social resources.

Characteristics commonly reported include: A chronic history of moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindication: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See PRECAUTIONS)

Warnings: Cylert is not recommended for children under six years of age since safety and efficacy in this age group have not yet been established.

Since Cylert (pemoline) and its metabolites are excreted primarily by the kidneys, caution should be observed in administering the drug to children with significantly impaired renal function.

Sufficient data on safety and efficacy of Cylert administration for periods beyond two years duration in children with minimal brain dysfunction are not yet available. Although a definite causal relationship has not been established, some temporary suppression of predicted growth pattern(i.e., weight and/or height) has been reported with the long-term use of stimulants in children. Therefore, patients requiring long-term therapy should be carefully monitored.

Drug Interactions: Interactions between Cylert and other drugs have not been studied in humans. As with most other drugs, concurrent administration with other agents, especially drugs with central nervous system activity, should be carefully monitored.

Usage in Pregnancy: Safety for use in pregnancy has not been established. Standard studies of fertility, teratology and reproduction were conducted in rats and rabbits. Daily oral doses of pemoline of 18.75 and 37.5 mg/kg beginning at conception produced no abnormalities in the fetuses and did not affect viability at birth. Further studies using similar dose levels with drug administration beginning 14 days before conception demonstrated an increased incidence of stillbirths in these animals.

Drug Dependence: Studies of the drug abuse potential of Cylert (pemoline) in primates have not demonstrated a potential for self-administration. However, the pharmacologic similarities between Cylert and other CNS stimulants with known abuse liability suggest that drug dependence of the stimulant type might occur. There have been isolated reports of transient psychotic symptoms in adults following long-term misuse of pemoline taken orally in excessive quantities. Therefore, caution should be observed in emotionally unstable patients considered to have a psychological potential for drug dependence.

Precautions: Delayed hypersensitivity reactions involving the liver have been reported in 1-2% of the patients receiving Cylert usually after several months of therapy. No clinical symptomatology has been observed, but mild to moderate increases in transaminase (SGOT and SGPT) levels have occurred in these cases. These effects appear to be completely reversible when drug treatment is discontinued. Transaminase levels should be determined periodically during therapy with Cylert to detect any such reactions.

Adverse Reactions: The most frequently reported adverse reaction with Cylert is insomnia. Insomnia has been observed prior to optimum therapeutic response and in the majority of cases was transient in nature or responded to dosage reduction. Anorexia with weight loss during the first few weeks of therapy has also been reported. With continuing therapy, a return to a normal weight curve usually occurred within three to six months. Other adverse reactions reported include stomachache, skin rash, irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations. Mild adverse reactions appearing early in treatment often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage reduction or discontinuation should be considered.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning. The recommended starting dose is 37.5 mg per day. This daily dosage should be gradually increased at one week intervals using increments of 18.75 mg until the desired clinical response is obtained. The mean daily effective dose ranges from 56.25 to 75 mg per day. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally to determine if behavioral symptoms sufficient to require continuing therapy recur.

Overdosage: Cylert overdosage has been reported to produce symptoms of tachycardia, hallucinations, agitation, or restlessness. The treatment of acute massive overdosage with pemoline is essentially the same as that for overdosage with any drug having CNS stimulatory effects. Management is largely symptomatic and may include induction of emesis, gastric lavage or other measures as appropriate.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (**NDC** 0074-6025-13)

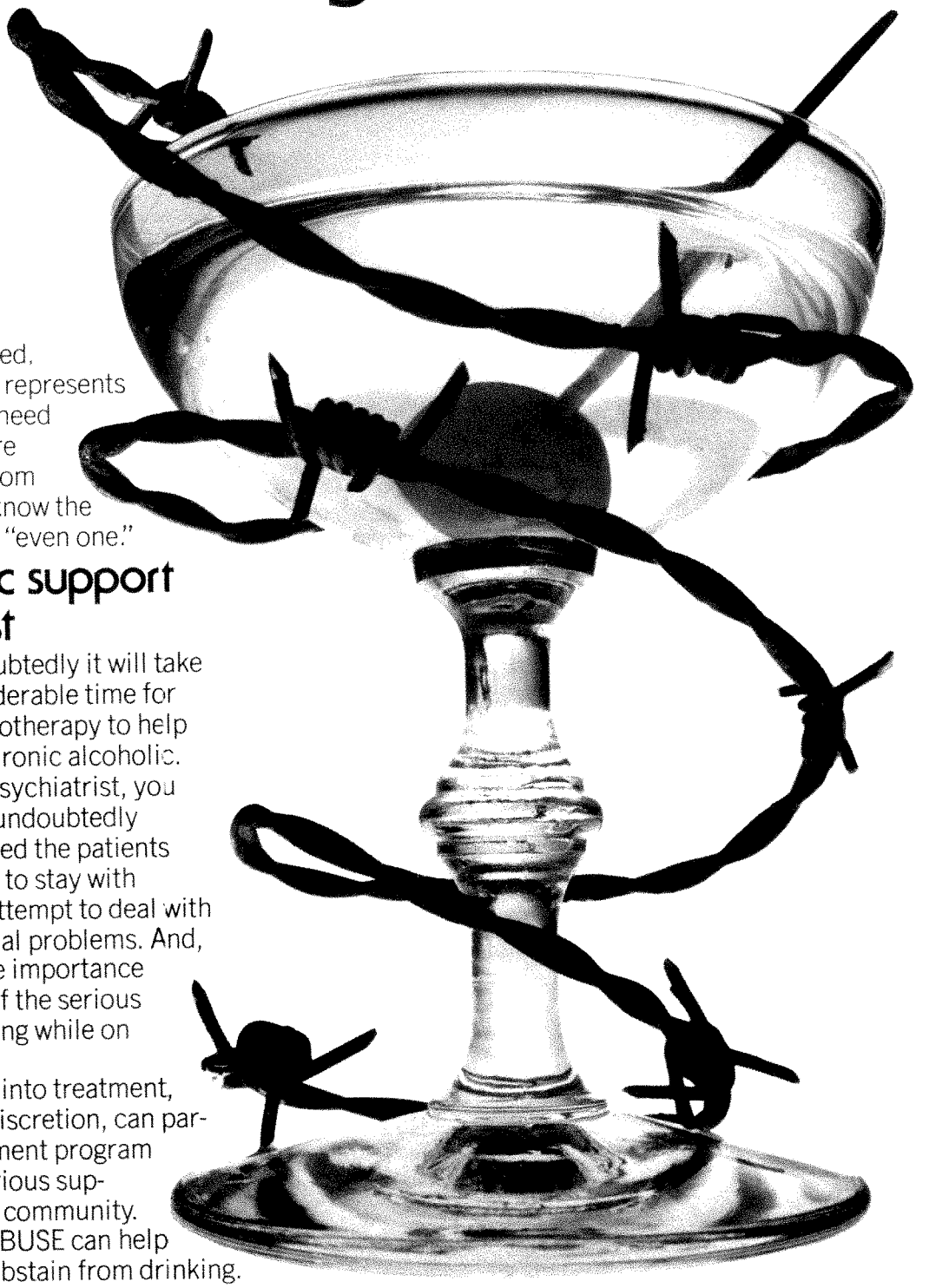
37.5 mg. tablets (orange-colored) in bottles of 100 (**NDC** 0074-6057-13)

75 mg. tablets (tan-colored) in bottles of 100 (**NDC** 0074-6073-13)

6013128R

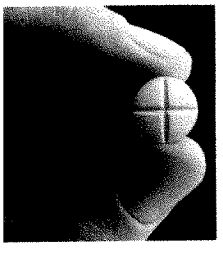
ABBOTT LABORATORIES
North Chicago, IL60064

ANTABUSE: "Social security" for the alcoholic who wants to stop drinking...



The temptation is resisted. ANTABUSE (disulfiram) represents the security alcoholics need in any situation. They are better able to abstain from drinking because they know the consequences of taking "even one."

until psychiatric support can do the rest



Undoubtedly it will take considerable time for psychotherapy to help the chronic alcoholic. As a psychiatrist, you have undoubtedly selected the patients who are best motivated to stay with ANTABUSE while you attempt to deal with the underlying emotional problems. And, of course, you know the importance of reminding patients of the serious consequences of drinking while on ANTABUSE.

Once sober, and well into treatment, the alcoholic, at your discretion, can participate in a total treatment program which makes use of various supportive resources in the community. In the meantime, ANTABUSE can help the chronic alcoholic abstain from drinking.

BRIEF SUMMARY
(For full prescribing information, see package circular.)

ANTABUSE® (disulfiram) in Alcoholism

INDICATION: ANTABUSE is an aid in the management of selected chronic alcoholic patients who want to remain in a state of enforced sobriety so that supportive and psychotherapeutic treatment may be applied to best advantage. (Used alone, without proper motivation and without supportive therapy, ANTABUSE is not a cure for alcoholism, and it is unlikely that it will have more than a brief effect on the drinking pattern of the chronic alcoholic.)

CONTRAINDICATIONS: Patients who are receiving or have recently received metronidazole, paraldehyde, alcohol, or alcohol-containing preparations, e.g. cough syrups, tonics, and the like, should not be given ANTABUSE.

ANTABUSE is contraindicated in the presence of severe myocardial disease or coronary occlusion, psychoses, or hypersensitivity.

WARNINGS: ANTABUSE should never be administered to a patient when he is in a state of alcohol intoxication or without his full knowledge.

The physician should instruct relatives accordingly.

The patient must be fully informed of the ANTABUSE-alcohol reaction. He must be strongly cautioned against surreptitious drinking while taking the drug, and he must be fully aware of possible consequences. He should be warned to avoid alcohol in disguised form, *i.e.* in sauces, vinegars, cough mixtures, and even aftershave lotions and back rubs. He should also be warned that reactions may occur with alcohol up to 14 days after ingesting ANTABUSE.

THE ANTABUSE-ALCOHOL REACTION: ANTABUSE plus alcohol, even small amounts, produces flushing, throbbing in head and neck, throbbing headache, respiratory difficulty, nausea, copious vomiting, sweating, thirst, chest pain, palpitation, dyspnea, hyperventilation, tachycardia, hypotension, syncope, marked uneasiness, weakness, vertigo, blurred vision, and confusion. In severe reactions there may be respiratory depression, cardiovascular collapse, arrhythmias, myocardial infarction, acute congestive heart failure, unconsciousness, convulsions, and death.

The intensity of the reaction varies with each individual, but is generally proportional to the amounts of ANTABUSE (disulfiram) and alcohol ingested. Mild reactions may occur in the sensitive individual when the blood alcohol concentration is increased to as little as 5 to 10 mg. per 100 cc. Symptoms are fully developed at 50 mg. per 100 cc., and unconsciousness usually results when the blood alcohol level reaches 125 to 150 mg.

The duration of the reaction varies from 30 to 60 minutes to several hours in the more severe cases, or as long as there is alcohol in the blood.

DRUG INTERACTIONS: Disulfiram appears to decrease the rate at which certain drugs are metabolized and so may increase the blood levels and the possibility of clinical toxicity of drugs given concomitantly.

Disulfiram should be used with caution in those patients receiving diphenylhydantoin and its congeners, since toxic levels of these antiepileptic agents have been reported during concomitant disulfiram therapy.

It may be necessary to adjust the dosage of oral anticoagulants upon beginning or stopping disulfiram, since disulfiram may prolong prothrombin time.

Patients taking isoniazid when disulfiram is given should be observed for the appearance of unsteady gait or marked changes in mental status and the disulfiram discontinued if such signs appear.

CONCOMITANT CONDITIONS: Because of the possibility of an accidental ANTABUSE-alcohol reaction, ANTABUSE (disulfiram) should be used with extreme caution in patients with any of the following conditions: diabetes mellitus, hypothyroidism, epilepsy, cerebral damage, chronic and acute nephritis, hepatic cirrhosis or insufficiency.

USAGE IN PREGNANCY: The safe use of this drug in pregnancy has not been established. Therefore, ANTABUSE (disulfiram) should be used during pregnancy only when, in the judgment of the physician, the probable benefits outweigh the possible risks.

PRECAUTIONS: It is suggested that every patient under treatment carry an Identification Card, stating that he is receiving ANTABUSE and describing the symptoms most likely to occur as a result of the ANTABUSE-alcohol reaction. In addition, this card should indicate the physician or institution to be contacted in emergency. (Cards may be obtained from Ayerst Laboratories upon request.)

Alcoholism may accompany or be followed by dependence on narcotics or sedatives. Barbiturates have been administered concurrently with ANTABUSE (disulfiram) without untoward effects, but the possibility of initiating a new abuse should be considered.

Base line and follow-up transaminase tests (10-14 days) are suggested to detect any hepatic dysfunction that may result with ANTABUSE therapy. In addition, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

ADVERSE REACTIONS: (See Contraindications, Warnings, and Precautions.)

Occasional skin eruptions are, as a rule, readily controlled by concomitant administration of an antihistaminic drug.

In a small number of patients, a transient mild drowsiness, fatigability, impotence, headache, acneform eruptions, allergic dermatitis, or a metallic or garlic-like aftertaste may be experienced during the first two weeks of therapy. These complaints usually disappear spontaneously with the continuation of therapy or with reduced dosage.

Psychotic reactions have been noted, attributable in most cases to high dosage, combined toxicity (with metronidazole or isoniazid), or to the unmasking of underlying psychoses in patients stressed by the withdrawal of alcohol.

There have been reports of polyneuritis and peripheral neuritis, and rare instances of optic neuritis. One case of cholestatic hepatitis has been reported, but its relationship to ANTABUSE has not been unequivocally established.

DOSAGE AND ADMINISTRATION: ANTABUSE (disulfiram) should never be administered until the patient has abstained from alcohol for at least 12 hours.

INITIAL DOSAGE SCHEDULE: In the first phase of treatment, a maximum of 500 mg. daily is given in a single dose for one to two weeks. Although usually taken in the morning, ANTABUSE may be taken on retiring by patients who experience a sedative effect. Alternatively, to minimize, or eliminate, the sedative effect, dosage may be adjusted downward.

MAINTENANCE REGIMEN: The average maintenance dose is 250 mg. daily (range, 125 to 500 mg.); it should not exceed 500 mg. daily.

NOTE: Occasional patients, while seemingly on adequate maintenance doses of ANTABUSE (disulfiram), report that they are able to drink alcoholic beverages with impunity and without any symptomatology. All appearances to the contrary, such patients must be presumed to be disposing of their tablets in some manner without actually taking them. Until such patients have been observed reliably taking their daily ANTABUSE tablets (preferably crushed and well mixed with liquid), it cannot be concluded that ANTABUSE is ineffective.

DURATION OF THERAPY: The daily, uninterrupted administration of ANTABUSE must be continued until the patient is fully recovered socially and a basis for permanent self-control is established. Depending on the individual patient, maintenance therapy may be required for months or even years.

TRIAL WITH ALCOHOL: During early experience with ANTABUSE, it was thought advisable for each patient to have at least one supervised alcohol-drug reaction. More recently, the test reaction has been largely abandoned. Furthermore, such a test reaction should never be administered to a patient over 50 years of age. A clear, detailed, and convincing description of the reaction is felt to be sufficient in most cases.

However, where a test reaction is deemed necessary, the suggested procedure is as follows:

After the first one to two weeks' therapy with 500 mg. daily, a drink of 15 cc. (½ oz.) of 100 proof whiskey or equivalent is taken slowly. This test dose of alcoholic beverage may be repeated once only so that the total dose does not exceed 30 cc. (1 oz.) of whiskey. Once a reaction develops, no more alcohol should be consumed. Such tests should be carried out only when the patient is hospitalized, or comparable supervision and facilities, including oxygen, are available.

MANAGEMENT OF ANTABUSE (DISULFIRAM)-ALCOHOL REACTION: In severe reactions, whether caused by an excessive test dose or by the patient's unsupervised ingestion of alcohol, supportive measures to restore blood pressure and treat shock should be instituted. Other recommendations include: oxygen, carbogen (95 per cent oxygen and 5 per cent carbon dioxide), vitamin C intravenously in massive doses (1 Gm.), and ephedrine sulfate. Antihistamines have also been used intravenously. Potassium levels should be monitored particularly in patients on digitalis since hypokalemia has been reported.

HOW SUPPLIED: No. 809—Each tablet (scored) contains 250 mg. disulfiram, in bottles of 100. No. 810—Each tablet (scored) contains 500 mg. disulfiram, in bottles of 50 and 1,000.

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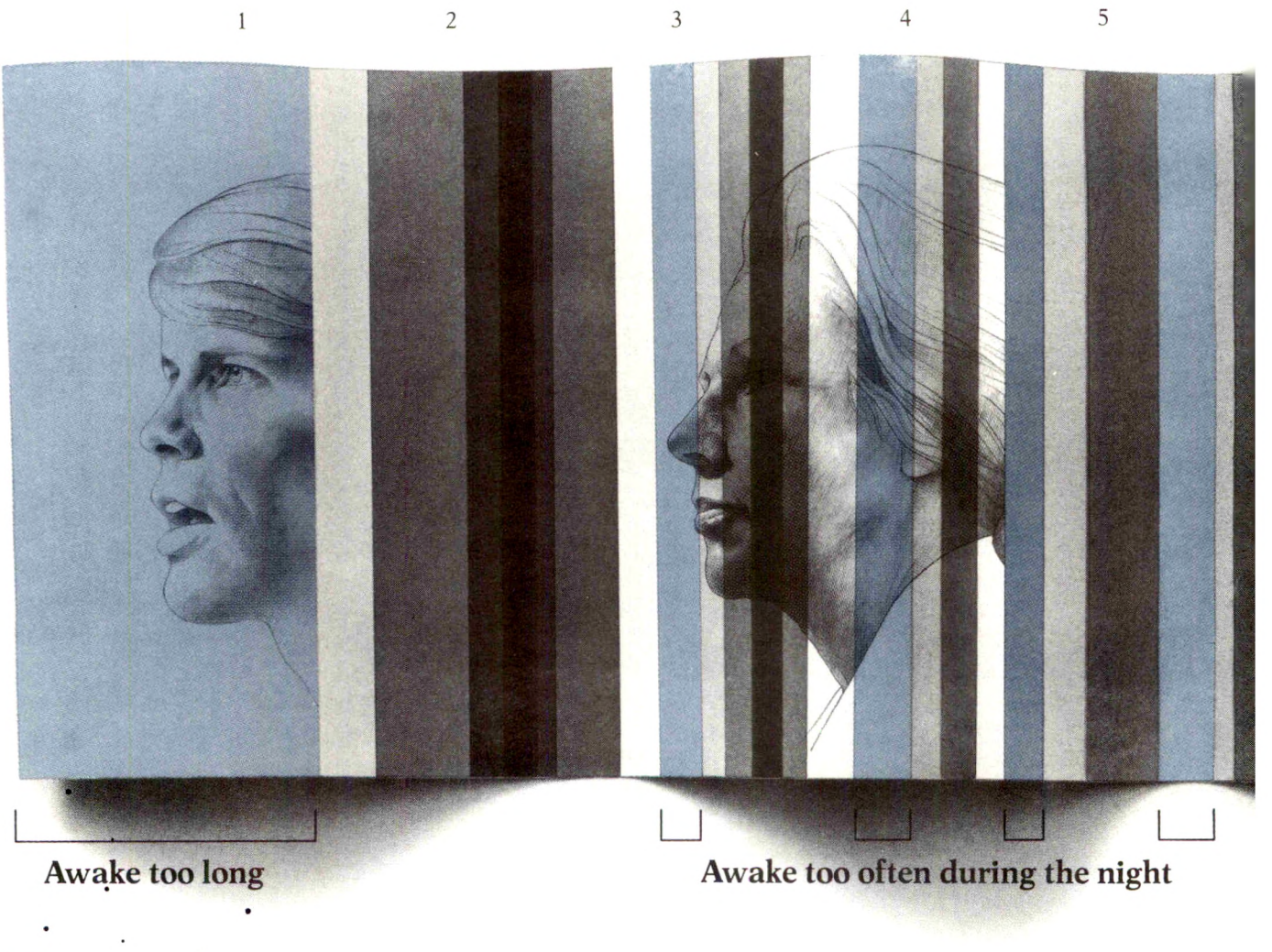
Awake too long, awake too often, awake too early...

These are the most common forms of insomnia, and may occur singly or in any combination. The night of troubled sleep depicted here comprises all three types. As the night progresses from left to right, each sleep stage is identifiable by its own shade of gray. Blue represents "Awake."

As you can see, this hypothetical "patient" takes well over an hour to fall asleep, awakens several times during the middle of the night and awakens too early in the morning.

Sleep Stages

- Awake
- REM
- Stage 1
- Stage 2
- Stage 3
- Stage 4



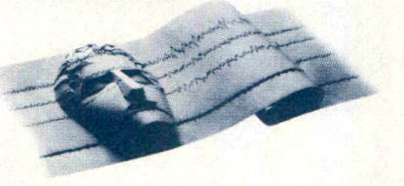
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The relative safety of Dalmane (flurazepam HCl) is well documented

Dalmane (flurazepam HCl) is relatively safe and well tolerated; morning "hang-over" has been infrequent. The usual adult dosage is 30 mg; in elderly or debilitated patients, limit initial dosage to 15 mg to preclude over-sedation, dizziness or ataxia. Caution patients about possible combined effects with alcohol and other CNS depressants.

Broad-spectrum medication for the most common forms of insomnia

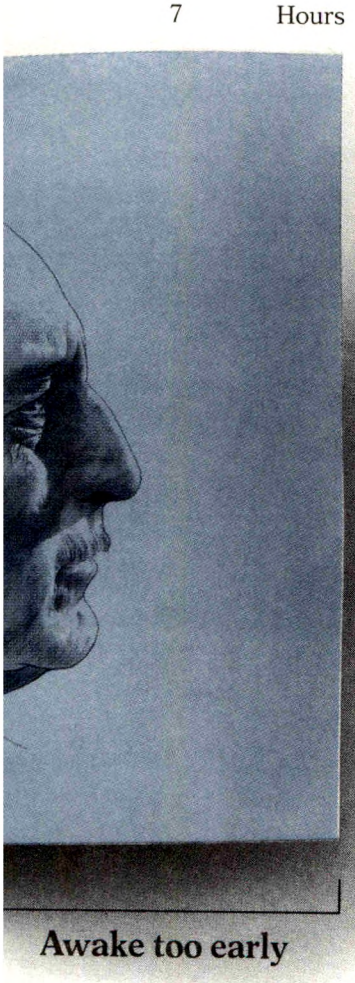


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One 15-mg capsule *h.s.*— initial dosage for elderly or debilitated patients.

- □ induces sleep rapidly
- □ reduces nighttime awakenings
- □ lengthens total sleep time

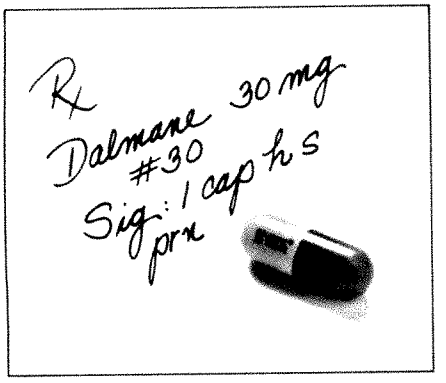
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- provides 7 to 8 hours sleep, on average, without repeating dosage

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; and in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Use in women who are or may become pregnant only when potential benefits have been weighed against possible hazards. Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, initial dosage should be limited to 15 mg to preclude oversedation dizziness and/or ataxia. If combined with other drugs having hypnotic or CNS-depressant effects, consider potential additive effects. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

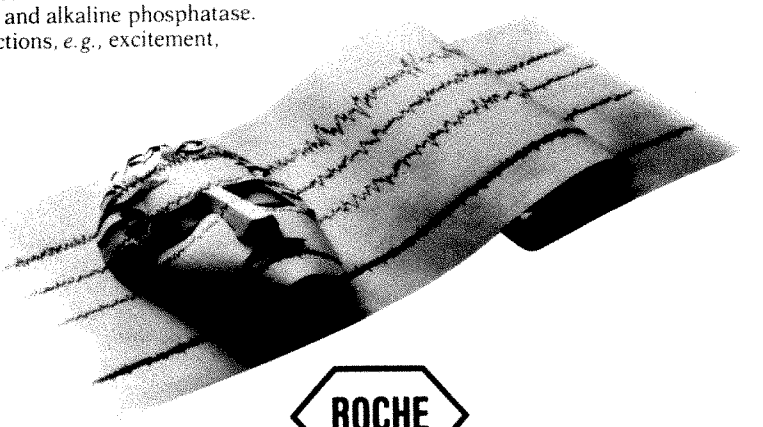
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported were headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase. Paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, have also been reported in rare instances.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:

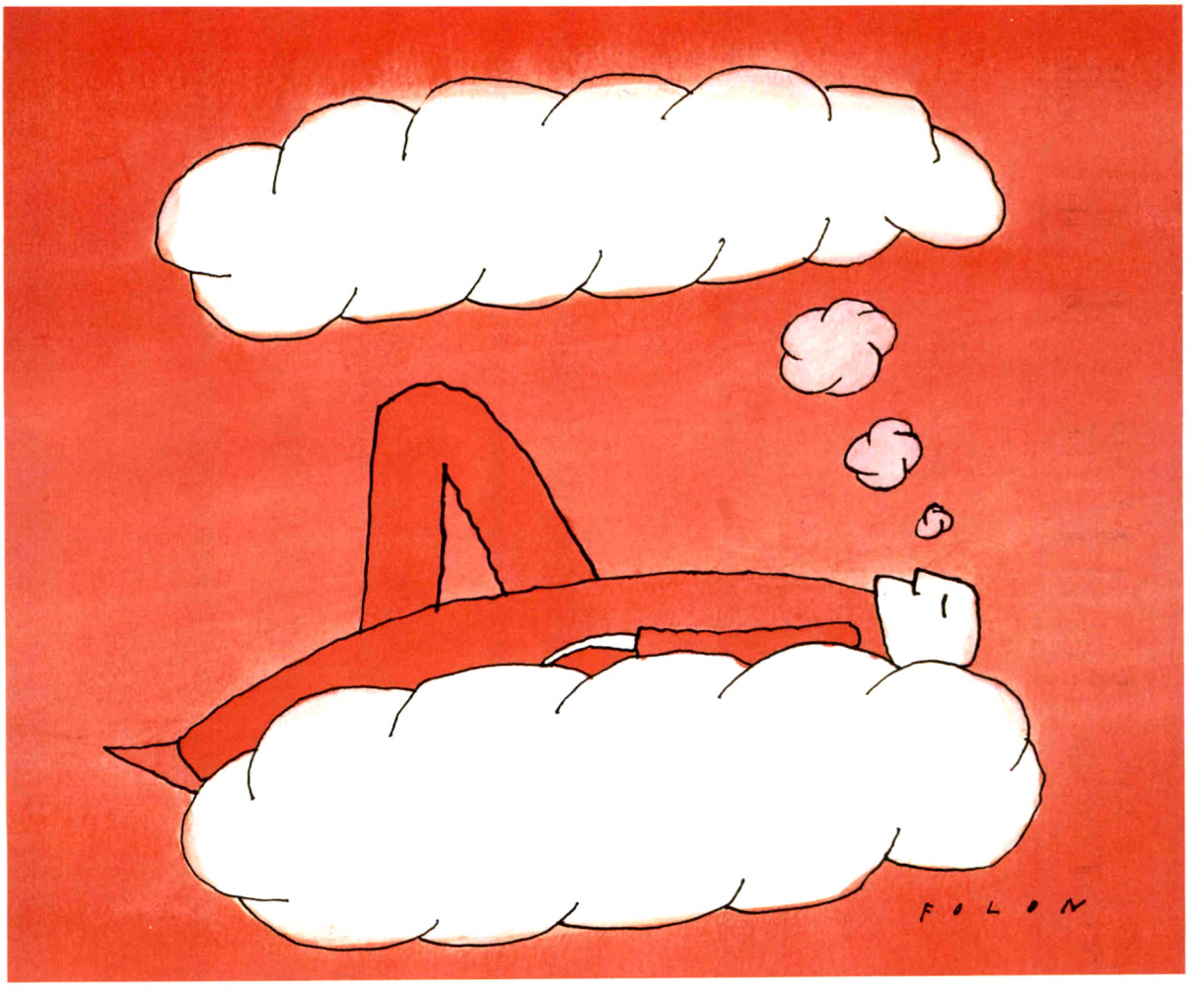
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Vol. 133, No. 6 June 1976

THE AMERICAN JOURNAL OF PSYCHIATRY

In this issue

The Syndrome of Autism: A Critical Review by Edward M. Ornitz and Edward R. Ritvo

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Basic tools of primitive psychiatry



Mankpo: Sceptres or staffs used by the Heviosso (lightning God) cult in medical ceremonies among the Ewe of Tongo and Fon of Dahomey. *Right,* staff with leopard's head, bared teeth and tongue (Ewe, Togo). *Left,* staff with ram's head—the sacred animal of the Heviosso—in the form of a serpent symbolizing the fire-spitting rainbow God of healing (Fon, Dahomey). From the collection of the Segy Gallery, New York City.

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Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings: Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudo-parkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.
NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

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By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

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- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

80pp. Paperbound

Single copy, $4.00; 10–49 copies; $3.80 each; 50 or more copies, $3.20 each.

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2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
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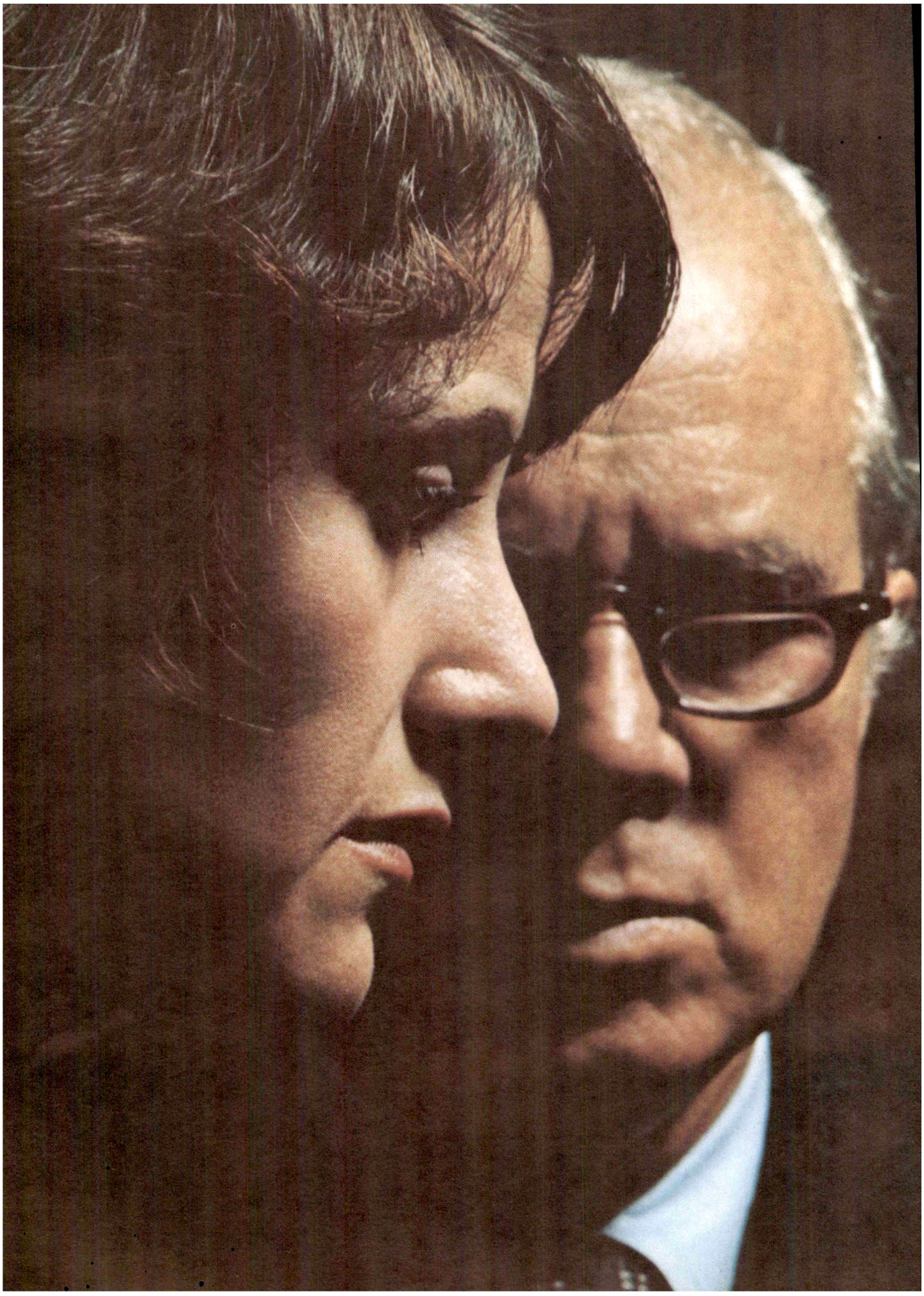
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WARNINGS: TRIAVIL should not be given with guanethidine or similarly acting compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, particularly in high doses, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. Caution patients performing hazardous tasks, such as operating machinery or driving motor vehicles, that drug may impair mental and/or physical abilities. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Side effects may be any of those reported with phenothiazine drugs: extrapyramidal symptoms (opisthotonus, oculogyric crisis, hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but sometimes persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. It has been suggested that fine vermicular movements of the tongue may be an early sign of the syndrome, and that the full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema, urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement; hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine-like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia, nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; hypnotic effects; pigmentary retinopathy; corneal and lenticular pigmentation; occasional lassitude, muscle weakness, mild insomnia. Other adverse reactions reported with various phenothiazine compounds include blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); liver damage (jaundice, biliary stasis); grand mal convulsions; cerebral edema; polyphagia; photophobia; skin pigmentation; and failure of ejaculation.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness; weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

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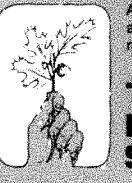
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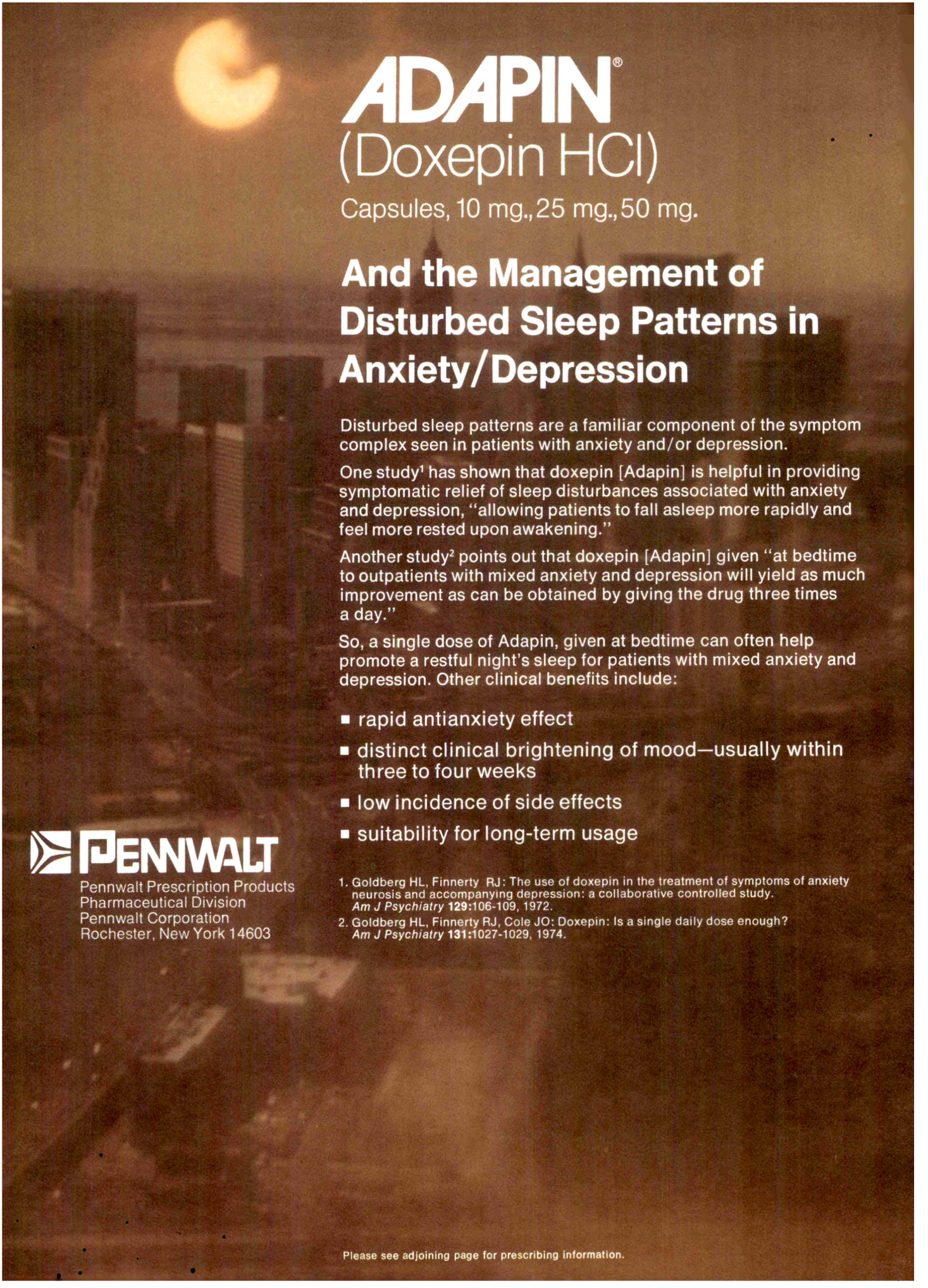
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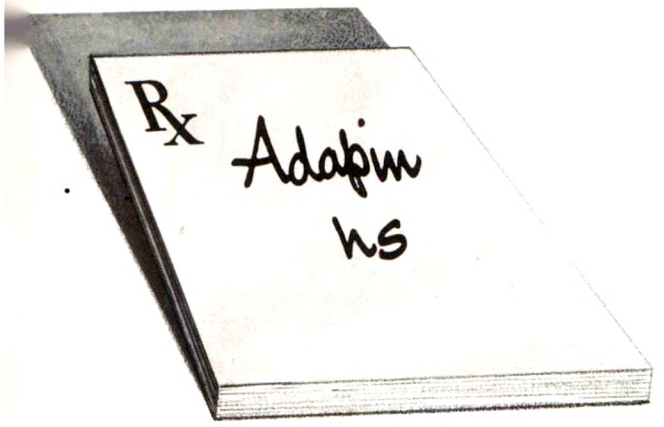
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1. Goldberg HL, Finnerty RJ: The use of doxepin in the treatment of symptoms of anxiety neurosis and accompanying depression: a collaborative controlled study. *Am J Psychiatry* **129**:106-109, 1972.
2. Goldberg HL, Finnerty RJ, Cole JO: Doxepin: Is a single daily dose enough? *Am J Psychiatry* **131**:1027-1029, 1974.

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25-mg. capsules



50-mg. capsules

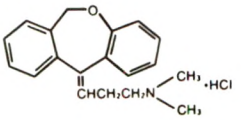
ADAPIN®

(Doxepin HCl)

Prescribing information:

DESCRIPTION

Adapin (doxepin HCl) is an isomeric mixture of N, N-dimethyl-dibenz(b,e) oxepin- $\Delta^{11(6H)}$, γ propylamine hydrochloride.



ACTIONS

Adapin has a variety of pharmacological actions with its predominant action on the central nervous system. While its mechanism of action is not known, studies have demonstrated that it is neither a monoamine oxidase inhibitor nor a primary stimulant of the central nervous system.

INDICATIONS

In controlled clinical evaluations, **Adapin** has shown marked antianxiety and significant antidepressant effects. **Adapin** has been found to be well tolerated even in elderly patients.

Adapin is indicated for the treatment of patients with:

1. Psychoneurotic anxiety and/or depressive reactions.
2. Mixed symptoms of anxiety and depression.
3. Anxiety and/or depression associated with alcoholism.
4. Anxiety associated with organic disease.
5. Psychotic depressive disorders including involutional depression and manic-depressive reactions.

Target symptoms of psychoneurosis that respond particularly well to **Adapin** include: anxiety, tension, depression, somatic symptoms and concerns, insomnia, guilt, lack of energy, fear, apprehension and worry.

Because **Adapin** provides antidepressant as well as antianxiety effects, it is of particular value in patients in whom anxiety masks depression. Patients who have not responded to other antianxiety or antidepressant drugs may benefit from **Adapin.**

In a large series of patients systematically observed for withdrawal symptoms, none were reported—a finding which is consistent with the virtual absence of euphoria as a side effect and the lack of addictive potential characteristic of this type of chemical compound.

CONTRAINDICATIONS

Because **Adapin** has an anticholinergic effect, it is contraindicated in patients with glaucoma or a tendency toward urinary retention.

Use of **Adapin** is contraindicated in patients who have been found hypersensitive to it.

WARNINGS

Usage in Pregnancy—Adapin has not been evaluated in pregnant patients. Therefore, it should not be used during pregnancy unless, in the judgment of the physician, it is essential to the welfare of the patient.

In animal reproduction studies of **Adapin (doxepin hydrochloride),** gross and microscopic examination of the offspring gave no evidence of drug-related teratogenic effect. Following doses of up to 25 mg./kg./day for 8 to 9 months, no changes were observed in the number of live births, litter size, or lactation. A decreased rate of conception was observed when male rats were given 25 mg./kg./day for prolonged periods—an effect which has occurred with other psychotropic drugs and has been attributed to drug effect on the central and/or autonomic nervous systems.

Usage in Children—The use of **Adapin** in children under 12 years of age is not recommended, because safe conditions for its use have not been established.

MAO Inhibitors—Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with **Adapin.** The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered, and the dosage involved.

PRECAUTIONS

Drowsiness may occur with **Adapin;** therefore, patients should be warned of its possible occurrence and cautioned against driving a motor vehicle or operating hazardous machinery while taking the drug.

Patients should also be cautioned that the effects of alcoholic beverages may be increased.

Since suicide is an inherent risk in depressed patients and remains a risk through the initial phases of improvement, depressed patients should be closely supervised.

Although **Adapin** has shown effective tranquilizing activity, the possibility of activating or unmasking latent psychotic symptoms should be kept in mind.

Compounds structurally related to **Adapin** can block the effects of guanethidine and similarly acting compounds. However, at the usual clinical dosages, 75 mg. to 150 mg. per day, **Adapin** has been given concomitantly with guanethidine without blocking its antihypertensive effect. But at dosages of 300 mg. per day or higher, **Adapin** has exerted a significant blocking effect.

Adapin, like other structurally related psychotropic drugs, potentiates norepinephrine response in animals. But this effect has not been observed with **Adapin** in humans, which is in accord with the low incidence of tachycardia reported clinically.

ADVERSE REACTIONS

Anticholinergic Effects: Dry mouth, blurred vision and constipation have been reported. These are usually mild, and often subside as therapy is continued or dosage reduced.

Central Nervous System Effects: Drowsiness has been observed. It usually occurs early in the course of therapy and tends to subside as therapy continues. (See Dosage and Administration section.)

Cardiovascular Effects: Tachycardia and hypotension have been reported infrequently.

Other infrequently reported adverse effects include extrapyramidal symptoms, gastrointestinal reactions, secretory effects (such as increased sweating), weakness, dizziness, fatigue, weight gain, edema, paresthesias, flushing, chills, tinnitus, photophobia, decreased libido, rash, and pruritus.

DOSAGE AND ADMINISTRATION

In most patients with mild to moderate anxiety and/or depression: 10 mg. to 25 mg. t.i.d. to start. A starting dosage of 10 mg. t.i.d. for a period of four days may reduce the initial drowsiness experienced by some patients, and may be tried in cases where drowsiness is clinically undesirable. Decrease or increase the dosage at appropriate intervals according to individual response. Usual optimum dosage is 75 mg. to 150 mg. per day.

In some patients with mild symptomatology or emotional symptoms accompanying organic disease, dosage as low as 25 mg. to 50 mg. per day has provided effective control.

In more severe anxiety and/or depression: 50 mg. t.i.d. may be required to start—if necessary, gradually increase to 300 mg. per day. Additional effectiveness is rarely obtained by exceeding 300 mg. per day.

Although optimal antidepressant response may not be evident for two to three weeks, antianxiety activity is rapidly apparent.

OVERDOSAGE

Symptoms—An increase of any of the reported adverse reactions, primarily excessive sedation and anticholinergic effects such as blurred vision and dry mouth. Other effects may be: pronounced tachycardia, hypotension and extrapyramidal symptoms.

Treatment—Essentially symptomatic; supportive therapy in the case of hypotension and excessive sedation.

HOW SUPPLIED

Each capsule contains doxepin, as the hydrochloride, 10 mg. (NDC 0018-0356), 25 mg. (NDC 0018-0357), and 50 mg. (NDC 0018-0358) capsules in bottles of 100 and 1000.

PENNWALT

Pennwalt Prescription Products
Pharmaceutical Division
Pennwalt Corporation
Rochester, New York 14603

ROCHE

Introducing

The unforgettable antidepressant

Endep™ 75 mg

amitriptyline HCl/Roche

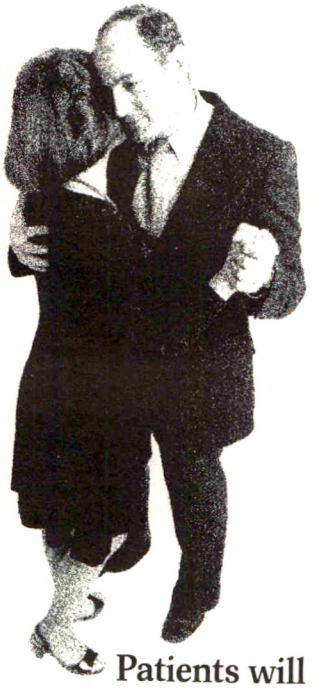
Here's what makes it unforgettable



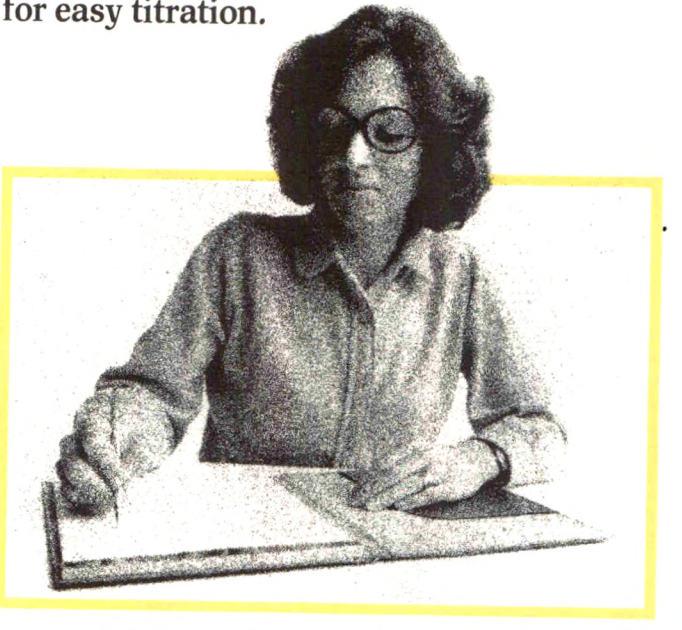
Patients are more likely to remember the single bedtime dosage of Endep (amitriptyline HCl).



You'll remember 1 or 2 tablets *h.s.*, because fewer forgotten doses produce a more consistent clinical effect, and because it is the only tricyclic antidepressant scored for easy titration.



Patients will also remember family/interpersonal relationships that they have forgotten or neglected.



In three or four weeks patients will usually remember how well they used to feel.

Endep™ 75 mg

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day

Before prescribing, please consult complete product information, a summary of which appears on the following page.

Introducing Endep™

amitriptyline HCl/Roche

The antidepressant you can prescribe at night to work all day



(orange)	(orange)	(orange)	(yellow)
10-mg scored tablets	**25-mg** scored tablets	**50-mg** scored tablets	**75-mg** scored tablets

■ Endep (amitriptyline HCl), in responsive patients, lifts mood and relieves symptoms such as anorexia, loss of interest, inability to concentrate, decreased libido or GI disturbances, often within three or four weeks after starting therapy.

■ Endep usually relieves accompanying sleep disturbances, particularly early in the treatment regimen.

■ All four dosage strengths are scored for greater flexibility and convenience in titration.

■ The 75-mg *h.s.* dose minimizes skipped doses, maximizes patient acceptance and compliance.

■ Specifying Endep/Roche assures quality plus potential economy.

Important Considerations

Should not be given concomitantly with an MAO inhibitor.

Not recommended for use during the acute recovery phase following myocardial infarction.

Contraindicated in patients with hypersensitivity to amitriptyline.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh potential benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia. purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels; increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier. *Initial Adult Dosage:* Outpatients – 25 mg t.i.d.; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate – 50 to 100 mg h.s., gradually increasing h.s. dose up to 150 mg/day. Hospitalized Patients – Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day. *Adolescent and Elderly Patients:* In general, 10 mg t.i.d. with 20 mg h.s. may be satisfactory for those who do not tolerate higher doses. *Maintenance Dosage:* With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg b.i.d. to q.i.d., or 10 mg q.i.d. Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg and 75-mg scored tablets – bottles of 100 and 500; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg), available singly and in trays of 10.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110



July 1976

A journal of the American Psychiatric Association

Hospital & Community Psychiatry



Two Hundred Years of Mental Health Care in America

. . . That's the theme of *Hospital & Community Psychiatry*'s Bicentennial issue, to be published in July. It's an issue you won't want to miss, one you'll want to keep and refer to again and again. With it you can explore the past, gain perspectives on the present, catch a light-hearted glimpse of the future.

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Title or Discipline _____
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WHEN IS DEPRESSION A DISEASE?

Does schizophrenia run in families?

IS SUICIDE A SICKNESS?

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THE TROUBLED MIND: A Guide to Release from Distress

"His book is directed to adults outside the mental health professions, especially to the mentally or emotionally distressed and their relatives and friends, and provides a thorough overview of the different treatment methods available."
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Milwaukee Psychiatric Hospital — Intensive, dynamic psychotherapy for adults and adolescents, individually-planned activity therapy.

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Dewey Center — Acute detoxification and inpatient treatment for persons with dependency on alcohol and other drugs, daily schedules, broad supportive services.

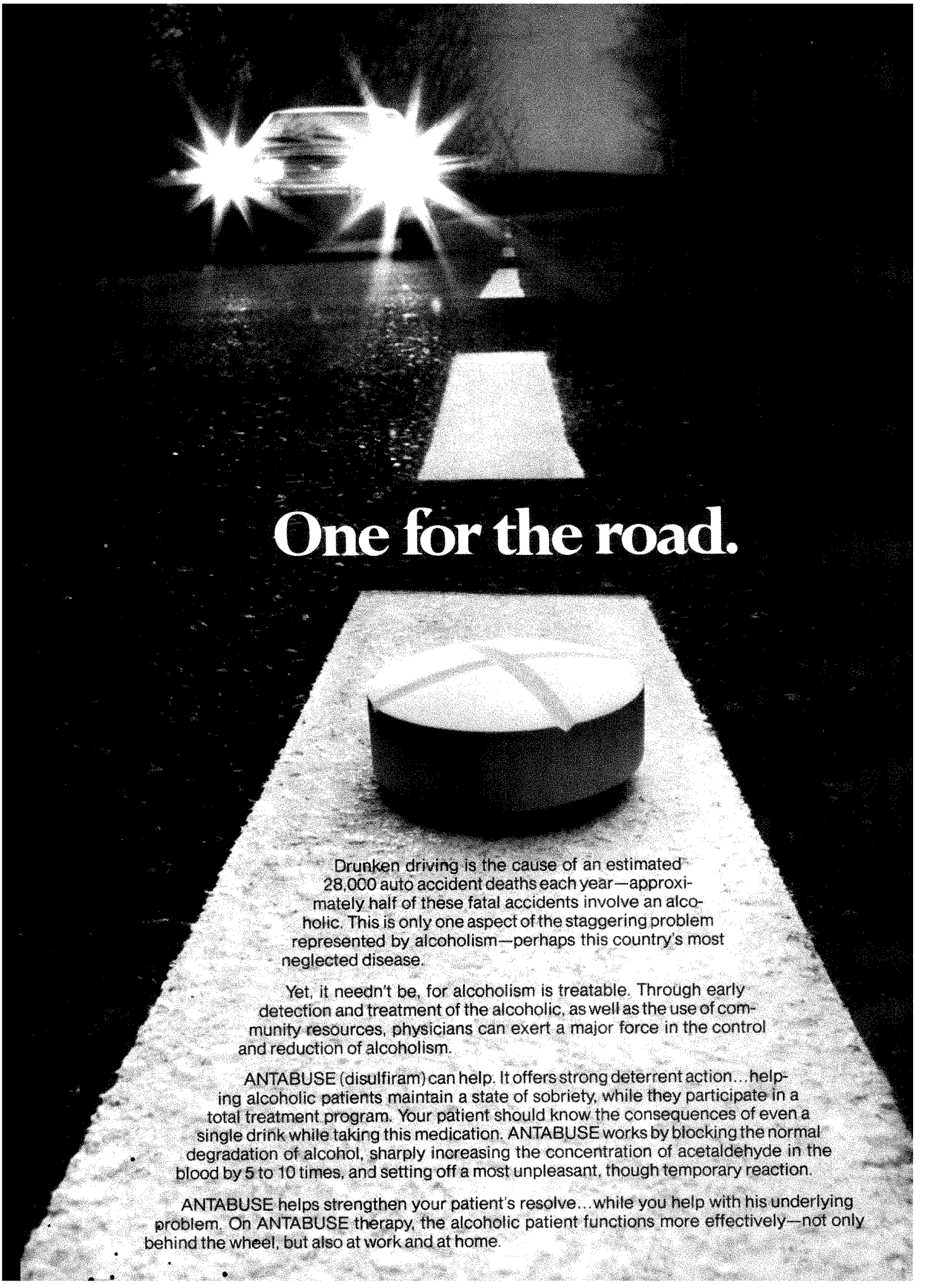
Milwaukee Sanitarium — Custodial services for persons with chronic emotional and geriatric illnesses.

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Drunken driving is the cause of an estimated 28,000 auto accident deaths each year—approximately half of these fatal accidents involve an alcoholic. This is only one aspect of the staggering problem represented by alcoholism—perhaps this country's most neglected disease.

Yet, it needn't be, for alcoholism is treatable. Through early detection and treatment of the alcoholic, as well as the use of community resources, physicians can exert a major force in the control and reduction of alcoholism.

ANTABUSE (disulfiram) can help. It offers strong deterrent action...helping alcoholic patients maintain a state of sobriety, while they participate in a total treatment program. Your patient should know the consequences of even a single drink while taking this medication. ANTABUSE works by blocking the normal degradation of alcohol, sharply increasing the concentration of acetaldehyde in the blood by 5 to 10 times, and setting off a most unpleasant, though temporary reaction.

ANTABUSE helps strengthen your patient's resolve...while you help with his underlying problem. On ANTABUSE therapy, the alcoholic patient functions more effectively—not only behind the wheel, but also at work and at home.

BRIEF SUMMARY
(For full prescribing information, see package circular.)
ANTABUSE® (disulfiram) In Alcoholism
INDICATION: ANTABUSE is an aid in the management of selected chronic alcoholic patients who want to remain in a state of enforced sobriety so that supportive and psychotherapeutic treatment may be applied to best advantage. (Used alone, without proper motivation and without supportive therapy, ANTABUSE is not a cure for alcoholism, and it is unlikely that it will have more than a brief effect on the drinking pattern of the chronic alcoholic.)
CONTRAINDICATIONS: Patients who are receiving or have recently received metronidazole, paraldehyde, alcohol, or alcohol-containing preparations, *e.g.* cough syrups, tonics, and the like, should not be given ANTABUSE.
ANTABUSE is contraindicated in the presence of severe myocardial disease or coronary occlusion, psychoses, or hypersensitivity.

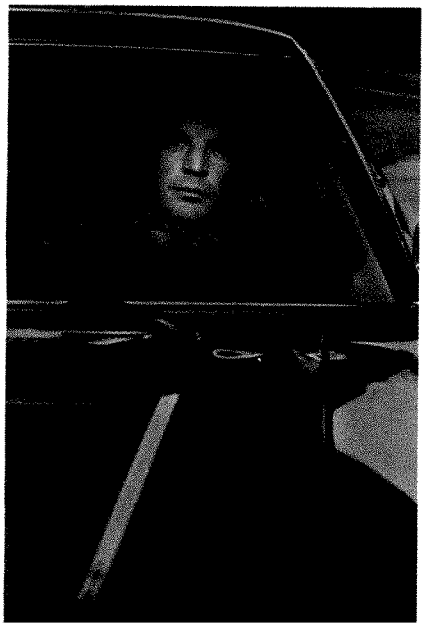
WARNINGS: ANTABUSE should never be administered to a patient when he is in a state of alcohol intoxication or without his full knowledge.
The physician should instruct relatives accordingly.

The patient must be fully informed of the ANTABUSE-alcohol reaction. He must be strongly cautioned against surreptitious drinking while taking the drug, and he must be fully aware of possible consequences. He should be warned to avoid alcohol in disguised form, *i.e.* in sauces, vinegars, cough mixtures, and even aftershave lotions and back rubs. He should also be warned that reactions may occur with alcohol up to 14 days after ingesting ANTABUSE.
THE ANTABUSE-ALCOHOL REACTION:
ANTABUSE plus alcohol, even small amounts, produces flushing, throbbing in head and neck, throbbing headache, respiratory difficulty, nausea, copious vomiting, sweating, thirst, chest pain, palpitation, dyspnea, hyperventilation, tachycardia, hypotension, syncope, marked uneasiness, weakness, vertigo, blurred vision, and confusion. In severe reactions there may be respiratory depression, cardiovascular collapse, arrhythmias, myocardial infarction, acute congestive heart failure, unconsciousness, convulsions, and death.
The intensity of the reaction varies with each individual, but is generally proportional to the amounts of ANTABUSE (disulfiram) and alcohol ingested. Mild reactions may occur in the sensitive individual when the blood alcohol concentration is increased to as little as 5 to 10 mg. per 100 cc. Symptoms are fully developed at 50 mg. per 100 cc., and unconsciousness usually results when the blood alcohol level reaches 125 to 150 mg.
The duration of the reaction varies from 30 to 60 minutes to several hours in the more severe cases, or as long as there is alcohol in the blood.
DRUG INTERACTIONS: Disulfiram appears to decrease the rate at which certain drugs are metabolized and so may increase the blood levels and the possibility of clinical toxicity of drugs given concomitantly.
Disulfiram should be used with caution in those patients receiving diphenylhydantoin and its congeners, since toxic levels of these antiepileptic agents have been reported during concomitant disulfiram therapy.
It may be necessary to adjust the dosage of oral anticoagulants upon beginning or stopping disulfiram, since disulfiram may prolong prothrombin time.
Patients taking isoniazid when disulfiram is given should be observed for the appearance of unsteady gait or marked changes in mental status and the disulfiram discontinued if such signs appear.
CONCOMITANT CONDITIONS: Because of the possibility of an accidental ANTABUSE-alcohol reaction, ANTABUSE (disulfiram) should be used with extreme caution in patients with any of the following conditions: diabetes mellitus, hypothyroidism, epilepsy, cerebral damage, chronic and acute nephritis, hepatic cirrhosis or insufficiency.
USAGE IN PREGNANCY: The safe use of this drug in pregnancy has not been established. Therefore, ANTABUSE should be used during pregnancy only when, in the judgment of the physician, the probable benefits outweigh the possible risks.
PRECAUTIONS: It is suggested that every patient under treatment carry an Identification Card, stating that he is receiving ANTABUSE and describing the symptoms most likely to occur as a result of the ANTABUSE-alcohol reaction. In addition, this card should indicate the physician or institution to be contacted in emergency. (Cards may be obtained from Ayerst Laboratories upon request.)
Alcoholism may accompany or be followed by dependence on narcotics or sedatives. Barbiturates have been administered concurrently with ANTABUSE (disulfiram) without untoward effects, but the possibility of initiating a new abuse should be considered.
Base line and follow-up transaminase tests (10-14 days) are suggested to detect any hepatic dysfunction that may result with ANTABUSE therapy. In addition, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.
ADVERSE REACTIONS: (See Contraindications, Warnings, and Precautions.)
Optic neuritis, peripheral neuritis, and polyneuritis may occur following administration of ANTABUSE.
Occasional skin eruptions are, as a rule, readily controlled by concomitant administration of an antihistaminic drug.
In a small number of patients, a transient mild drowsiness, fatigability, impotence, headache, acneform eruptions, allergic dermatitis, or a metallic or garlic-like aftertaste may be experienced during the first two weeks of therapy. These complaints usually disappear spontaneously with the continuation of therapy or with reduced dosage.
Psychotic reactions have been noted, attributable in most cases to high dosage, combined toxicity (with metronidazole or isoniazid), or to the unmasking of underlying psychoses in patients stressed by the withdrawal of alcohol.
One case of cholestatic hepatitis has been reported, but its relationship to ANTABUSE has not been unequivocally established.
DOSAGE AND ADMINISTRATION: ANTABUSE (disulfiram) should never be administered until the patient has abstained from alcohol for at least 12 hours.
INITIAL DOSAGE SCHEDULE: In the first phase of treatment, a maximum of 500 mg. daily is given in a single dose for one to two weeks. Although usually taken in the morning, ANTABUSE may be taken on retiring by patients who experience a sedative effect. Alternatively, to minimize, or eliminate, the sedative effect, dosage may be adjusted downward.
MAINTENANCE REGIMEN: The average maintenance dose is 250 mg. daily (range, 125 to 500 mg.); it should not exceed 500 mg. daily.
NOTE: Occasional patients, while seemingly on adequate maintenance doses of ANTABUSE, report that they are able to drink alcoholic beverages with impunity and without any symptomatology. All appearances to the contrary, such patients must be presumed to be disposing of their tablets in some manner without actually taking them. Until such patients have been observed reliably taking their daily ANTABUSE tablets (preferably crushed and well mixed with liquid), it cannot be concluded that ANTABUSE is ineffective.
DURATION OF THERAPY: The daily, uninterrupted administration of ANTABUSE must be continued until the patient is fully recovered socially and a basis for permanent self-control is established. Depending on the individual patient, maintenance therapy may be required for months or even years.
TRIAL WITH ALCOHOL: During early experience with ANTABUSE, it was thought advisable for each patient to have at least one supervised alcohol-drug reaction. More recently, the test reaction has been largely abandoned. Furthermore, such a test reaction should never be administered to a patient over 50 years of age. A clear, detailed, and convincing description of the reaction is felt to be sufficient in most cases.
However, where a test reaction is deemed necessary, the suggested procedure is as follows:
After the first one to two weeks' therapy with 500 mg. daily, a drink of 15 cc. (1/2 oz.) of 100 proof whiskey or equivalent is taken slowly. This test dose of alcoholic beverage may be repeated once only so that the total dose does not exceed 30 cc. (1 oz.) of whiskey. Once a reaction develops, no more alcohol should be consumed. Such tests should be carried out only when the patient is hospitalized, or comparable supervision and facilities, including oxygen, are available.
MANAGEMENT OF ANTABUSE (DISULFIRAM)-ALCOHOL REACTION: In severe reactions, whether caused by an excessive test dose or by the patient's unsupervised ingestion of alcohol, supportive measures to restore blood pressure and treat shock should be instituted. Other recommendations include: oxygen, carbogen (95 per cent oxygen and 5 per cent carbon dioxide), vitamin C intravenously in massive doses (1 Gm.), and ephedrine sulfate. Antihistamines have also been used intravenously. Potassium levels should be monitored particularly in patients on digitalis since hypokalemia has been reported.
HOW SUPPLIED: No. 809—Each tablet (scored) contains 250 mg. disulfiram, in bottles of 100.
No. 810—Each tablet (scored) contains 500 mg. disulfiram, in bottles of 50 and 1,000. **7626**

Antabuse®

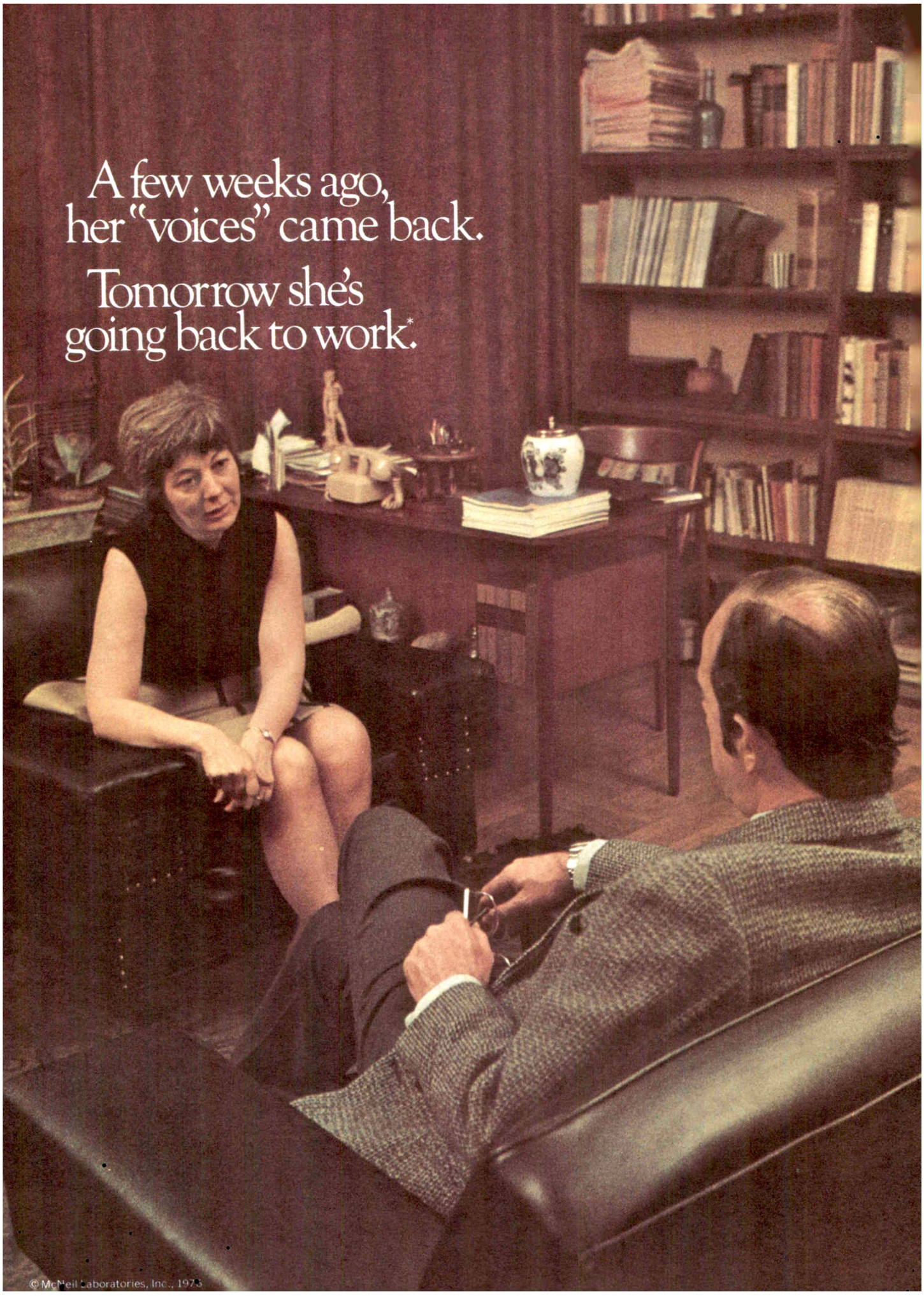
BRAND OF DISULFIRAM

Pharmacologic Deterrent Therapy
Tablets, 250 mg. and 500 mg.

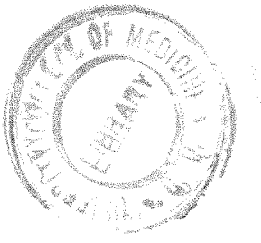


A first step in rehabilitation of the alcoholic

Ayerst®
AYERST LABORATORIES
New York, N.Y. 10017


A few weeks ago,
her "voices" came back.
Tomorrow she's
going back to work.*
© McNeil Laboratories, Inc., 1973

Haldol®
(haloperidol)
tablets/concentrate/injection



For prompt control with minimal risk of hypotension or oversedation

Highly effective in a wide range of psychotic symptoms[1-7]
such as hallucinations, delusions, suspiciousness, hostility, mania, psychomotor agitation, etc., in both acute and chronic disorders.

Helps keep patients functioning and avoid hospitalization.[1-3]
HALDOL haloperidol usually leaves most patients alert and responsive, able to function at work and at home without impairment, and more amenable to other therapeutic measures.

Permits aggressive titration to effective dosage levels[1,4]
up to 100 mg/day orally (with minimal risk of usual troublesome reactions) to achieve optimal response when patient is inadequately controlled at lower dosage...to help you rapidly control and stabilize new patients, promptly regain control during periods of exacerbation.

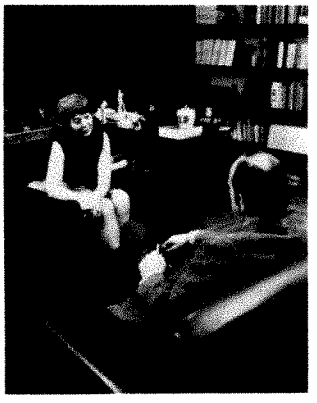
Common side effects easily controlled.[1,2,4]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Reduces risk of certain troublesome reactions.[1,2,4-6]
Transient hypotension occurs rarely and severe orthostatic hypotension has not been reported. Marked sedation is rare, although some instances of drowsiness have been reported. In addition, with chronic use, it is unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes...and has minimal effect on renal function.

Please turn page for information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions.

Important: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.



Haldol® (haloperidol)

tablets/concentrate/injection

For prompt control of psychotic symptoms with minimal risk of hypotension or oversedation

A Dosage Form for Every Need:

5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.

A tasteless, odorless, colorless liquid concentrate for better patient acceptability: 2 mg. per ml.

A rapid-acting injection for psychiatric emergencies: 5 mg. per ml., with 1.8 mg. methylparaben and 0.2 mg. propylparaben per ml., and lactic acid for pH adjustment to 3.4±0.2.

Indications: HALDOL haloperidol is indicated for use in the management of manifestations of psychotic disorders.

It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxically further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9/74

References:

1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. **2.** Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. **3.** Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. **4.** Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. **5.** Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. **6.** Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. **7.** Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973.

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OBESITY: Etiology, Treatment and Management *edited by* **Milton V. Kline,** *The Institute for Research in Hypnosis, New York;* **Lester L. Coleman,** *Albert Einstein College of Medicine, New York; and* **Erika E. Wick,** *St. John's Univ., Jamaica, New York. (39 Contributors)* In addition to full clinical coverage, material is presented on classification of obesity within a psychodynamic and clinical orientation, and types of obesity which are encountered in the daily practice of psychotherapy and medicine. *'76, 480 pp., 14 il., 34 tables, $21.75*

IDENTITY GROUP PSYCHOTHERAPY WITH ADOLESCENTS *by* **Arnold W. Rachman,** *New York, New York.* The main objective of this book is to present a unique, relevant, contemporary theory of group psychotherapy with adolescents. The author presents an enthusiastic, positive feeling while at the same time he is aware of the realistic difficulties and concerns about working with adolescents in groups. *'75, 336 pp., $16.75*

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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence. In pregnancy, lactation or women of childbearing age, weigh potential benefit against possible hazard.

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

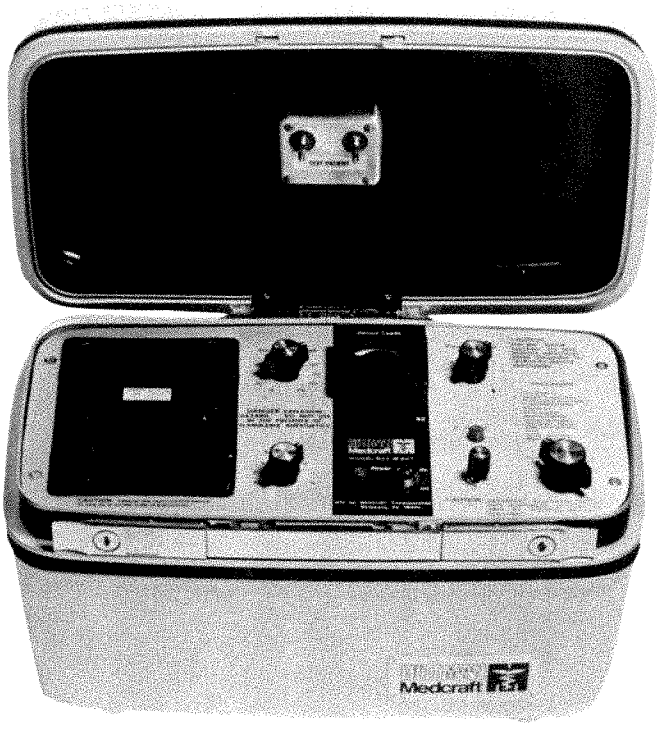
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline (primidone) for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards—used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate—As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail—A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

Ayerst®

Mysoline® (primidone)

Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

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may be the start of a better life for the epileptic

initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

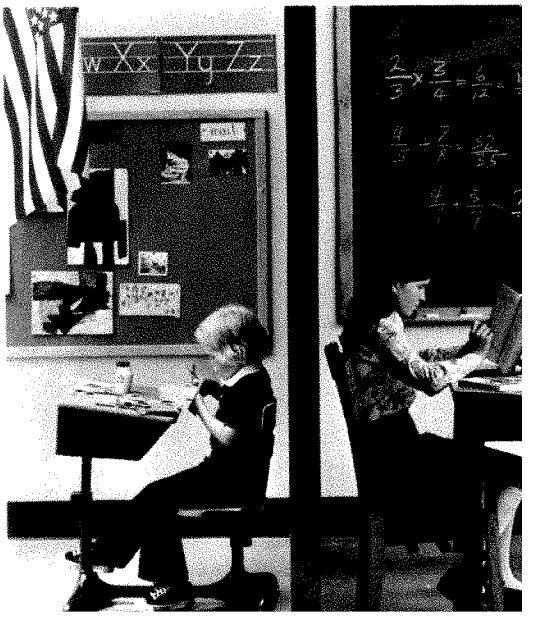
In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (teaspoonful) contains 250 mg. of primidone, in bottles of 8 fluid-ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. *37*:49 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. *47*:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. 68:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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In 14 double-blind studies of four weeks duration, 339 patients with depressive neurosis received Mellaril. In these studies, 55% of the overall improvement was observed by the end of the first week, and a total of 293 patients (86%) improved during the four weeks.*

Mellaril usually does not cause euphoria or undue sedation and is nonaddicting. Patients, therefore, generally remain alert and better able to respond to therapy. (The physician should, however, caution patients against participating in activities which require complete mental alertness, e.g., driving.)

*Data on file at Sandoz Pharmaceuticals.

MELLARIL® (THIORIDAZINE)

TABLETS: 25 mg and 50 mg thioridazine HCl, U.S.P.

for the short-term treatment of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg. should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling. The following reactions have occurred with phenothiazines and should be considered: *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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The Syndrome of Autism: A Critical Review

BY EDWARD M. ORNITZ, M.D., AND EDWARD R. RITVO, M.D.

The authors review the clinical features and behavioral characteristics of autism; differential diagnosis of the syndrome; clinical, neurophysiological, and biochemical research; and its medical management and treatment. They conclude that autism is a behaviorally defined, specific syndrome that is manifested at birth or shortly thereafter. Its symptoms are expressive of an underlying neuropathophysiological process that affects developmental rate; modulation of perception; language, cognitive, and intellectual development; and the ability to relate. The long-term prognosis is guarded; almost all patients manifest severe symptomatology throughout their lives. Further basic research into the neuropathophysiological process underlying the syndrome is necessary in order to reach the ultimate goal of developing etiologically specific treatment programs.

OF ALL THE SYNDROMES now classified as developmental disabilities, autism is one of the most difficult to understand. Wide differences in severity, periodic changes of the symptoms (1, 2), confusing and inconsistent nosology, and the lack of specific physical signs make diagnosis a difficult procedure. The behavior of autistic children is bewildering to parents, and thus it is often difficult to obtain an adequate developmental history. Unless the physician suspects autism and knows which symptoms and signs to elicit, the diagnostic process may be thwarted from the outset. Too often parents are told, "Let's wait, he is just slow in developing and will probably catch up soon."

Initial parental concern arises at different ages. A particularly observant mother may comment to her pediatrician that her 2-month-old reacts very differently than her other children did. At the other extreme are parents who do not seek medical attention until their child is 6 or 7. Most patients are usually first evaluated when 2 to 3 years old. The most frequent initial complaint is that of a delay in speech development, earlier and more subtle symptoms having been overlooked or denied. Thus autism must always be included in the differential diagnosis when one is evaluating any child with a developmental disability.

DIAGNOSTIC TERMINOLOGY

The syndrome of autism was first identified by Kanner in 1943 when he described a group of children as having "autistic disturbances of affective contact" (3). In 1944 he adopted the term "early infantile autism" and drew attention to the fact that its symptoms occurred in early infancy (4). The term "autism" and its synonyms—"childhood autism," "primary autism," "infantile autism," and "autistic child"—have become the most commonly accepted ways of referring to this condition.

"Atypical development" and "atypical ego development" have been used by psychoanalytically oriented investigators to describe similar patients (5–7). The term "symbiotic psychosis" has also been used to de-

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The authors are Associate Professors, Mental Retardation and Child Psychiatry Program, Department of Psychiatry, School of Medicine, University of California, Los Angeles, Calif. 90024.

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scribe children who are similar in many ways but who may relate differently (8). These patients at times cling tenaciously to their parents. Since this clinging behavior can alternate with emotional unresponsiveness, this term does not necessarily describe an independent disorder (2).

The labels "pseudo-retarded" and "pseudo-defective" have been used to emphasize the differences between "typical" or "simple" mental retardation and autism (9, 10). Since studies (11, 12) have demonstrated that most autistic children have intellectual and cognitive delays, these terms are not sufficiently specific to be clinically useful. The more general and less well-defined terms "infantile psychosis," "childhood psychosis," and "early onset psychosis" are also used extensively (6, 7, 11–20). They emphasize psychological damage to the developing personality of the child and, depending on the investigator, may or may not overlap with "autism."

Finally, a term that is also widely used for similar patients is "childhood schizophrenia" (1, 9, 10, 21–26). Semantic confusion and controversy exist with regard to the use of this label. Certain investigators view autism as phenomenologically distinct from adult forms of schizophrenia (16–19, 27). Others postulate a continuum of disease process from childhood to adulthood (9, 15, 28–30). The distinctions and similarities between patients labeled autistic and schizophrenic will be discussed later. Articles referring to childhood schizophrenia and schizophrenic children will be cited in this review when the clinical features of the patients conform to those described in the following section.

CLINICAL FEATURES OF THE AUTISTIC SYNDROME

Age of Onset

Autism is probably present at birth. Since the first symptoms may not be recognized or recalled by parents, two courses of the illness have been reported. In the first, deviant behavior is noted shortly after birth even though mothers are not always able to specify the subtle nature of their infants' "strange behavior." The infants may cry infrequently, not respond to companionship, and apparently not need stimulation. They become limp or rigid when held and are often described as "very good babies" who never fuss. On the other hand, they may be intensely irritable and overreact to any form of stimulation and may have flaccid muscle tone. In the second course of development, the parents report relatively normal development up to 18 to 24 months, at which time they first note symptoms. The obvious appearance of symptoms invariably occurs before 30 months of age (2, 16, 18). In our experience, the subsequent clinical course is the same regardless of the age at which symptoms are first reported. A recent report (31) of better prognosis when the apparent onset of symptoms is delayed suggests that symptoms are not recognized at birth in milder forms of the illness.

Very careful history-taking usually elicits the fact that subtle signs did indeed occur during the first year of life. These may be forgotten, overlooked, or denied due to parental anxiety or unfamiliarity with normal development.

Behavioral Characteristics

The behavioral symptoms of autism can be separated into the following five subclusters. These include disturbances of perception, developmental rate, relating, speech and language, and motility.

1. *Disturbances of perception.* Disturbances of perception are most likely due to an underlying neuropathophysiological process that is common to all autistic patients (32). It results in faulty modulation of external sensory input (2, 29, 33), distortion of the normal hierarchy of receptor preferences (34), and an impaired ability to use internal sensory input to make discriminations in the absence of feedback from motor responses (35).

The failure of adequate modulation of sensory input constitutes a striking and unique aspect of autism (2, 25, 33, 34, 36). Behaviorally, it appears as hyporesponsive or hyperresponsive states that alternate in the same child (25, 26). When hyporeactivity to auditory stimuli occurs, the patient does not react to either verbal commands or sounds. There may be no startle response to very loud sudden noises (37) and delayed attention to those (personal observation) and other sounds (38). The patient may have no visual reaction to new persons or objects in the environment, and he may walk into objects as if he did not see them. A similar lack of response to tactile and painful stimuli frequently occurs during the first two years of life: objects placed in the hand fall away, and painful stimuli such as bruises, cuts, and injections may evoke no reaction.

Contrasting starkly to such hyporeactivity are decidedly exaggerated reactions to the same stimuli. In this state, patients show heightened *awareness* and heightened *sensitivity* to stimuli (25). They may also seek out stimulation. Behaviors such as making sounds by scratching surfaces while putting the ears close to listen, being distracted by background stimuli of marginal intensity, rubbing, banging, or flicking at the ears, and teeth-grinding are all activities that induce auditory input. Visually, autistic patients may regard their hand and finger movements and scrutinize fine details of surfaces and have brief episodes of intense staring. They may induce tactile input by rubbing the surfaces of furniture or fabrics. Many behaviors suggest that they are actively inducing vestibular and proprioceptive stimulation (9, 10). For example, whirling, rocking, and head-rolling are common. Repetitive handflapping also may provide proprioceptive input.

One can often observe distress induced by routine stimuli in all sensory modalities. When in such a state, a child may become severely agitated by a siren, a vacuum cleaner, or a barking dog. He may cup his hands

over his ears in an attempt to shut out such intense sounds (25, 33). Sudden changes in illumination or confrontation with an unexpected object may elicit similar fearful reactions. In the tactile mode, the autistic patient may suddenly develop a severe intolerance for certain fabrics, and the introduction of rough-textured foods may evoke distress. The child who whirls himself may at other times show a pronounced aversion to vestibular stimulation induced by roughhouse play or riding in a car or elevator.

Autistic patients have been described as showing a preference for proximal receptors (touch, smell, and taste) rather than distal receptors (hearing and seeing) (34, 39). They also have been shown to have fewer eye movements and to spend less time regarding visual displays than do nonautistic individuals (35). Many autistic patients who have normal or even advanced form perception (40) make poor use of visual discrimination when learning. It has been observed that they are more dependent than normal individuals on feedback from their motor responses to make sense out of their perceptions (35).

2. *Disturbances of developmental rate.* Autistic children show deviations from the normal sequential motor, language, and social milestones (1). In particular, the even course of normal development is disrupted (41). For example, an autistic child may precociously sit without support but be delayed in pulling himself up to a standing position. Sequences of spurts and plateaus are characteristic (1), as are the "special abilities" of some otherwise very retarded autistic children (42). These observations can be used to distinguish them from other types of severe retardation (43).

3. *Disturbances of relating.* Behaviors that indicate disturbances of relating are due to arrests or developmental delays in personality formation. Behaviors indicative of such ego pathology include poor or deviant eye contact (44); delayed or absent social smile; delayed or absent anticipatory response to being picked up (3); apparent aversion to physical contact; a tendency to react to only a part of another person; disinterest in playing games with others; and delayed, absent, or overreactive anxiety to strangers (27). Disturbances in relating may be subtle and intermittent.

Experiments conducted under well-controlled laboratory conditions suggest that disturbed relatedness in autism may be secondary to disturbances of perception (35). Disturbed relatedness to objects is expressed by flicking, twirling, and spinning toys rather than using them in an apperceptive manner. This observation led Kanner and Eisenberg (3, 45) to conclude that these children wanted "to maintain sameness in their environment." We believe that they are upset by observing different perceptual sets and hence try to maintain "sameness." With increasing age, the autistic child's disturbances in relatedness lead to rigidity and inflexibility in the use of play material (46), inability to form peer relationships, and intermittent responsiveness to parents.

4. *Disturbances of speech and language.* Speech and language development may be totally delayed (muteness), or fixations may occur along the normal course of development (3, 10, 47). Echolalia is a common feature and is usually accompanied by the misuse or reversal of pronouns (3, 27). Echoing of feelings as well as words occurs. When functional speech develops, it is usually atonal and arrhythmic, lacking inflection and failing to convey subtle emotion (26). The atonal and poor affective qualities of speech in young autistic children usually persist into adulthood even in those few children who develop communicative language.

5. *Disturbances of motility.* The strange and bizarre appearance of many autistic children is due to their peculiar mannerisms and patterns of motility. These may be complex and repeated serially (stereotypies), and they do not appear to be involuntary movements typical of seizure patterns. Disturbances of motility may appear intermittently in some autistic children; these disturbances may be continuously present in others (48, 49).

The most typical motility disturbance involves the hands and arms (2, 48). The hands usually are moved within the visual field. Laboratory studies have demonstrated that the motility disturbance of the hands (hand-flapping and oscillating) occurs at the same frequency over time and that groups of patients flap at the same rate (50). Furthermore, these behaviors are relatively unaffected by environmental factors such as the presence of other persons or toys (51). These studies have led us to conclude that disturbances in motility can best be understood as an expression of a central nervous system (CNS) dysfunction. Some researchers have explored the possible significance of the relationship between motility and perceptual disturbances for a neuropathophysiological explanation of autism (36, 52, 53). Motility disturbances may also involve the feet, as in the case of toe-walking. Toe-walking is frequently intermittent while the autistic child is running, but it may also be continuous (54).

Motility disturbances of the trunk and the body include staccato lunging and darting movements, body-rocking and swaying, and head-rolling or head-banging. These disturbances are not constant and do not have the persistent, irritable quality typical of children labeled "hyperactive." In fact, the motility patterns of autistics are usually interrupted by episodes of immobility and posturing. For example, patients less than 3 years of age may be observed to arch the back and hyperextend the neck for several minutes at a time.

When examining a patient, we have him look at a spinning top. This frequently elicits motility disturbances such as hand-flapping, posturing, and twirling.

A detailed behavioral description of symptoms is important in order to avoid misdiagnosis. Nonspecific descriptions such as "the child shows bizarre behaviors" are insufficient. We have found that the accuracy of diagnosis is enhanced by our conducting at least three separate diagnostic examinations at weekly inter-

vals. While parental descriptions of the child are necessary, they are clearly not sufficient to establish the diagnosis of autism.

DIFFERENTIAL DIAGNOSIS

For clinical and research purposes, we reserve the diagnosis of autism for patients who clearly show, prior to 36 months of age, irregularities in development and disturbances in the modulation of sensory input, relatedness, and language as described in the previous section. The presence of these symptoms is necessary and sufficient to establish the diagnosis. While motility disturbances are frequently present, they are not necessary to establish the diagnosis. Seventy-six percent of a group of 72 autistic patients we studied had a history of hand-flapping (55).

When reviewing the differential diagnosis, one must consider specific diseases that are known to occur in association with autism. These include phenylketonuria, congenital rubella, other specific organic brain syndromes, mental retardation, and seizure disorders. In addition, one must differentiate diseases with overlapping symptoms, such as environmental deprivation (maternal deprivation and hospitalism), anaclitic depression, developmental aphasia, and sensory deficits. This last category includes syndromes that have been described in the research literature under the rubrics of disintegrative psychosis (56); late onset psychosis (16); acquired autism (57); and childhood schizophrenia, nonorganic type (26).

Figure 1 shows the possible etiological and phenomenological interrelationships among the clinically and behaviorally defined syndromes of autism, primary cognitive and intellectual retardation, and psychologically induced psychosis (severe ego pathology). This model is based on the hypothesis that the syndrome of autism results from CNS pathology of a specific type. This neuropathophysiology may occur on an idiopathic basis or in conjunction with specific diseases of known etiology that affect the CNS. The same neuropathophysiological process underlying autism may also produce cognitive and intellectual retardation and disrupt ego development, producing secondary psychosis. According to this model, primary ego pathology (psychosis) can produce cognitive and intellectual retardation and vice versa, but neither of these alone can "cause" autism.

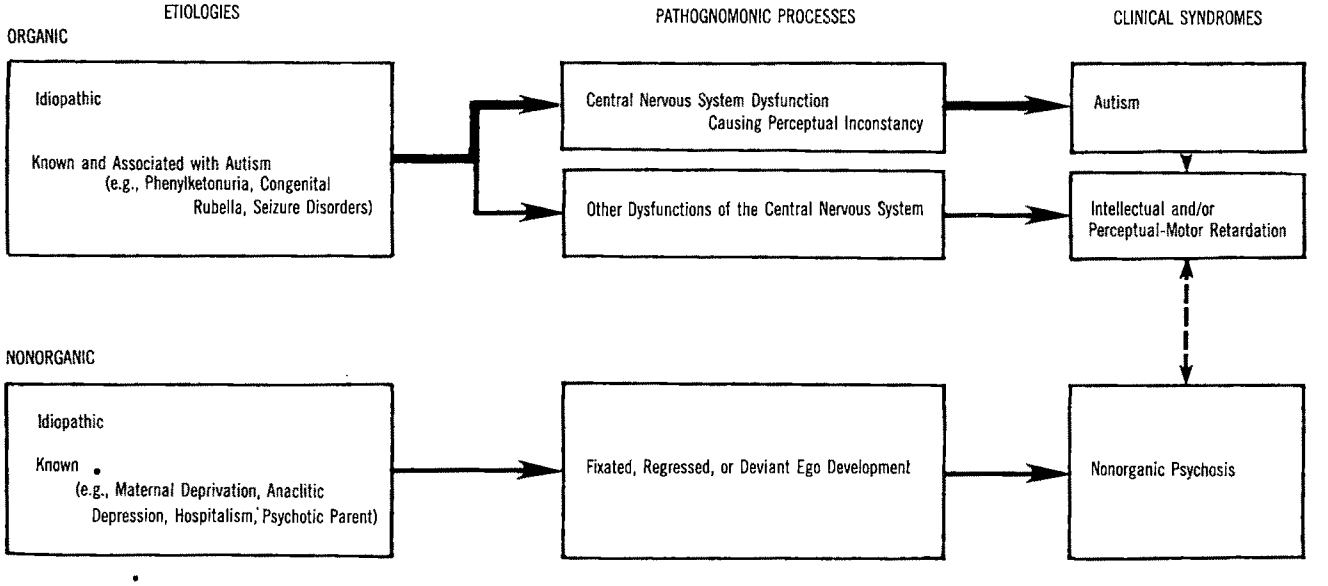
Idiopathic CNS Dysfunction

This is the category in which most young autistic children have to be placed. However, as an individual patient grows older, there is an increasing likelihood that evidence of specific CNS pathology will become manifest by the onset of specific signs or symptoms (e.g., seizures), and the designation "idiopathic" will be revised (58).

Major Sensory Deficits

Specific sense organ pathology can precipitate severe emotional reactions and developmental arrests in infants and young children. The combination of sensory deficits and secondary ego disturbances may result in a clinical picture that can be confused with autism (59). Therefore, since disturbances of language are part of the syndrome of autism, specific otological and audiological examinations should be carried out (60). Complete or partial blindness is often accompanied by "blindisms" (61). These mannerisms, which

FIGURE 1
A Model of the Possible Etiological and Phenomenological Interrelationships Among Autism, Primary Cognitive and Intellectual Retardation, and Psychologically Induced Psychosis



include gesturing with the arms and hands, do not have the repetitive stereotyped characteristics of the hand-flapping seen in autistic patients, and they occur in response to environmental stimuli. Furthermore, blind children usually develop an interest in their environment and do not have disturbances in relating typical of autism. When their visual deficit is recognized and if other CNS pathology does not interfere, they can develop quite normally. It is of interest that autism has been reported in association with retrolental fibroplasia but not with other types of visual impairment (61). Since retrolental fibroplasia is associated with generalized brain damage, autism in these cases probably results from the CNS impairment and not from the visual deficit per se.

Developmental Aphasia

Both developmental aphasia (27) and autism are accompanied by abnormal responses to sounds, delay in the acquisition of language, and difficulties in articulation (62). Due to their difficulties with expressive and/or receptive language, aphasic individuals may develop secondary disturbances in relating and social responsiveness. They do not, however, manifest the perceptual disturbances (sensory hyperreactivity and hyporeactivity) characteristic of autistics. They can relate by nonverbal gestures and expressions, are sensitive to the gestures and expressions of others, and can learn to point toward desired objects (63). When they do acquire speech, it does not show the lack of "communicative intent and emotion" (64) and delayed echolalia (65) characteristic of autism. Thus the language disability of speaking autistic children is more severe and more deviant than that of aphasic children (66).

Cognitive and Intellectual Arrests and Delays

Clinical experience with autistics and follow-up studies demonstrate that low scores on developmental tests or reports of "untestability" during the early years are associated with retarded intellectual functioning later in life (11, 12). Most autistic individuals cannot perform many IQ test items (43, 67). The notion that autistics have a primary affective deficiency that interferes with the expression of their assumed normal or superior intellectual and cognitive potentials has given way to the recognition that their intellectual deficiencies are every bit as real as those of patients with primary retardation. Accumulating clinical evidence indicates that two-thirds to three-fourths of all autistic patients will perform throughout life at retarded levels (58). Thus mental retardation and autism can clearly coexist (68).

Specific Organic Brain Syndromes

Some children with autism also show evidence of specific organic brain syndromes. Attempts to link autism with Schilder's disease and Heller's syndrome (69) have not been successful since the natural history and clinical course of these degenerative diseases are different. Autism has been reported *in association with* prenatal and perinatal complications (70), neonatal conditions associated with brain damage (e.g., retrolental fibroplasia) (61), infantile spasms (14, 16, 71), cerebral lipoidosis (14), metabolic diseases (e.g., phenylketonuria) (27, 48), Addison's disease (72), celiac disease (73), and infectious diseases (e.g., congenital rubella) (74). An extensive study of a series of patients with rubella revealed that 8% to 10% had specific symptoms indicative of autism (75). Conversely, a number of patients first diagnosed as autistic were shown to have had congenital rubella (74).

Seizure Disorders

Alterations in consciousness and behaviors due to epileptic seizures must be distinguished from autism, particularly since momentary posturing and bursts of hand-flapping, frequently seen in autism, may be confusing. Autism and seizures frequently coexist (14, 16), and it has been demonstrated that two EEGs with sleep tracings are required to evaluate this possibility (76). Seizure disorders are more likely to become clinically manifest in autistic children as they become older. Twenty-five percent of a well-followed series of autistic children first developed seizures between 11 and 19 years of age. Most of these children had had normal EEGs and neurological examinations earlier in life (11, 63). Both grand mal (16, 63) and psychomotor seizures have been observed in autistic children (77).

Maternal Deprivation

It has been suggested that deficient human stimulation, e.g., maternal deprivation, may cause autism (78). The immediate and long-term sequelae of environmental deprivation in infants raised in institutions (79) and at home (80) have been well documented. While environmental deprivation has been shown to induce serious developmental disturbances, it does not produce the syndrome of autism.

Deprived patients characteristically exhibit a uniform delay in the acquisition of motor skills, speech, and the adaptive use of toys. In contrast, autistic children show uneven motor and speech development characterized by spurts and lags. Deprived infants may show unusual bursts of motor activity such as athetoid-type movement of the hands. However, these are not like the repetitive hand-flapping and other motility disturbances seen in autistic children. In addition, the whirling, toe-walking, darting, and lunging movements of autistics have not been observed in deprived patients. These patients do, however, engage in total body-rocking and hand-posturing. These behaviors are easy to interrupt or totally eliminate, in contrast to the more intractable motility disturbances in autistic children.

Deprived patients may also show abnormal patterns of relating and may adapt poorly to being held. They may flick at toys, drop them, or fail to develop an interest in them. In contrast, autistic patients usually use toys in peculiar ways such as spinning, feeling, or

sighting along them. Deprived patients usually fail to seek out adults when given the opportunity. Unlike autistic children, who do not appear to value eye contact, deprived children may intensely engage in visual contact with adults. While autistic patients rarely develop an active interest in playing games with others, deprived children do, although their interest develops later than in normal children. Similarly, their language development may be delayed, but they usually catch up when their deprivation is relieved. Once speech has been acquired, it does not have the atonal, arrhythmic, hollow-sounding quality of autistic speech. Echolalia and misuse of pronouns has not been reported in deprived children, nor have perceptual disturbances indicative of faulty modulation of sensory input. While autistic patients engage in tactile exploration, deprived patients show diminished tendencies to touch themselves and to put objects in their mouths.

A difficult problem in differential diagnosis occurs if a mother has had a postpartum depression or other serious emotional disorder during her child's first year. If an adequate surrogate was not present, the child may have developed an environmental deprivation syndrome. On the other hand, the ill mother might have given birth to an autistic child whose inability to positively relate and respond aggravated preexisting disturbances in the mother.

Anaclitic Depression

Anaclitic depression develops after the sixth month when there is an interruption in a previously adequate mother-infant relationship (81). The interruption may be due to death, psychological withdrawal, or temporary absence of the mother. Such infants become weepy, demanding, and clinging. If the mothering person does not return within a few weeks, this stage may presage psychomotor retardation, delay in development of language, weight loss, and persistent crying. After 2 or 3 months, these signs are replaced by quiet whimpering, facial rigidity, and finally lethargy and apathy. Symptoms of perceptual disturbances, developmental spurts and lags, and disturbances in language and motility characteristic of autism are not seen in children with anaclitic depression. Their failure to relate to adults is characterized by apathy and withdrawal.

Relationship of Childhood Schizophrenia to Autism

The diagnosis of childhood schizophrenia has been applied to a wide spectrum of patients. These range from autistics, as defined in this review, to children without severe developmental or intellectual disabilities but who have psychotic level ego functions. Clinical subtypes have been defined by Bender and associates (10, 21, 82–84) (pseudoretarded or autistic, pseudoneurotic, pseudopsychopathic), Fish (41) (according to language and intellectual development), and Goldfarb (26) (organic, nonorganic). Their use of the term implies that there is a continuum of schizophrenic illness from the prenatal period (maturational lag at the embryonic level, according to Bender and associates) to adulthood, with different ages of onset (onset of autism, prior to 3 years; onset of childhood schizophrenia, 3 years to adolescence; and onset of adult schizophrenia, adolescence or later).

In contrast, Rutter (56) has suggested that the term "childhood schizophrenia" is confusing and should be abandoned. In his view, any patient, regardless of age, whose clinical picture fits the classic description of schizophrenia should be so labeled. He considers autism a separate disease that is first manifested during the first 3 years of life. Once established, it should then remain the primary diagnosis regardless of increasing age and any change in symptoms. He also identified a separate group of children in whom psychotic ego development occurs between the ages of 3 and 5 after an apparently normal course of development and labeled these rare cases "disintegrative psychosis."

In support of the position that there is a continuum of one disease process that includes autism and childhood schizophrenia are clinical reports that when some typically autistic preschoolers reach ages 8 to 12, their clinical picture has changed to fit the criteria of childhood schizophrenia (28). In addition, there are reports of typically autistic children who, as adults, exhibit clinical signs of schizophrenia (82–84) and of some adult schizophrenics whose histories indicate they were typically autistic when preschoolers (85).

We have been able to observe several such cases. One boy, at 4 years of age, clearly met our criteria for autism (developmental delays and disturbances in the modulation of perception, relatedness, language, and motility). He was seen again at age 9 and asked why he rocked and put his hands up before his face. He said, "The floor is coming up at me" and "The walls are moving in on me." Another bright, verbal boy had a similarly documented early history of autism. At age 9, when asked why he flapped his hands before his face, he said, "I am pushing the thoughts back in my mouth." When he was younger, his motility disturbances had included hand-flapping. He was obviously trying to consciously explain, albeit on a psychotic level, the persistence of this symptom.

In support of the alternative hypothesis, namely that autism and childhood schizophrenia are distinct syndromes and should be carefully distinguished, Rutter (56) detailed the following differences: 1) familial factors (absent in autism but present in schizophrenia), 2) ages of onset, 3) symptoms, and 4) natural history and course of the disease.

For heuristic purposes, we are exploring theoretically and in the laboratory the possible neuropathophysiological processes that may produce the symptoms common to both schizophrenia and childhood autism, whether they are the same syndrome or not (29, 30). It is incumbent upon each physician to understand both the descriptive elements of the two terms and that these nosological differences have more than just semantic meaning. Indeed, they have marked implications for total patient management and for the

application of specific supportive treatments. For example, developmentally retarded autistic patients with severe language disturbances, as described in this review, respond best to a combination of behavior therapy and special education (86). On the other hand, the thought and affective disorders typical of childhood schizophrenia respond best to a combination of psychotropic medication, psychotherapy, and milieu therapy (87).

CLINICAL RESEARCH

Prenatal and Perinatal Factors

Several studies have shown that premature birth is not significantly associated with autism (16, 88, 89). Other reports have shown that certain prenatal and perinatal complications are more common in autistic individuals than in control groups (16, 70, 88–90). When all prenatal and perinatal complications are considered, there is a suggestion that they may be significantly associated with autism (16, 70). These findings must be viewed with caution due to the lack of uniformity in both the diagnostic criteria for autism and in the selection of prenatal and perinatal complications by the investigators.

Constitutional and Genetic Factors

With the exception of two studies (91, 92), neither an increased incidence of schizophrenia nor a history of autism has been reported in the parents or nontwin siblings of autistic children (16, 18, 20, 93). Twin studies have suggested that monozygotic pairs are likely to be concordant for and dizygotic pairs discordant for autism.[1] It is of particular interest to note that autism occurs in diverse racial populations and in all parts of the world.

Attempts to find chromosomal abnormalities in autistic patients have been unsuccessful (94). Studies reporting unusual dermatographic patterns (95) and hand morphology (96) in some "psychotic children" with "autistic features" have yet to be replicated.

Family Characteristics

The old notion that parents of autistic children are highly intelligent, preoccupied with abstractions, have limited interest in people, and are emotionally "cold or refrigerated" (45) is not supported by recent research (16, 63, 97). In addition, the concepts of unconscious parental hostility and rejection (98), parental "perplexity" (99), and lack of communicative clarity (100) do not apply to the parents of autistic children.

Controlled studies have shown that the extreme emotional stress of having an autistic child can induce or precipitate emotional disorders in susceptible parents (91). Furthermore, the physician's assumption that there is something wrong with the parents which causes autism may produce a iatrogenic emotional disorder in the parents (101). The possibility of disturbances in family dynamics at an early critical period has also been suggested as etiological (5–7). However, no objective evidence has been presented to support this hypothesis, and other studies have found normal family structure concurrent with autism (102).

Since Kanner first described the syndrome in 1943 (3), several reports have suggested that parents of autistic children are of above-average intelligence (27, 45, 88, 91, 93). These studies used group tests, which are known to result in higher scores for parents from higher social classes (18). Two well-controlled comparisons of the verbal IQs of mothers and fathers of autistic and normal children matched for socioeconomic status found no significant differences (103, 104). These findings are confirmed by our unpublished data and clinical experience.

Several reports have suggested that families of autistic children have a higher social class distribution than would be expected to occur randomly or when compared with other specific groups of patients (3, 16, 20, 88, 91, 93, 105). These results have *not* been confirmed by three other surveys. The first of these compared the distribution of autistics and "non-autistic psychotics" among five social classes (106). Ritvo and associates (107) compared the families of autistics with those of matched nonautistic patients and the expected distribution based on population data. A third study compared families of autistic patients with those of matched normal subjects (103).

Studies of Prevalence

Lotter (108) found a prevalence of 4.5 autistic children per 10,000 8- to 10-year-olds in Middlesex County, England. Treffert (105) found a prevalence of 0.7 per 10,000 in the state of Wisconsin. He surveyed children aged 3 to 11 and used different epidemiological techniques and possibly different diagnostic criteria than Lotter. Both prevalence figures can be considered low since autistic children younger than 4 and older than 6 are often misdiagnosed. In any event, autism is not so rare a condition as was once thought, and it represents a significant social problem.

Studies of Prognosis

Between 7% (14) and 28% (58) of autistic children who have shown no clinical evidence of neurological disease in early childhood develop seizures by age 18. Approximately 75% of all autistics are classified as mentally retarded throughout life (15, 58). Those who have seizures and other indications of specific organic brain dysfunction also tend to be the most retarded, underdeveloped, and impaired (58). Failure to develop communicative language by age 5 is also highly correlated with poor prognosis for further personality development (11, 15, 24, 45). Failure to develop the capacity to use toys appropriately is also associated with a poor prognosis (109). Thus the 5-year-old autistic child who does not have communicative language, i.e., remains mute or solely echolalic, does not play appropri-

[1]For a review of this subject see reference 52.

ately with toys, and has severe cognitive deficits has the poorest prognosis. Autistic children with low IQs show the poorest response to treatment (110); these patients will most likely require lifelong, 24-hour care.

The minority of autistic children who show relatively normal intellectual, cognitive, and motor development and have communicative language before age 5 have a somewhat different prognosis. Clinical experience indicates that most of these patients do not develop a thought disorder with delusions and hallucinations (58). However, most of the patients in this group become extremely shy, introverted, and passive adolescents. Some develop disturbances in reality testing typical of pseudoneurotic childhood schizophrenia or the borderline state (9, 15, 21). A study of one series of cases (13, 28) and several individual case reports document transitions from typical autism to typical schizophrenia in late childhood (2), adolescence (29), and adulthood (82–85). As previously noted, follow-up studies of the relationship between early autism and later schizophrenia deserve consideration (29, 30).

Although it has been reported that some autistic adults are able to live alone and obtain employment, we must emphasize that they represent a small minority of cases. Even those with the best combination of good prognostic factors will probably have significant residual personality and cognitive impairments in adolescence and adulthood (13). Thus even those exceptional patients who show significant improvement during childhood remain aloof and literal and lack social judgment and empathy (111).

Neurological and EEG Studies

The findings from neurological examinations of most autistic children are unremarkable (112). However, "soft" neurological signs such as poor muscle tone, poor coordination, clumsiness, hyperactive knee jerks, hypotonia, generalized hyperreflexia, drooling, short attention span, hyperkinesis, hypokinesis, ankle clonus, and strabismus have been reported in 40% to 75% of several series of patients (26, 89).

EEG findings are quite variable. Two reports of unusually low voltage EEGs suggestive of hyperarousal (16, 49) were not confirmed in two other studies (113, 114) when stimulus conditions were controlled. While some researchers have reported that 50% to 80% of autistic patients have abnormal EEGs characterized by focal or diffused spike, slow wave or paroxysmal spike and wave patterns (89, 113, 115), others have reported a lower incidence of EEG abnormalities (16, 112).

NEUROPHYSIOLOGICAL RESEARCH

Because of the usual extreme lack of cooperation by autistic children, neurophysiological studies have been quite limited in scope and number. Autistic children have a normal sleep cycle with normal amounts of rapid eye movement (REM) sleep (116). However, the REM activity of REM sleep is reduced in autistic children and is similar to that found in normal infants, suggesting a maturational defect (117). While the transcephalic direct current potential is similar in autistic and normal children, an unusually high percentage of DC bursts have been found in 6- to 13-year-old autistic children and 1- to 5-year-old normal children (73). Unfortunately, that study did not have adequate controls for eye movement activity, so this interesting maturational finding may only reflect the tendency of older autistic and younger normal children to have more vertical eye movements.

The contingent negative variation (CNV) is intact in autistic children when two stimuli are paired together, but the autistics, unlike normal controls, do not show a differential response to slides of familiar and strange faces (118). This does not seem to represent a neurophysiological abnormality since the basic ability to establish a CNV is intact; rather, it reflects the ordinary clinical experience that autistic children do not show adequate differential responses to strangers and do not participate in social interaction.

Studies of auditory evoked responses (AER) during sleep have revealed only marginal differences between autistic children and controls and have been characterized by great variability from subject to subject (119). Studies of the recovery cycle of the AER during sleep have also demonstrated normal responses in autistic children (120). One study of evoked responses to flashes and clicks in waking autistic children was inconclusive since no statistical analysis of the data was presented (121). An inadequate galvanic skin response to both auditory and visual stimuli has been demonstrated in autistic children (122). This finding is consistent with the clinical observation that autistic children do not show an adequate startle response. It was not possible, however, to replicate the finding by using changes in heart rate as the experimental measure (123). Increased variability in the heart rate has been reported, but adequate control of the activity level and stimulus conditions under which autistic and normal children were observed was not documented (124). Thus this assertion that evidence was found for brain stem dysfunction is somewhat tenuous.

However, other evidence implicating a brain stem disturbance has been found. Three separate studies have demonstrated a consistent suppression of vestibular nystagmus in autistic children (125–127). Ornitz and associates (128) have demonstrated that this finding is not dependent on visual fixation but rather reflects a pathophysiological interaction of the visual and vestibular systems. It has also been found that vestibular stimulation does not influence the REMs of REM sleep in autistics as it does in normals (53). Ornitz (129) has discussed the possible significance of these investigations for a theory of brain stem dysfunction involving the central connections of the vestibular system. He also suggested that there may be a strong motor component to the perceptual processes of autis-

tic children (129). This notion is supported by a report that slow waves (in AERs) provoked by movement appeared in autistic children during sensorial conditioning that required no movement (130).

METABOLIC, BIOCHEMICAL, AND HEMATOLOGICAL RESEARCH[2]

There have been several studies of indoleamine metabolism in autistic patients. Although the results were preliminary and inconsistent, they suggest that this field of investigation requires further serious study. One study (131) demonstrated decreased urinary excretion of 5-hydroxyindoleacetic acid in response to a tryptophan load, but this finding was not replicated in a later study (132). Heeley and Roberts (133) observed the effect of tryptophan loading and reported results that suggested abnormal function of the kynurenine pathway. Sankar and associates (134) found increased amounts of indolic substances in the urine of 5- to 16-year-old hospitalized "schizophrenic" patients. However, they did not provide sufficient clinical information to determine whether these children could be considered autistic as described in this review. In contrast to the findings of Sankar and associates, Jorgensen and associates (135) observed a significant reduction in the urinary excretion of tryptophan in 3- to 6-year-old "psychotic children." Another study (136) reported the presence of bufotenin (N,N-dimethylserotonin) in the urine of some autistic subjects; the significance of this finding is still uncertain.

Another group of studies has focused on the level of serotonin in peripheral blood, where it is transported in platelets. In 1961 Schain and Freedman (137) reported that 25% of a group of 23 institutionalized patients diagnosed as autistic had elevated blood serotonin levels. Ritvo and associates (138) found a significantly higher amount of whole blood serotonin in five autistic children 36 to 47 months of age than in seven age-matched normal children but no significant difference between older autistics and their age-matched controls. They also noted that platelet counts decreased in normal children aged 2–8 years and increased in autistic children through that age range; however, these differences did not reach statistical significance. Yuwiler and associates (139) were unable to explain these results on the basis of differences between autistic and nonautistic children in circadian rhythmicity for either whole blood serotonin or platelets.

Ritvo and associates (140) also studied the effect of prolonged (6-month) administration of L-dopa on autistic children. Blood serotonin levels decreased in three out of four autistics and platelet counts tended to increase. However, no change was rated in the clinical course of these patients or in their sleep patterns while they were receiving L-dopa. Zarcone and associates (141) reported that chronic oral ingestion of the serotonin precursor 5-hydroxytryptophan had no effect on the behavior of two autistic children. However, it did cause an increase in REM sleep time and in the number of REMs during REM sleep, an effect that also occurs in normal subjects.

A group of related investigations into the transport of serotonin by blood platelets has been conducted. Sankar and associates (134) found that the in vitro rate of uptake of serotonin by platelets was less in "schizophrenic" children than in a control group of hospitalized patients. Boullin and associates (142) reported an increased efflux of serotonin from the platelets of patients diagnosed as autistic by the Rimland E-2 questionnaire. They did not find any unusual differences in uptake or loss of dopamine from the platelets of autistics similarly diagnosed. We have been unable to replicate findings of different uptake and efflux rates between autistics and carefully matched control subjects despite our use of similar and improved methods (143, 144).

A number of other biomedical measures have been reported. The plasma cortisol level, eosinophile count, and histamine wheal test do not reveal remarkable findings in autistics. Plasma and erythrocyte cholinesterase activity, serum magnesium, copper ceruloplasmin, and ascorbic acid levels are normal in autistic patients. Baseline free fatty acid levels and plasma free fatty acid response to glucose ingestion are also reported to be within normal limits, except for increased variability in the baseline plasma free fatty acid level. These may be associated with greater variability of daily caloric intake. In one study (145), plasma and red blood cell inorganic phosphate were found to be elevated in autistic subjects. One report (146) described pituitary hypofunction (reduced secretion of the gonadotropins adrenocorticotropin and follicle-stimulating hormone) in psychotic patients with the onset of illness in the first 3 years and behavior suggesting autism; this study should be replicated.

A study of smears of peripheral blood from "schizophrenic children" (147) showed an atypical leukocyte pattern. These abnormalities were similar to those associated with increased antibody production, responses to viral infection, and allergic sensitivity. They could not be distinguished from those found in blood smears of children with colds or asthma. These results may thus reflect the presence of an infectious disorder. Preliminary reports from one laboratory (148) on a serum factor that increases the susceptibility of mice to seizures deserve further investigation in view of the increased incidence of seizures with increased age in autistics (14, 58).

MEDICAL MANAGEMENT AND TREATMENT

We must emphasize that there is no rational or specific treatment for autism. In spite of strong claims by partisans of particular treatment approaches and much

[2]A more complete list of references to these studies appears in the review by Ornitz (52).

dedicated effort that has gone into their implementation, no treatment has been demonstrated to alter the natural history of the disease (149). Many different approaches have been attempted including family therapy, psychotherapy and counseling for parents, psychotherapy for the autistic child, behavior therapy, speech therapy, and special education. Therapeutic approaches also include day treatment, residential treatment, psychiatric hospitalization, medication with a number of different psychotropic drugs, hormones, megavitamins, electroconvulsive therapy, sensory stimulation, sensory isolation, and even LSD. The literature on these various treatment approaches and the application of the different treatment modalities has been critically reviewed elsewhere (52, 150).

A recent review by Campbell (151) confirmed our clinical experience that psychotropic medications do not alter the natural history or course of autism. However, they are useful adjuncts to treatment on a symptomatic level. The target symptoms amenable to medication (e.g., hyperactivity, aggressiveness, and sleep disorders) are not specific to autism. Furthermore, we also concur with Campbell that excessive sedation often results when therapeutic levels of psychotropic medication are reached. This unwanted side effect can reduce the patient's participation in other therapeutic programs (e.g., educational ones). Thus we reserve the use of psychotropic medications for those cases where behavioral-based therapies alone have proven ineffective.

Since autistic patients vary greatly in their intellectual and cognitive capacities, use and understanding of language, general developmental level, age at the time symptomatic treatment is instituted, level of ego development, and family circumstances, it is not surprising that some treatment approaches have ameliorated symptoms in certain cases but not in others. Response to a symptomatic treatment is determined primarily by the degree of impairment and secondarily by the type of treatment (109). Thus, although autistics may improve in specific respects, they remain definitely autistic.

We (52, 150) and others advocate early recognition, continued medical supervision, and a flexible approach to treatment. Periodic reevaluations are necessary to adapt supportive treatments to changes in developmental level and symptoms (151). Parents should be viewed as paraprofessionals who frequently can best implement symptomatic treatments such as behavior therapy. Patience is the hallmark of management, and one must recognize that spontaneous improvements and regressions are likely to occur despite the influence of the most optimistically presented symptomatic treatment plans.

CONCLUSIONS

Ontogeny recapitulates phylogeny in the history of diseases as well as man. Thus, since its conception a scant 30 years ago, autism has undergone many vicissitudes. It was conceived as an adjective ("autistic disturbances of affective contact"), relegated to symptom status ("mental retardation with 'autistic features' "), made a stepchild of a stepchild ("childhood schizophrenia, autistic type"), and finally emerged as a noun (the syndrome "autism").

In this review we have tried to survey those studies which were based upon serious scientific efforts and avoid those which simply expressed opinions. The current state of our medical art leads us to the following conclusions.

1. Autism is a clinically and behaviorally defined specific syndrome.
2. It is manifested at birth or shortly thereafter and remains throughout the lifetime of the patient.
3. Its symptoms are expressive of an underlying neuropathophysiological process that affects developmental rate, the modulation of perception (sensorimotor integration), language, cognitive and intellectual development, and the ability to relate.
4. No known factors in the psychological environment of a child can cause autism.
5. To establish the diagnosis it must be demonstrated that before the age of 36 months the patient had specific disturbances in developmental rate, perception, relatedness, and language. Many, but not all, patients demonstrate typical motility disturbances.
6. Autism occurs on an idiopathic basis or in conjunction with other diseases that affect the CNS.
7. It afflicts children in all parts of the world, of all racial and ethnic backgrounds, and is found in families with the normally expected distributions of intelligence, social class, and personality factors.
8. No etiologically based rational treatment is available that alters the course of the disease.
9. Complete medical and neurological evaluations are essential to establish the diagnosis, and they should be repeated at yearly intervals. Particular attention should be paid to the likelihood that seizure disorders may appear in individual patients as they grow older.
10. Supportive and symptomatic therapeutic measures (e.g., behavior therapy, special education, and residential treatment) are helpful in most cases. These should be monitored by a physician-in-charge and administered by specialists and parents trained in these modalities. Periodic reevaluations and updating of supportive therapeutic programs are necessary to keep abreast of the clinical changes that can be expected to occur in the majority of cases.
11. The long-term prognosis, unfortunately, is guarded. Approximately two-thirds of all patients manifest intellectual and cognitive deficits severe enough that they remain classified as retarded throughout their lives.
12. Further basic research into the neuropathophysiological process underlying the syndrome is necessary if we are to develop a rational treatment program, which is the ultimate goal of our efforts.

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EEG Sleep Changes as Predictors in Depression

BY DAVID J. KUPFER, M.D., F. GORDON FOSTER, M.D., LOUIS REICH, M.D., KENNETH S. THOMPSON, AND BRIAN WEISS, M.D.

The authors conducted a study of 18 depressed patients to see whether EEG sleep measurements might provide a predictive tool for response to antidepressant medication. They found that although the sedative characteristics of amitriptyline did not differentiate good responders from poor responders until the third week of drug treatment, the good responders showed significant increases in REM latency, decreases in REM sleep time, decreases in REM sleep percent, and decreases in REM activity after only 2 nights of drug treatment.

THE CORRECT DIFFERENTIAL DIAGNOSIS and appropriate treatment of any psychiatric syndrome are of obvious importance, but they are particularly vital in the treatment of affective disease because therapeutic response may not begin to occur until several weeks after the initiation of treatment (1, 2). Clinicians have known for a long time that the tricyclic antidepressants do not begin to act in the treatment of recurrent depression for at least 10 to 14 days (3, 4). Indeed, it has been argued that a 3-week trial of this medication at therapeutic levels is necessary to assess its effectiveness. Therefore, the development of prognostic criteria for treatment response in affective disease would be extremely helpful to both the practicing clinician and the clinical investigator (5).

Recently there have been several attempts to determine who is likely to respond to tricyclic therapy that have used clinical data and psychobiological measurements obtained in the pretreatment phases of hospitalization (6). Responders to tricyclics might also be determined by monitoring physiological or psychological measurements during the first few days and nights of drug administration. Both of these research strategies imply the applicability of psychobiological measurements in the differential diagnosis and treatment of affective states.

As reported previously (7), EEG sleep measurements can be used as a diagnostic indicator in unipolar and bipolar depression. Investigations have shown that shortened REM latency is a feature of primary depression and, furthermore, that the hypersomnic-hyposomnic determination may help categorize the depression as either bipolar or unipolar (8–10). Even though a more practical nosology of depressive syndromes will probably be based in part on such psychobiological measurements as sleep and motor activity, the development of prognostic indicators aided by psychobiological measurements is of equal importance. With this in mind we decided to study EEG sleep in relation to the administration of tricyclic antidepressants to determine whether treatment response could be predicted on the basis of EEG sleep during the first few nights of drug administration.

This investigation was made easier by the fact that our previous studies have shown that patients do not always need to be studied longitudinally; in fact, one can ascertain the main EEG sleep features with regard to primary depression on the basis of even two nights' study (11). We were therefore able to examine the interaction among clinical changes, drugs, and EEG sleep using a design involving intermittent EEG sleep recording.

METHOD

We studied 18 patients (11 women and 7 men with a mean age of 51.4±2.3 years) who had been admitted to our clinical research unit with the diagnosis of unipolar depression. As in our previous studies, all of the patients had been drug free for a minimum of 2 weeks before the study, were selected on the basis of independent evaluations by at least two attending psychiatrists, and gave informed consent. Clinical diagnoses were made on the basis of the criteria described by Feighner and associates (12). In addition, systematic assessments of behavioral dimensions with emphasis on depressive and psychotic symptoms were obtained by using self-rating and clinical rating scales (13–15).

All of the patients were studied in their rooms on the research service by means of continuous nightly recordings of their EEGs, horizontal electrooculo-

Dr. Kupfer is Professor of Psychiatry and Director of Research and Dr. Foster is Assistant Professor of Psychiatry and Director of Clinical Research, University of Pittsburgh Western Psychiatric Institute and Clinic, Pittsburgh, Pa., where Dr. Reich was Assistant Instructor, Mr. Thompson was Research Assistant, and Dr. Weiss was Assistant Professor of Psychiatry. Dr. Reich is currently Chief, Inpatient Service, USAF Medical Center, Keesler Air Force Base, Miss., and Dr. Weiss is currently Assistant Professor of Psychiatry, University of Miami School of Medicine, Miami, Fla. Address reprint requests to Dr. Kupfer at the Department of Psychiatry, University of Pittsburgh School of Medicine, Western Psychiatric Institute and Clinic, 3811 O'Hara St., Pittsburgh, Pa. 15261.

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grams (EOGs), and submental electromyograms (EMGs). Their records were evaluated according to the standard criteria (16) by a scorer who was unaware of any patient's clinical status or treatment. In addition to the EEG categorization of sleep stages, each minute of REM sleep was scored on a 9-point scale (0–8) for intensity of REM patterns reflecting both the amplitude and the number of conjugate eye movements (17). Sleep efficiency was measured by using a ratio of minutes of time spent asleep (TSA) to minutes of the total recording period (TRP).

The experimental design required that the 18 patients be studied during three periods: at least 3 consecutive nights before drug administration (baseline), the first 2 nights of drug treatment, and 4 consecutive nights during the third week of drug treatment. During the first 2 nights of drug administration. 7 patients each received orally a total of 150 mg and 11 patients each received a total of 200 mg of amitriptyline. During the third week of treatment the 18 patients were receiving daily therapeutic doses of the drug that ranged from 150 to 250 mg (mean 24-hour-dose, 191±4.7 mg). Amitriptyline was the only medication used in this study; all of the patients were receiving therapeutic doses (at least 150 mg) by the fourth day of treatment.

The sleep characteristics reported here are based on the mean EEG sleep values obtained during each of the three periods of time. A variety of REM measurements (REM time, REM latency, REM activity, cycle lengths) and non-REM measurements (sleep latency, intermittent awakening, early morning awakening, delta sleep, time spent asleep, and sleep efficiency) were examined. For each patient the baseline sleep measurements were compared with those for the first 2 nights of drug treatment and with those for the third week of drug treatment. Student's two-tailed t test for ungrouped data and Pearsonian correlations were the main statistical procedures used in this study.

During the third week of the study a clinical assessment was made that divided the group into patients who had successfully responded to amitriptyline administration and those who had either a poor or a weak response. The basis for this clinical judgment was the Kupfer-Detre System (KDS) clinical ratings for depression (excluding items relating to sleep disorders) (13–15). A change score of 3 or greater was considered the criterion for a positive clinical response. The clinical raters were unaware of the nature of the study being conducted. At the end of the 3-week study period, 7 of the 18 patients had responded unequivocally to amitriptyline. An analysis of the two groups (responders versus nonresponders) indicated no significant differences in terms of age (50.6 years versus 52.0 years), amount of drug received on the first 2 nights (183 mg versus 185 mg), or amount of drug received during the entire 3-week period (186 mg versus 195 mg). In addition, there were no differences in their baseline EEG sleep measurements (see table 1).

RESULTS

In order to analyze the different responses of the two groups of patients, we examined the differences in EEG sleep between the baseline period and the first 2 nights of drug administration (see table 2). On the basis of the measurements obtained on the first and second nights of drug administration, the good clinical response group consisted of patients who showed a marked decrease in REM sleep percent, a marked increase in REM latency (particularly on the first night of drug administration), and decreased REM activity (see table 3). No significant differences in EEG measurements that are usually associated with sleep continuity or any of the other stages of sleep were detected.

TABLE 1
EEG Sleep Measurements at Baseline

Item*	Good Responders (N=7) Mean	±SEM	Poor Responders (N=11) Mean	±SEM
Time spent asleep	317.70	18.90	287.80	21.40
Sleep latency	43.10	11.80	56.30	16.50
Early morning awakening	33.40	11.10	36.20	7.60
Wakefulness	51.60	12.00	42.40	8.70
TSA:TRP**	.71	.04	.69	.05
REM latency	37.10	6.40	45.80	6.90
REM sleep time	78.60	7.00	64.70	8.80
REM sleep percent	24.40	1.00	21.80	2.60
REM activity	166.00	19.60	126.30	21.70
REM activity:TSA	.52	.06	.45	.07
Delta sleep percent	4.30	1.10	2.70	1.10

* In minutes unless otherwise indicated.
** Ratio of time spent asleep (TSA) to total recording period (TRP).

TABLE 2
EEG Sleep Measurements for First 2 Nights of Amitriptyline Administration

Item*	Good Responders (N=7) Mean	±SEM	Poor Responders (N=11) Mean	±SEM
Time spent asleep	394.90	14.40	366.30	8.40
Sleep latency	22.70	4.40	39.00	7.10
Early morning awakening	14.80	3.60	18.90	6.10
Wakefulness	31.70	7.80	26.80	7.00
TSA:TRP**	.85	.02	.81	.02
REM latency	156.80	30.10	105.30	17.10
REM sleep time	46.10	6.80	52.50	9.40
REM sleep percent	11.60	1.70	13.90	2.30
REM activity	106.80	26.50	105.80	24.10
REM activity:TSA	.27	.06	.29	.07
Delta sleep percent	1.70	1.00	2.40	1.20

* In minutes unless otherwise indicated.
** Ratio of time spent asleep (TSA) to total recording period (TRP).

TABLE 3
Comparison of Baseline EEG Sleep Measurements with Those for First 2 Nights of Amitriptyline Administration

Item*	Change in Measurements: Good Responders (N=7) Mean±SEM		Poor Responders (N=11) Mean±SEM		Significance
REM sleep percent	−12.90	.90	−7.00	.90	p<.01
REM latency	+119.60	28.30	+52.80	14.00	p<.05
REM sleep time	−32.60	5.80	−12.30	4.20	p<.05
REM activity	−59.50	15.30	−20.40	10.60	p<.05
REM activity:TSA**	−.25	.05	−.16	.04	n.s.

* In minutes unless otherwise indicated.
** Ratio of REM activity to time spent asleep (TSA).

TABLE 4
EEG Sleep Data for Third Week of Amitriptyline Administration

Item*	Good Responders (N=7) Mean±SEM		Poor Responders (N=11) Mean±SEM	
Time spent asleep	387.30	12.70	332.30	15.70
Sleep latency	25.30	4.80	44.60	11.40
Early morning awakening	9.00	3.40	30.00	9.60
Wakefulness	20.20	8.60	26.60	5.80
TSA:TRP**	.88	.03	.77	.04
REM latency	127.50	15.20	110.20	19.20
REM sleep time	55.70	5.10	45.60	5.50
REM sleep percent	14.50	1.50	13.30	1.40
REM activity	121.50	34.50	115.00	16.90
REM activity:TSA	.35	.07	.33	.04
Delta sleep percent	2.40	.70	1.70	1.00

* In minutes unless otherwise indicated.
** Ratio of time spent asleep (TSA) to total recording period (TRP).

The changes in REM sleep percent correlated significantly with clinical response ($r=-.69$, $p<.01$), as did REM latency ($r=-.55$, $p<.05$) and REM activity ($r=-.55$, $p<.05$).

For the purposes of prediction, the mean sleep changes for both nights might be most applicable for deriving cutoff scores for new cases. For example, by using a mean cutoff level of 10% REM reduction during the 2 nights, 82% of the patients were placed in the proper groups. If a REM percent reduction of 10% or a REM latency change of greater than 100 minutes is used, the correct identification goes up to 88%.

With respect to the follow-up period (the third week of drug administration), both groups of patients showed marked changes in their EEG sleep in comparison with their baseline levels (see table 4). Although the good responders showed a significant increase in total sleep time (70 minutes, $p<.01$) and in sleep efficiency (17%, $p<.01$) during the follow-up period in comparison with their baseline levels, these patients also had an increase in REM latency of 90 minutes ($p<.01$), which was mirrored in a 10% decrease in REM sleep percent ($p<.01$). On the other hand, although the poor responders also showed a moderate increase in time spent asleep (44 minutes, $p<.05$), they showed a nonsignificant increase in sleep efficiency. The poor responders showed an increase in REM latency of 64 minutes ($p<.05$) as well as a 9% overall decrease in REM sleep percent ($p<.01$).

Regardless of clinical response, there were obvious drug-induced changes in the sleep of both groups. These EEG sleep changes reflected alterations in sleep continuity (more sleep efficiency, $p<.01$; reduced early morning awakening, $p<05$); in increased total recording period, $p<.01$; and in REM sleep (particularly REM latency, $p<.001$; decreased REM sleep percent, $p<.001$; and even a reduction in REM intensity, $p<.001$). At the time of the follow-up period, amitriptyline still exerted a pronounced effect on EEG sleep in both groups, especially in relation to time spent asleep ($p<.01$), prolonged REM latency ($p<.01$), and reduced REM percent ($p<.001$). Interestingly, although REM intensity was restored to normative levels in the good-response group, it was still significantly reduced in the poor-response group at the time of follow-up.

DISCUSSION

Our study and earlier investigations have established a significant and pronounced effect of amitriptyline on the sleep of psychiatric patients, but there have been no investigations that differentiated clinical response on the basis of the effects of the drug on sleep. Indeed, because sleep changes are very often symptoms of affective disorders and sleep changes have sometimes preceded clinical improvement, EEG sleep measurements might represent an objective indicator that would not only aid us in monitoring the course of depressive illness but in fact predict clinical response.

That amitriptyline exerts a significant drug effect on EEG sleep measurements, with respect to both sleep continuity and REM sleep, has been shown (18, 19). Amitriptyline seems to possess a sedative effect in that patients show increased time spent asleep along with less intermittent and early morning wakefulness. Similarly, amitriptyline seems to produce pronounced changes in REM measurements, e.g., increases in REM latency and decreases in REM percent, REM time, and even the intensity of REM sleep.

Despite the acknowledged sedative properties of the drug, the good responders in our study could not be differentiated from the poor responders on the basis of sedation (increased sleep time and increased sleep efficiency). These characteristics, which have been reported as an early drug effect, did not separate good from poor clinical responders until the 3-week follow-up period. This unexpected finding suggests that the

extent of sedation may not predict eventual clinical response, at least not when sedation is defined as an increase in sleep continuity. If sedation is defined in terms of the patient's perception of sedation (which includes a manifestation of a general sense of restlessness and may be reflective of REM sleep alterations), it may be that what the patient reports as a sedative effect represents a clinical lead on which to base a differentiation of good responders from poor responders.

This preliminary investigation would seem to indicate that the REM sleep changes in the good clinical responders compared with the poor responders were more intensive and pronounced than can be explained on the basis of drug effect. That is to say, although REM latency was increased significantly in both responders and nonresponders, it was significantly more increased in the good responders. Similarly, REM sleep percent reduction was significantly more pronounced in the good-response group as contrasted to the poor-response group. The significance of the REM suppression that occurs within the first 2 days of drug administration and its persistence over a period of weeks are still relatively unknown (18).

The immediate REM suppression after administration of tricyclic antidepressants is somewhat similar to the report that the administration of amphetamine, which can cause a marked REM suppression without any associated sedative effect, may produce at least a partial clinical remission after only 1 night (20). This investigation (20) also indicated that response to dextroamphetamine may be a possible predictor of improvement with tricyclic treatment of depression. Other investigators (21–23) have suggested this kind of differential prediction in relation to amphetamine, MHPG levels, and treatment outcome. The possible use of REM sleep changes as predictors of drug response also bears a strong relationship to recent studies suggesting that prolonged REM deprivation may be a treatment technique for endogenous depression (24).

The differential response of the good responders and the poor responders in terms of REM suppression was transient; several weeks later both treatment groups were experiencing a similar degree of REM suppression (9% to 10% REM sleep reduction, compared with baseline levels). On the basis of the REM deprivation idea, one would expect that both groups would have had a similar clinical response. However, our patients were not monitored continuously; the true extent of the cumulative REM deprivation as manifested in decreased REM sleep and prolonged REM latency is therefore not known. Such a study requires a longitudinal approach. One might speculate that at least 7 to 10 nights of a sustained elevation of REM latency of 200 minutes or greater and less than 10% REM sleep are necessary to achieve a clinical remission.

With regard to future strategies using this kind of approach, it may be that a more systematic evaluation can be devised if an intramuscular injection of amitriptyline or a test dose of 150 mg at bedtime is administered in a single night trial. This technique might be contraindicated in older patients unless a 100-mg dose is used. The monitoring of blood levels of amitriptyline would be extremely helpful to determine whether the initial blood levels achieved in the first 24–48 hours are predictive of clinical response. In addition, as we reported previously (25), the telemetric monitoring of motor activity may add a further measurement technique to this type of investigation.

The advantages of such psychobiological techniques are apparent. If EEG sleep changes or other psychobiological measurements could be used early in the course of treatment of depression to predict clinical response to tricyclic therapy, at least three major benefits to the patient would result: the incidence and duration of hospitalization would be reduced, his suffering could be diminished, and his work and social functioning could be preserved. In addition, the cost/benefit ratio of treating inpatients for endogenous depressive disorders with tricyclic therapy could be reduced tremendously if such a predictive tool could be applied successfully. This strategy may also prove to be of great relevance in the outpatient treatment of affective disorders. Because the majority of affective episodes are treated on an outpatient basis, this would be extremely useful.

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Cocaine, Kindling, and Psychosis

BY ROBERT M. POST, M.D., AND RICHARD T. KOPANDA, M.A.

The authors review the evidence indicating that repetitive administration of central nervous system stimulants and other compounds may be associated with progressively increasing effects on pathological behavior and seizures rather than tolerance. They suggest that the progressive effects of these compounds may be related to electrical kindling, a phenomenon in which repetitive subthreshold stimulation of the limbic system is eventually associated with major motor seizures. They review the studies supporting a pharmacological kindling mechanism and suggest a kindling model for psychological stimuli that combines neurophysiological, biochemical, and psychological perspectives.

IN AN EARLIER PAPER (1) it was suggested that the effects of cocaine and related stimulants in man may progress from the predominantly affective to the schizophreniform spectrum with increased duration of administration. Clinical work with depressed patients and addict volunteers (2, 3) indicated that although cocaine is capable of producing mood elevation in some patients, it may be associated with dysphoric effects when administered intravenously in high doses. These data, in combination with the clinical reports that chronic administration of cocaine or amphetamine can produce a psychosis resembling paranoid schizophrenia (4–10), suggest that an identifiable mechanism might be involved in the progression from the initial euphoric-dysphoric effects to the psychotogenetic.

The evolution of these clinical syndromes may be related to the finding that repetitive administration of the same dose of cocaine or amphetamine to animals produces increasing effects on some aspects of behavior. We will discuss the evidence and some possible mechanisms for these progressive changes (reverse tolerance), including a pharmacological kindling of the limbic system (11, 12). Kindling describes the phenomenon in which repetitive subthreshold electrical stimulation of the limbic system produces increasing effects on electrical activity and behavior, eventually resulting in major motor convulsions in reaction to a stimulation that previously produced no effect (13–16).

Kindling has been used as a model for neuronal plasticity and epileptogenesis, but it has not been directly related to pharmacological or behavioral phenomena. A kindling model for increased behavioral effects of repetitive electrical or pharmacological stimulation over time may provide an important explanatory concept for the progressive development of some psychopathological behavior in man. We postulate that repetitive psychological stresses may also be capable of activating critical limbic system mechanisms involved in the modulation of emotional and cognitive behavior in a progressive or kindling-like fashion.

REVERSE TOLERANCE EFFECTS

The studies supporting the concept that repetitive stimulant administration can be associated with progressive alterations in behavior are summarized in table 1. Tatum and Seevers (17) and Downs and Eddy (18, 19) reported that repetitive administration of cocaine to dogs and rats was associated with increasing excitability and susceptibility to cocaine-induced convulsions. Similar findings have been reported by other investigators in other animals (20–23). Likewise, repetitive administration of amphetamines, which are generally similar to cocaine pharmacologically and behaviorally (32–35), has been associated with increasing effects on activity (27, 28), stereotypy (25–29), and disruption of learned behavior (30).

The intriguing possibility that patients might react to stimulant administration with increased sensitivity over time has been set forth by Kramer (31), who suggested that amphetamine addicts may experience an almost immediate reactivation of paranoid ideation when they take amphetamine, even after a prolonged period of abstinence.

The review in table 1 is not inclusive; only studies demonstrating that chronic amphetamine or cocaine administration could be associated with reverse tolerance effects were selected; the many studies demonstrating no change in behavior or tolerance (32–36) were purposely omitted. The schedule of drug administration, the dose levels, and the parameter studied (32, 37) may be critical determinants of whether tolerance or reverse tolerance effects are manifest.

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Dr. Post is Chief, 3-West Clinical Research Unit, and Mr. Kopanda was Research Assistant, Section on Psychobiology, Adult Psychiatry Branch, National Institute of Mental Health, Bethesda, Md. Address reprint requests to Dr. Post at Bldg. 10, Rm. 3S239, National Institute of Mental Health, 9000 Rockville Pike, Bethesda, Md. 20014.

TABLE 1
Selected Studies Supporting Progressive Behavioral Effects of Repetitive Stimulant Administration

Study	Drug	Animal Tested	Effect
Tatum and Seevers (17)	Cocaine	Monkey, dog	Excitement, activity
Downs and Eddy (18)	Cocaine	Rat	Seizures, death
Downs and Eddy (19)	Cocaine	Dog	Excitement
Gutierrez-Noriega and Zapata-Ortiz (20)	Cocaine	Dog	Hyperthermia, seizures, death
Gutierrez-Noriega (21)	Cocaine	Dog	Catalepsy
Post (22); Stripling and Ellinwood (23); Ho (24)	Cocaine	Rat	Stereotypy
Post and Kopanda (11); Post and associates (25)	Cocaine	Monkey	Abnormal visual searching, catalepsy, seizures
Mago (26); Segal and Mandell (27)	Amphetamine	Rat	Stereotypy, hyperactivity
Klawans and Margolin (28)	Amphetamine	Guinea pig	Stereotypy, dyskinesias
Ellinwood and Kilbey (29)	Amphetamine	Monkey	Stereotypy, dyskinesias
Ranje and Ungerstedt (30)	Amphetamine	Rat	Disruption of learning
Kramer (31)*	Amphetamine	Man	Reactivation of paranoia

*This report provided data suggestive of an increased reactivity to amphetamine following previous abuse of the drug.

Recent data from our laboratory (25) suggested that repetitive daily administration of the same dose of cocaine to rhesus monkeys produces an increase in a variety of abnormal behaviors as well as an increased susceptibility to convulsions. After an initial phase during which most of the animals showed prominent hyperactive and stereotypic behavior in response to cocaine administration, they showed an increasing severity of motor inhibition, catalepsy, and abnormal visual behavior, including staring and apparent tracking of nonexistent objects about the laboratory. Moreover, animals that did not have convulsions following the initial injections developed increasingly frequent seizures following later injections. Cocaine-induced dyskinesias also developed with progressive severity in some animals treated for more than 10 weeks. The inhibitory syndrome and late dyskinesias appeared to be very similar to the reactions of the monkeys described by Ellinwood and associates (29, 38) following chronic, increasing doses of amphetamine.

Since both cocaine and amphetamine strongly potentiate catecholamines at the synapse (36, 39) by blocking the reuptake of catecholamines or increasing their release, it is tempting to relate the progressive effects of these drugs over time to their effects on neurotransmitter amines. In order to further clarify the role of cocaine's local anesthetic effects versus its amine-potentiating effects in producing the progressive behavioral syndromes over time, we investigated lidocaine, a compound with equal local anesthetic potency but no known amine potentiation at physiological doses (12). Like cocaine (40), lidocaine produces prominent spindle, seizure, and after-discharge activity in limbic system structures when administered in relatively high doses (41–43).

In a study reported in detail elsewhere (12), high doses of lidocaine (60 mg/kg) were administered intraperitoneally once daily to Sprague-Dawley rats. In contrast to the results we saw with cocaine (22), hyperactivity and stereotypy were not observed; rather, the animals engaged in a variety of abnormal eating behaviors (omniphagia and coprophagia). Although none of the control rats ate straw or feces, after 8 weeks of lidocaine administration more than 60% of the rats given the drug consistently ate these and other foreign objects.

Lidocaine-treated animals, like cocaine-treated animals (17–19, 22, 25), began to develop seizures in response to what was previously a subconvulsant dose (12). Following an average of 16 injections unassociated with convulsions, the animals developed generalized clonic seizures and then seized with increasing frequency and duration after subsequent lidocaine injections. Thus the progressive development of pathological eating behaviors as well as seizures with lidocaine suggest that effects other than those related to the stimulant effects of cocaine or amphetamine might be sufficient to produce some aspects of the reverse tolerance phenomenon.

MECHANISMS FOR PROGRESSIVE EFFECTS

Preliminary data suggest that the increased effects of chronic drug administration over time are not related to alterations in brain, plasma, or cerebrospinal fluid (CSF) levels of the stimulants or local anesthetics. Not only are cocaine and lidocaine metabolized very rapidly (36), but Mule (44) demonstrated that chronic administration of cocaine for 21 days in rats does not produce an increase in peak levels or half-life of cocaine in brain or plasma compared with an acute injection. Our preliminary data (22), obtained in collaboration with Richard Hawks, also suggest that the blood or CSF levels of cocaine in a monkey treated for more than 2 months are similar to those after the first injection.

Segal and Mandell (27) reviewed the evidence on blood levels of amphetamine and concluded that they are not likely altered with chronic administration. However, according to Kuhn and Schanberg (45), a focal brainstem area may have increased amphetamine

uptake. Ranje and Ungerstedt (30) demonstrated that amphetamine could interfere with learned maze behavior following injections as much as 12 days apart, and Mago (26) reported increased effects of amphetamine after a 6-week interval. Thus alterations in blood levels are an unlikely explanation for the progressive effects.

Conditioning phenomena, as described by Tilson and Rech (46), do not appear to adequately account for the progressive effects. Animals treated with cocaine did not demonstrate the typical stimulant-induced syndrome when given saline (22, 25, 27), and the de novo appearance of a variety of behaviors, such as stereotypies, dyskinesias, and convulsions, makes it unlikely that conditioning alone could account for the findings.

KINDLING

We were struck with the similarity in quality and time course of the effects of cocaine and lidocaine and the phenomenon of electrical kindling. The kindling effect was initially demonstrated by Goddard and associates (13) and has been extensively studied in various species by Racine (14), Wada and Sato (15), Pinel and Van Oot (16), and other investigators. Repetitive intermittent electrical stimulation of subcortical structures, particularly the amygdala, is associated with increasing duration and spread of after-discharges and local seizures, resulting in an animal's consistently having major motor convulsions in response to what was previously a subthreshold electrical stimulation. Although cortical tissue can be kindled with difficulty (13, 47), subcortical sites are more sensitive to the kindling procedure. The amygdala and related limbic areas are most sensitive, followed by the caudate and the hippocampus (13, 48). Wada and Sato (15) traced the development of the spread of seizure discharge following repetitive amygdala stimulation and indicated that it moves to the neostriatal system and then to the mesencephalic reticular formation before finally causing bilateral neocortical and generalized convulsive activity.

It should be emphasized that repetitive but intermittent stimulation is critical for the development of kindling and that chronic, nonintermittent stimulation will actually retard the kindling phenomenon (13, 49, 50).

Once an animal is kindled, the reduction in seizure threshold may last for more than 6 months, suggesting that a relatively permanent synaptic reorganization has occurred (13, 14, 49, 51). Most noteworthy for our purposes is the finding that the kindling phenomenon is not dependent on the manifestation of generalized seizures. The threshold for localized after-discharges unaccompanied by motor phenomena is also lowered following repetitive electrical stimulation (14, 51, 52). Thus seizures may serve as a marker for the kindling process but are not themselves a prerequisite.

A final note particularly relevant for later clinical interpretations of the kindling data is the finding that a chronically kindled animal may eventually exhibit spontaneous seizures (16); i.e., the kindling process may so facilitate neuronal pathways and seizure mechanisms that they discharge autonomously.

Pharmacological Kindling

Cocaine and lidocaine both have potent effects on limbic system structures, causing localized spindles, after-discharges, and seizures before they affect the cortical EEG (40–43, 53). Amphetamine alone or in combination with disulfiram also produces prominent alterations in limbic system activity (54, 55). We postulate that the stimulants and local anesthetics exert their progressive effects on behavior partly by means of a pharmacological kindling mechanism. That is, spindle and seizure activity in limbic system structures may increase after repeated doses, may be associated with increasing behavioral effects, and may become fully manifest in convulsions like those which result from electrical kindling.

In support of this concept, our data indicate that the progressive development of lidocaine-induced seizures is similar to the development of seizures evoked with electrical kindling. Not only do seizures develop and occur with an increased frequency in response to what was previously a nonconvulsant dose, but the seizures following lidocaine administration are qualitatively similar to those described with electrical kindling (13–16): rats rear back on their haunches and demonstrate intermittent clonic convulsions of head, trunk, and forepaws (12) in a posture characteristic of limbic seizures (13). Thus the time course, quality, and physiological focus of the effects of cocaine and lidocaine are all suggestive of a pharmacological kindling mechanism within the limbic system. Recent studies of the kindling hypothesis by Stripling and Ellinwood (23) and Ellinwood and associates (56, 57) indicated that repetitive cocaine administration produces progressive alterations in amplitude and synchronization of limbic spindles in the rat (23) and cat (57).

Pinel and Van Oot (16) emphasized the generality of the phenomenon of the progressive development of seizures after repetitive stimulation of the central nervous system. Table 2 presents a summary of studies of pharmacological, electrical, and audiogenic agents that have been associated with increased susceptibility to seizures. That the brain in some circumstances has the potential to increase facilitation or reactivity to repetitive, intermittent stimulation is supported by the variety of treatments capable of eliciting this effect.

The generality of a kindling-like phenomenon is also highlighted by the fact that repetitive administration of one treatment modality may sensitize the organism to another (16). For example, an electrically kindled animal is more susceptible to the effects of pentylenetetrazol (Metrazol) or the alcohol withdrawal syndrome; previous pentylenetetrazol and ECT treatments also exacerbate the alcohol withdrawal syn-

TABLE 2
Selected Studies of Agents Associated with Kindling-Like Increases in Susceptibility to Seizures

Study	Agent	Frequency of Administration	Animal Tested
Tatum and Seevers (17)	Cocaine	Once daily	Monkey
Downs and Eddy (18); Post (22)	Cocaine	Once daily	Rat
Post and Kopanda (11); Post and associates (25)	Cocaine	Twice daily	Monkey
Prichard and associates (58)	Flurothyl ether	Once daily	Rat, mouse, guinea pig
Leech (59)	Audiogenic stimulation	Once daily	Mouse
Mason and Cooper (60); Pinel and Van Oot (16)	Pentylenetetrazol	Once every 3 days'	Rat
Ramer and Pinel (61); Pinel and Van Oot (16)	ECT	Once every 3 days	Rat
Modigh (62)	ECT	Once daily	Mouse
Vosu and Wise (48)	Carbachol*	Once daily	Rat
Post and associates (12)	Lidocaine	Once daily	Rat

*Administered intracerebrally.

drome (16). In contrast to the generality of agents that are capable of progressively eliciting seizures, a specific and critical variable for the occurrence of progressive effects may be the frequency of their administration. Goddard and associates (13) found that there was an optimal interval (i.e., 24 hours) between stimulations for electrical kindling. Likewise, when many of the treatments listed in table 2 were given at intervals much more frequent than once a day they were not associated with increasing effects on seizures (16).

Kindling of seizures has been produced by direct intracerebral techniques, suggesting that cholinergic and dopaminergic pathways can be involved. For example, repeated, direct instillation of a previously nonconvulsive dose of carbachol (48), a drug that stimulates cholinergic receptors, produced seizures in rats. The amygdala was most sensitive to the drug, followed by the caudate and the hippocampus, which is the precise rank order of sensitivity of these anatomical areas to electrical kindling. Stevens and Livermore (63) reported electrical kindling of dopaminergic neurons in the area dorsal to the interpeduncular nucleus (A_{10}). These neurons, which project to the mesolimbic system, have been suggested as one locus of abnormal function in the schizophrenic syndromes (64, 65) as well as a site of action of the neuroleptics (66, 67).

Amine mechanisms, particularly alterations in receptor site sensitivity, are also implicated in some aspects of the reverse tolerance or kindling-like effects noted. Modigh (62) reported that repetitive ECT sensitizes mice to the activating effects of apomorphine and clonidine with reserpine pretreatment. Klawans and Margolin (28) demonstrated that chronic administration of amphetamine potentiates apomorphine-induced stereotypic behavior. Since apomorphine is a relatively specific dopamine receptor stimulator, these data suggest that alterations in dopamine receptor function, an "innervation supersensitivity" (28), may be involved in the increased effects of the stimulants on behavior over time. Rebec and Groves (68) confirmed these effects on the single neuron level, reporting increased suppression of neurons in the striatum and reticular formation with apomorphine following chronic, daily amphetamine pretreatment.

Increased receptor site sensitivity (28) on a depletion (69), denervation (70), or other (71) basis, although a possible biochemical mechanism for progressive behavioral and convulsive effects, would not be incompatible with a neurophysiological or kindling-like mechanism. Modification of receptor sites might mediate the synaptic facilitation (13) that appears integral to the kindling process. Moreover, Garcia and Mora (72) reported that repeated stimulation of the hippocampus produces histological changes consistent with an increase in RNA in synaptically related neurons.

Kindling Mechanisms and Behavior

We suggest that the amine potentiation produced by cocaine and amphetamine confers a unique behavioral syndrome on an underlying kindling-like or sensitization mechanism in the limbic system. Cocaine and amphetamine are thought to be potent stimulants largely because of their effects on catecholamines, which may be involved in effects on arousal, activity, stereotypy, insomnia, anorexia, and dependence (39, 67, 73–75). The amine effects in combination with a kindling-like mechanism may therefore help explain why the acute effects of the stimulants on mood progress to effects productive of a psychotic process. Acute cocaine administration is associated with prominent euphoria and dysphoria in man and hyperactivity and stereotypy in animals. However, more chronic administration of cocaine may be associated with paranoid psychosis in man and catatonic-like behavior and bizarre visual tracking and staring in animals (1, 21, 25). As in the electrical kindling paradigm, repetitive stimulant administration may also be associated with progressive increases in duration and spread of discharges associated with increasingly pathological behavior (23, 53, 57, 68). Accordingly, this model psychosis potentially implicates catecholamines, a limbic system focus, and a kindling-like or progressive mechanism.

Although lidocaine also has a limbic system focus for seizure activity (41–43) and produces progressive effects on behavior (12), its lack of effect on amine systems may explain why the behavioral changes produced with chronic lidocaine administration are qualitatively different from those produced by cocaine. The behavior seen during chronic cocaine or amphetamine administration in man and animals resembles that of the paranoid-catatonic spectrum, but the lidocaine-induced coprophagia and omniphagia (12) are markedly different.

Arieti (76) discussed the bizarre eating patterns in chronic "terminal stage" schizophrenic patients, who progress from a state in which they grab food at the sight of it to the habit of placing anything in their mouths, paying no attention to edibility. They may eat a variety of foreign objects, including "crumbs, cockroaches, stones, rags, paper, wood, clothes, pencils, leaves," and feces. Surgery has been required to remove intestinal obstructions, and a profusion of objects, including "spoons, stones, pieces of scrap iron, wood," have been found in the stomachs of patients on autopsy. It is noteworthy, in relationship to the chronic time course of kindling-like phenomena, that Arieti stated that this stage of schizophrenia occurs with chronicity, usually 7 to 40 years after the onset of the illness.

The almost automatic placing of objects in the mouth is reminiscent of the Kluver-Bucy syndrome produced by bitemporal hippocampalectomies in monkeys and man (77, 78); the tendency to put objects in the mouth in this syndrome is accompanied by an excessive reaction to every visual stimulus, visual agnosia, hypersexuality, and decreased aggression. Arieti (76) suggested that the behavior of some chronic schizophrenic patients, normal infants, and patients with bilateral temporal-limbic lesions may be related to a common primitive level of neuronal organization that is ordinarily inhibited or regulated by higher centers. The lidocaine-induced progressive development of omniphagia and seizures may represent a pharmacological means of producing this behavior by disrupting limbic-temporal areas rather than producing major effects on aminergic systems. When amine effects are added, as during chronic cocaine administration, the behavioral effects are qualitatively different, including anorexia and prominent alterations in activity, posture, muscle tone, and attention.

Psychological Kindling

What are some of the clinical implications of such a pharmacological kindling model for psychosis? MacLean (79) emphasized the importance of the limbic system in his description of the triune brain, suggesting that it is a critical intermediary between the phylogenetically older reptilian brain necessary for basic instinctual mechanisms and the newer neocortical overlay so prominently developed in the primate. Repetitive stimulation in a kindling-like fashion could produce a functional lesion or functional disconnection of the higher centers from the lower centers. Papez (80), Gellhorn (81), Heath (82), MacLean (79), Stevens and associates (64, 65), Gloor (83), Torrey and Peterson (84), and others have postulated that disruption of the critical limbic areas involved in emotion and cognition could be related to the onset or maintenance of pathological affective and schizophreniform states. What is new in our formulation is the suggestion of a mechanism that might mediate such disruption of limbic function, its progressive nature, and its possible interrelationship with known neurotransmitters.

One might speculate that the limbic system could also be progressively altered by spontaneous or endogenous activation of a kindling-like mechanism in man. In particular, it is possible that repetitive, stress-induced alterations in limbic system activity could produce progressive effects on behavior over time using mechanisms similar to those activated by cocaine or amphetamine. It is of interest that the effects of various experimental stresses on biogenic amines in animals and man can be similar to those produced by the psychomotor stimulants (85).

A rare insight into the possible relationship of limbic mechanisms and stress in man was provided by a study of Lesse and associates (86) that demonstrated that prominent limbic spindling could be associated with the mere recollection of previously stressful experiences. The idea of increased reactivity of the limbic system on the basis of previous experimental or psychological stimulation is consistent not only with electrical and pharmacological data but also with the known involvement of limbic mechanisms (particularly the hippocampus) in memory and learning processes (87, 88). The limbic system may be capable of memory-like or facilitatory processes for more complex, emotional cognitive behavioral patterns as well as seizures.

The Relationship of Kindling to Seizures

Although we are using a seizure model of kindling to explicate possible limbic and cortical mechanisms mediating some progressive behavioral effects of cocaine, seizures per se are not a prerequisite of the kindling model. High doses of cocaine and lidocaine were purposely used in several of the studies cited so that marked behavioral effects could be produced and seizures could then be used as precise end points for assessing increased response to repetitive administration. Studies using much lower (subconvulsant) doses of stimulants have also elicited profound effects in man (89) and increasing effects on the behavior of animals (22, 27, 28). Thus we are not suggesting that actual seizures, even in subcortical sites inaccessible to normal EEG techniques, are a necessary concomitant of psychopathological behavior. Abnormally facilitated pathways may have disruptive effects on behavior in the absence of seizures.

This idea is consistent with the kindling studies that demonstrated a decreased threshold to repetitive elec-

trical stimulation of the limbic system even in the absence of convulsions (13, 51, 52). Dissociation of the behavioral and convulsive effects of local anesthetics may also be achieved with diazepam, which blocks lidocaine convulsions but does not block omniphagia (according to our unpublished observations). Similarly, diazepam blocks kindled seizures (47, 90) but not the increased sensitivity to a given stimulus, as manifested by a decreasing threshold for after-discharge (90). Because temporal lobe, limbic, and local anesthetic seizures may be closely associated with behavioral effects and may share common pharmacological mechanisms that are opposite those for most classical (cortical) models (40, 53, 89–91), behavioral effects rather than classical seizures would be the expected outcome of a kindling-like mechanism in man.

CLINICAL AND THEORETICAL IMPLICATIONS

A kindling mechanism may provide a critical conceptual link between the occurrence of chronic temporal lobe epilepsy and the later onset of psychosis. With repeated temporal lobe seizures, a kindling process could occur in which the temporal lobe activity progressively expanded to include other neural substrates, possibly those deeper in the limbic system.[1] The incidence of psychotic behavior in temporal lobe epilepsy increases with either the duration of the seizure disorder (93) or the interval between seizures (94). These clinical measurements match both the repetitive and intermittent nature of the electrical stimulation required to produce kindling.

As mentioned above, the kindling model might provide an explanation for the sensitivity of a former amphetamine addict to the recurrence of delusions even after months of abstinence (31). It is noteworthy that Bell (95) reported that the only 2 patients of 16 given high-dose, intravenous amphetamine who did not develop a paranoid psychosis were later found not to have had a history of long-term abuse. Previous repetitive stimulant administration may have altered the patients' susceptibility to amphetamine psychosis.

The kindling model for the progressive development of psychopathological behavior could also help conceptualize mechanisms by which environmental events might sensitize patients to acute exacerbations of illness. Chapman (96) emphasized the paroxysmal onset of some psychotic behaviors in acute schizophrenic patients. Episodic emergence of psychopathology could be interpreted in terms of sudden reactivation of previously facilitated pathways triggered by adequately stressful stimuli. Similarly, activation of predominantly excitatory or inhibitory limbic pathways (78, 83, 97) might be considered in the acute onset of affective psychoses (98). Although some patients with affective illness appear to have identifiable precipitants to their episodes, others have episodes with such precise regularity (e.g., every 48 hours) that specific psychological determinants for each mood can virtually be ruled out. Like seizures that can begin to erupt spontaneously with very chronic electrical kindling (16), "spontaneous" affective episodes might also emerge following sufficient repetition and stress. As lithium has been shown to increase thresholds for after-discharges in the limbic system (99), its unexplained effects in both mania and depression might be explored in terms of a common effect on a kindling-like mechanism.

Finally, the kindling model fits the criterion for a "functional" alteration, which is required for models of affective and schizophreniform psychoses since no neuropathology has been demonstrated in these illnesses. Moreover, abnormal discharge of neural pathways, an "active lesion," may be capable of producing more profound deficits in behavior than are destructive lesions or ablations and may be less readily compensated for (100). One of the advantages of the cocaine-kindling model would be that it focuses on functional synaptic changes and plasticity in the central nervous system without requiring a specific anatomical or pharmacological lesion.

Thus we could postulate abnormal facilitatory or inhibitory pathways that involve a variety of neuroanatomical structures and transmitters corresponding to a variety of different types of psychopathological presentations in the acute psychoses. The neurophysiological-biochemical model does not require a given lesion to explain multiple behavioral syndromes; it invokes a pathological process involving activated pathways that could act as a series of lesions. The model highlights the capacity of the central nervous system to react to a variety of stimuli in a progressively pathological manner, potentially making use of substrates ordinarily involved in normal learning and memory. Exploration of a kindling-like sensitization process in relation to behavior may help close the methodological and conceptual gaps between neurobiological and psychological approaches in psychiatry.

[1]McIntyre and Goddard (92) demonstrated that repetitive cortical stimulation is capable of producing kindling-like effects in limbic structures, as well as vice versa.

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The "Intoxication State of Consciousness": A Model for Alcohol and Drug Abuse

BY MARC GALANTER, M.D.

The author describes a model of intoxicant use based on altered states of consciousness and reviews his own and others' research on marijuana to illustrate the utility of this model, which is derived from both introspective reports and observed data. The relationship of social behavior and cognitive functioning to the "intoxication state of consciousness" is discussed. This state of consciousness may have an adaptive value in engendering and stabilizing social cohesion. Possible treatment implications include cognitive labeling of cues that precipitate episodes of abuse, training for moderated drug use while patients are intoxicated, and providing abusers with altered consciousness through other means, such as meditation.

DIFFERENT PSYCHIATRIC traditions have generated clinically useful models of alcohol- and drug-seeking behavior. This paper, based on studies conducted at the National Institute of Mental Health on the social intoxicant marijuana (1–6), will integrate our data with those of other investigators in an attempt to support the utility of a model of intoxicant use related to altered states of consciousness. I will also discuss the relevance of the concept of an intoxication state of consciousness in dealing with drugs of abuse.

MODELS OF DRUG ABUSE

Psychoanalytic thinking has engendered a model of alcohol abuse predicated on the development of oral fixations and unmet dependency needs generated by oral frustrations in childhood (7). The necessity to deal constructively with these needs in order to sustain treatment has been acknowledged (8).

Interpersonal relationships are also thought to play a role in precipitating a pattern of intoxicant abuse. For example, Berne (9) applied his model of transactional analysis to characterize the "alcoholic life game," and other games typical in alcohol abuse have been described (10). Alcohol use has also been said to play a role in maintaining the homeostasis of family systems (11) and in stabilizing nonfamilial interpersonal networks (12).

Another approach, drawn from the *medical model*, is to designate intoxicant abuse as a disease. Jellinek elaborated on this model in *The Disease Concept of Alcoholism* (13). He outlined the stages in the course of the disease along with their respective symptoms in a way that parallels the evolution of other chronic illnesses. Also related to the medical model is Dole and Nyswander's hypothesis that since heroin addiction is a disease with physiological substrates, some addicts may become permanently dependent on opiate administration (14).

Another model is Wikler's application of a *pharmacological conditioning paradigm* to opiate addiction (15, 16). One treatment approach suggested by this model is based on extinction of the conditioned drug-craving response by administration of the drug under circumstances in which reinforcement is not possible, such as after administration of a narcotic antagonist (17). Extinction of a craving response in alcoholics has been attempted by administering apomorphine to precipitate vomiting and thus serve as a negative reinforcer as one engages in drinking behavior in a treatment setting (18).

ALTERED STATES OF CONSCIOUSNESS

Contemporary psychiatric research is generally conducted on the level of behavioral phenomenology and/or neurophysiology. Around the turn of the century, however, it was felt that psychological investigation should be introspective and that its subject matter should be the immediate conscious experience of the subject within himself (19). Introspection, the controlled observation of the contents of one's own consciousness, was deemed the principal tool for the scientific study of psychology. William James reasoned that "the distribution of consciousness shows it to be exactly such as we might expect in an organ added for the sake of steering a nervous system grown too complex to regulate itself" (20, p. 144).

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Dr. Galanter is Career Teacher in Alcoholism and Drug Abuse and Assistant Professor, Department of Psychiatry, Albert Einstein College of Medicine, Bronx, N.Y. 10461.

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This approach was useful in understanding alterations of consciousness seen in certain religious experiences, as reflected in James's *The Varieties of Religious Experience* (21), and highlights some of the difficulty in applying contemporary psychiatric nomenclature in dealing with the recent revival of fundamentalist religious sects (22).

Rapaport (23) described variables that may distinguish states of consciousness within the psychoanalytic frame of reference as follows: 1) the degree of reflective awareness, i.e., the capacity to be aware of thought content and become aware of this awareness, 2) the ability to exert effort or will, and 3) the formal characteristics of thought, such as primary or secondary process thinking. Ludwig defined an altered state of consciousness (ASC) as

> any mental state(s), induced by various physiological, psychological or pharmacological maneuvers or agents, which can be recognized subjectively by the individual himself (or by the objective observer of the individual) as representing a sufficient deviation in subjective experience or psychological functioning from certain general norms for that individual during alert, waking consciousness. (24, p. 235)

Tart (25, 26) further elaborated on what is in effect a typology of discrete states of consciousness. These states are defined on the basis of their "hardware" (biological and physiological givens) and "software" programs (engendered by learning and acculturation). A multidimensional space, with each dimension representing a hardware or software variable, may be used to map out various discrete states of consciousness.

As one defines such boundaries for particular states of consciousness, similarities may be noted among the various states. For example, in our marijuana studies we found increased alpha power in the EEG during intoxication (4, 5). Certain meditative states may be similarly characterized (27, 28). Indeed, intoxication with psychotomimetic drugs is similar to various meditative states along a variety of experiential dimensions (29).

Work by Sperry and associates (30), who studied commissurotomy patients, may suggest an approach to understanding the stabilization of ASCs. Galin (31) related the cerebral dissociation phenomena that Sperry and associates described to Freud's topographic model of the mind, wherein repressed mental contents function in a separate realm inaccessible to conscious recall. They liken certain aspects of right cerebral function to the cognition referred to as primary process, being predicated on nonverbal representation of imagery, nonlinear logic, and lack of involvement in the perception of time and sequence. One might similarly speculate that other neurophysiological or functional neuroanatomical features of the brain may serve as a basis for the differentiation of ASCs under certain circumstances.

A systems approach is useful in defining a state of consciousness as an open system influenced by a variety of factors that impinge on it and alter its nature and stability. Our findings (1, 2, 4, 6) and those of other investigators (32, 33) indicate that the context is of major importance in determining the nature of the marijuana-induced subjective state. For example, very few if any medical reports from nineteenth century Europe indicated that patients for whom marijuana was prescribed were "stoned" or that their significant interpersonal relations were materially affected (34).

Just as one drug may produce somewhat different states depending on context, the same state-dependent parameters may be engendered by different determinants. We found, for example, that average evoked response patterns in marijuana-intoxicated subjects bore certain resemblances to those of schizophrenics when the same protocol was experimentally applied to both groups (5). This appears to reflect an ability of marijuana-intoxicated subjects to "tune out" the outside world and establish a temporary autism. This autism was also reflected in our findings on intoxication in a social context (6): the principal cognitive factor differentiating the active drug state from the no-drug and placebo states was a sense of detachment from the ongoing interactions of the social group.

CHANGES IN EXTERNAL PERCEPTION

Relating neurophysiological findings to metapsychological constructs may be useful in integrating experimental data. Shevrin (35) studied average evoked responses using subliminal cueing and illustrated a relationship between this physiological indicator and unconscious thought processes involving attention and perception. As I have mentioned, our marijuana-intoxicated subjects showed a decrease in the magnitude of evoked responses to both frequent and infrequent stimuli. It seemed plausible to presume that during intoxication our subjects paid less attention to auditory stimuli that they were told to ignore than they did after smoking no drug or placebo marijuana. Apparently, after smoking marijuana these subjects were able to turn down the "volume" of environmental input.

A related phenomenon is observed in marijuana-induced changes in the contingent negative variation (CNV). The CNV is a slow cortical potential that can be recorded immediately before a stimulus that is anticipated by the subject (36). Kopell and associates (37) found that THC enhances the amplitude of the CNV and surmised that this illustrates an increase in the complex function of attention-arousal that is represented by the CNV. This change in attention may be due to the reduction in distracting external stimuli that has been reported by users of the drug (38).

Another example of enhanced function is found in the work of Hill and associates (39), who studied the critical flicker fusion during marijuana intoxication and found an increased sensitivity in distinguishing intermittent light flashes, as well as an increased sensitivity to painful and nonpainful stimulation. These findings

illustrate some of the enhancement of certain functional capacities by marijuana, which may be construed as beneficial by the smoker. Various subjective reports have indeed indicated that marijuana users find their perceptual awareness heightened when they are intoxicated (38, 40, 41).

THE SUBJECTIVE EXPERIENCE OF INTOXICATION

Because of their predictive value in studying physical phenomena, inductive, reductionistic scientific techniques have popularized the assumption that reality is reducible to objectively defined physical components. Paradoxically, if one is studying subjectively experienced states of consciousness, subjective experience must be a primary source of data. Such data are often regarded as obstructive or irrelevant to accurate quantitative measurement.

We found, however, certain remarkable consistencies in the measurement of a smoker's subjective experience during marijuana intoxication (1). It appeared that this internal state was as reliable an indicator as physiological measures such as pulse change and blood concentration of labeled cannabinol. For example, subjects' totals for individual symptoms on a symptom checklist administered during intoxication correlated remarkably highly with their scaled global rating for the subjective variable of how "high" they were ($r=.94$, $p<.01$, Pearson product-moment correlation). In addition, there was a high rank-order correlation between symptoms in the active drug and placebo states ($r=.544$, $p<.01$). Apparently, the subjective experience of the marijuana state can be summoned up with remarkable accuracy by the cue of smoking placebo cigarettes.

What was puzzling was our finding that the only subjective experiences reported consistently after smoking marijuana in an experimental sensitivity group setting were somesthetic (e.g., dry throat and mouth, hunger, dreaminess, slower movement, and head feeling heavy) rather than affective (6). Apparently, individual subjects responded relatively consistently to marijuana, as indicated by self-report, but the subject-specific affective responses "washed out" when the data were analyzed for all subjects as a group. This is compatible with the assumption that ASCs may best be studied by having the individual characterize his own introspectively described experience rather than attempting to generalize across subjects. This approach would hark back to the introspectionist school of psychology.

STATE-DEPENDENCY

State-dependent learning was first observed in experimental animals exposed to learning paradigms after the administration of curariform drugs (42). These animals were better able to reproduce the learned material when the drug was readministered than when they were in the drug-free state. In recent years this phenomenon has been reproduced in human subjects intoxicated with alcohol (43–45). It has also been demonstrated with marijuana (46–49), both for recall of specific items and for reconstruction of rules that the subject himself had generated to learn experimental material.

It may be that certain aspects of state dependency contribute to the inability of drug abusers to carry over volitional capacities from sobriety to the drug state. The differential organization of cognitive traits during drug intoxication may result in ASCs in which basic learned and unlearned traits related to personality and adaptation are evident only in the particular ASC itself.

ETHOLOGY

One approach to understanding the role of an intoxication state of consciousness is derived from ethology, the comparative study of animal behavior. In the past 25 years ethologists such as Lorenz (50) and Tinbergen (51) have made major contributions to behavioral biology. Bowlby (52), discussing the relevance of ethology to the study of complex human behaviors, pointed to nest building and courtship behavior among birds to illustrate the complexity of behaviors whose substrates may be genetically transmitted. He also described the work of Dennis (53) and Spitz and Wolf (54) on the smile response in infants, a social behavior that may be physiologically based and organized in such a way that it may be evoked by appropriate "social" stimuli at around 6 weeks of age.

Ethologists describe similar behaviors present in different species as analogous when they have been developed by the different species in order to adapt to similar external circumstances (50). Animal behaviors similar to those of humans have been studied in order to shed light on human behavior (55–57). The roles played by ASCs as part of ritualized group phenomena have analogous counterparts in almost all cultures. Such states may be precipitated by social intoxicants or by interpersonal cues. In the context of an ethological approach, one might presume that ASCs have now or have had in the past an adaptive value, thus assuring their ubiquitous presence (58).

Displacement activity may occur when discharge of a surplus of instinctually derived motivation through normal channels is prevented. This most commonly happens when two antagonistic drives are operating or when the expression of an instinctual need is blocked by external factors. The displacement activity then becomes a strongly motivated behavior typical of another drive, with no adaptive value in the context where it appears. Weigert (59) likened this phenomenon to the development of neurotic symptoms. Under certain circumstances, one might consider the expenditure of excessive amounts of time in a drug-induced ASC as a

displacement activity generated by obstruction of the discharge of instinctual drives. An illustration would be the use of intoxicants for anxiety that results from conflicting drives.

It might be added that the ethological approach to explaining certain behaviors need not contradict the application of learning theory. For example, a responsivity necessary to initiate and terminate biologically based behaviors may be acquired by conditioning (52). This fact can be related to the constructs developed by Wikler (16) and Ludwig and associates (60) regarding the conditioning of interoceptive and exteroceptive cues to initiate the addictive use of opiates or alcohol or to precipitate relapse.

INTOXICANTS AND SOCIAL BEHAVIOR

In our study on marijuana and social behavior (6), subjects assessed their experience in sensitivity group meetings conducted after they had smoked either active marijuana, placebo, or no drug. Before the meetings these subjects had characterized their previous experiences of the social use of marijuana as happier, friendlier, calmer, and less defensive than their experiences in social situations while not "high." Meetings that were held in the active drug state were assessed by subjects as more relaxed and happier. This illustrates one aspect of the role that intoxicants may play in facilitating more stable and cohesive group interactions. Transcultural studies (61) support the adaptive value of ASCs in terms of the development of social cohesion and dealing with (or "curing," as the case may be) behavioral deviancy.

The capacity of intoxicants to stabilize a social system is illustrated by phenomena such as the diminution of aggressive and pathologically regressive behavior among alcoholics experimentally given the opportunity to share in the function of alcohol acquisition (62), the drinking gang in naturalistic settings (12), and the alteration by alcohol of roles within the family system serving to maintain homeostasis (11). Similarly, the widespread use of marijuana among farm laborers in Jamaica has been suggested to serve the adaptive function of increasing social cohesion in the group labor situation (63).

RELEVANCE OF THE MODEL

If there is a biologically based adaptive proclivity for altered consciousness, such as that produced by social intoxicants, we may be able to observe analogous behavior in other species. Crowley and associates (64) pointed out that other primates may be appropriate subjects for the study of pharmacological effects of drugs of abuse on social behavior and reported on initial studies in this area.

The adaptive value of social intoxicants may be underestimated by physicians in an attempt to label the alcohol abuser as victim of a disease. The altered state the abuser achieves may be valuable in assisting him to sustain an equilibrium within his particular social context. It thus becomes of central importance to ascertain what adaptive traits the intoxication state provides a given patient and to then assist him to develop the capacity to achieve such ends without recourse to the intoxicants (65).

The discrete quality of the intoxicated state, particularly in relation to state-dependent learning, may enable us to better understand how cognitive and volitional capacities that exist during sobriety may be unavailable during intoxication. Many of our endeavors in combating intoxicant abuse are dependent on the capacity of the abuser to carry over learning from sobriety to intoxication. Perhaps some of the training should be conducted while the subject is intoxicated. Social drinkers have been trained to gauge their own blood alcohol concentrations with feedback from breath analyzers (66, 67). Similar approaches are now being used with alcoholic patients to provide them with a cognitive framework to apply during the intoxication state (68).

In order for the intoxicated subject to develop a more effective capacity to sustain his volition in the altered state, cognitive labeling (17) of the interoceptive or exteroceptive cues that precipitate an episode of abuse should be maximized. This might enable the abuser to prevent loss of control after he passes into the intoxicated state. The potential viability of such an approach is suggested by the experimental use of verbal cues to reverse state-dependent learning deficits during marijuana intoxication (69).

If it is the altered state of consciousness that attracts the abuser, then the option of achieving such altered states by other means should be considered as potential treatment. One such approach is the use of meditative states. Many practitioners of meditation were involved in the wave of adolescent drug use in the late sixties. Benson (70, 71) reported preliminary work indicating decreased intake of alcohol and other drugs of abuse among initiates to Transcendental Meditation.

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Prompt Publication Policy

The *Journal* would like to remind authors who are particularly interested in early publication (whether to establish priority of an idea, reveal innovative results of new research, or for other pressing reasons) of its prompt publication policy. Under this policy, short (maximum of six double-spaced pages) manuscripts will be considered for publication in the Brief Communications section of the first available issue. These manuscripts must still undergo expert scrutiny before being accepted, and two months must be allowed for the printing process. However, the time required for publication of these short articles can be lessened considerably.

Authors who would like to have their manuscripts considered under this policy should indicate this desire, as well as the reason for it, when they submit their manuscripts.

The Future of Alcohology: Craft or Science?

BY MELVYN KALB, PH.D., AND MORTON S. PROPPER, M.S.W.

The authors discuss the problems that have resulted from the influx of professionals into the field of alcoholism, which has historically been dominated by paraprofessional workers. The interaction of the professional alcohologist, who operates from a scientific model, with the paraprofessional worker, whose model of treatment has followed craft lines, has created unique problems and tensions. A synergism has been created which has been detrimental rather than complementary. The authors suggest that the future of the alcoholism field will have to be established along craft (paraprofessional) or scientific (professional) lines because the two models cannot profitably co-exist.

A NEW TYPE of manpower, the professional alcohologist, has emerged on the alcoholism treatment scene as an outgrowth of the recent rich infusion of federal attention and funding through the establishment of the National Institute of Alcohol Abuse and Alcoholism (NIAAA).

While manifestly operating in harmony with the veteran nonprofessional, this influx of new professionals in the alcohol field has created unique problems and tensions. Unfortunately, the conflicts and frictions between the professional and paraprofessional have not been allowed to surface. Rather than attempting to resolve the problem, the approach has been to deny its existence.

The purpose of this paper is to illustrate and review the evidence of these conflicts, to trace their history and consequences, and to provide the reader with some explanatory hypotheses. Our intent is not only to provide a perspective on the problem but to stimulate dialogue in the belief that only with candid self-examination is there hope that the clinical alcoholism enterprise can meet the high expectations of the "new era" in alcoholism study and treatment.

When this work was done, Dr. Kalb was Coordinator of Research and Education, Santa Clara County Alcoholism Program, San Jose, Calif., where Mr. Propper is Clinical Social Worker. Dr. Kalb is now Clinical Psychologist, Department of Psychiatry, Kaiser-Permanente Medical Group, 900 Kiely Blvd., Santa Clara, Calif. 95051.

The opinions expressed herein are those of the authors and do not necessarily reflect the views of the Santa Clara County Alcoholism Program, the Santa Clara County Public Health Department, or the Kaiser-Permanente Medical Group.

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"CRAFT" VERSUS "SCIENTIFIC" MODELS OF ORGANIZATION

The context in which the conflicts between the professional and nonprofessional in the alcoholism field can best be understood is the distinction between craft and scientific models of organization. A craft as opposed to a scientific organization can be defined by differences in acquisition of knowledge, implementation of knowledge, and the nature of loyalty among its members.

A craftsman gains his qualifying skill (knowledge) primarily through direct observation and experience under the tutelage of a master craftsman. The knowledge he acquires is a product of the experiences of his teacher. The test of the craftsman's learning is his demonstrated ability to consistently replicate the performance of his teacher. Acceptable deviations are limited largely to elaborations of style; critical analyses of the overall traditions of the craft are actively discouraged. In fact, mutual agreement is the basis on which loyalty is established among the membership of a craftlike organization.

By contrast, the scientist-professional learns his skill not only experientially in a journeyman-apprentice relationship but also in a context relying on the cognitive teachings of many others. The scientist-professional in training is exposed to a broad variety of competing viewpoints and is encouraged to exercise autonomy in establishing his own conceptualization of the issues (1). Although there are craftlike aspects within the scientist-professional model (2), the demonstrated ability to engage in unique, independent thinking and to critically evaluate the work of one's teachers and peers is the sine qua non of the scientist-professional model.

It is this critical analysis and quest for new solutions using a body of mutually held methods (empiricism, rationality, and freedom of investigation) that establishes a bond of loyalty among scientists. The members of a scientific profession are not united by presenting a unified front against their critics but rather by a methodology that provides the tools for resolving disagreement.[1]

Paraprofessional preparation for service in the field

[1]Alfred North Whitehead (3) made the following distinction between a craft and a profession: "A craft is based upon customary activities and modified by the trial and error of individual practice. A profession [consists of activities] which are subject to theoretical analysis and are modified by theoretical conclusions derived from that analysis" (pp. 73–74).

of alcoholism has clearly followed the craft model outlined above. The indoctrination of new members in the philosophy and principles ("12 steps") of Alcoholics Anonymous by teachers who are recovered alcoholics (and who followed the same course of learning and rehabilitation), the prescription to think and act as their teachers do, and the goal of one day being like their teachers and teaching others the same philosophy characterize the apprenticeship of the paraprofessional.[2] The strong devotion of most paraprofessionals in the field of alcoholism to traditional concepts, their general resistance to serious consideration of alternative views and studies on the nature, course, and treatment of alcoholism, and their steadfast refusal to question their own premises in the light of conflicting evidence all serve to create an intense loyalty and unity.

As long as the alcoholism treatment enterprise was composed of an overwhelming preponderance of paraprofessionals, this craftlike organizational structure was appropriate and beneficial. It was only when professionals with their own scientific organizational model came on the scene that the perpetuation of the craft model became questionable.

It is our thesis that the future of alcohology will have to be established along either craft lines, exemplified by the paraprofessional alcoholism counselor, or scientific lines, embodied by the professional scientist. Because the defining properties and operational principles required for membership in a craft are different from those of a science, a détente would be difficult to achieve. The point is *not* that either approach is superior, but that the marriage of the two prevents growth and progress. The synergism that is created acts in a negative rather than a complementary way.

HISTORY OF THE PROBLEM

The clinical treatment of alcoholism has been governed largely by lay groups of recovered alcoholics. This group of dedicated individuals, acting in response to the benign neglect and abdication of the helping professions, banded together to fill the vacuum in the treatment of alcoholics. The predominant group has been the fellowship of Alcoholics Anonymous. Prior to the implementation of NIAAA in 1971, professionals in alcoholism treatment represented a scant fraction of the manpower in the field (5, 6).

While the number of professionals in the alcoholism field has increased dramatically, professionals are still a small percentage of the total manpower pool (5). This disproportionate distribution of professionals and paraprofessionals has continued to place the power, both political and clinical, in the hands of the nonprofessional.[3]

This distribution of power between professionals and paraprofessionals in the treatment of alcoholism is in marked contrast to that in the mental health field. Professionals have entered the field of alcoholism treatment after more than two decades of paraprofessional dominance, while the trend in the mental health field in the last decade has been in the opposite direction—the recruitment of paraprofessionals to join the ranks of the professional (7, 8). Moreover, while the paraprofessional in the mental health field has functioned adjunctly to and under the supervision of the professional, the paraprofessional in the alcoholism field has been a teacher or colleague rather than a student.

By virtue of this power, lay groups in the alcoholism treatment field have been and continue to be the gatekeepers of clinical knowledge and clinic operating style. Thus, despite a growing research literature that has pointed to new directions in the conceptualization of etiologic and therapeutic models, empirically unvalidated clinical notions regarding the nature of alcoholism persist. These clinical notions have firmly established themselves as traditional and inviolate wisdoms. Their survival is ensured by the protection provided by their disciples in the form of intense loyalty to them. Examples of this loyalty and the sometimes fierce attacks visited on the "heretic" are numerous (9–13).

Jerome Ravetz, in a recent book on the philsophy of science and its social problems, discussed the general problems of the relationship between the growth of an empirically based scientific establishment and the attendant conflict with the preexisting body of beliefs and doctrine (which Ravetz labels "folk science").

> Value [of the folk science] is determined by the degree to which a problem situation is central to the experience of the audience; and adequacy by the success in offering reassurance and the promise of understanding. . . . The conflicts between academic science and strongly based folk sciences provide many insights into the social situation of science, and into some insoluble problems inherent in that situation. [One such problem] occurs when the results of a disciplined scientific inquiry contradict the beliefs of a folk science, usually a popular one which is also adopted by the established cultural organs of society. (2, pp. 388–389)

As is the case in all craft organizations, this loyalty to traditional concepts is the "glue" that binds the nonprofessionals in alcoholism treatment (14). Toch (15) pointed out that all self-help groups have by definition an inherent antiprofessional bias. Pattison noted that a self-help group also has

> a self-fulfilling prophecy of success, and the validation of its theories is ideological rather than scientific. However, these elements of a self-help group are the "social glue" that holds a self-help group together, promotes co-

[2]It is interesting that the book *Alcoholics Anonymous* (4) is commonly referred to as "the Bible." The implications of this nickname are obvious.

[3]It is obvious that if nonprofessionals suddenly stopped working with alcoholics, alcohol treatment programs would be brought to a halt. This would not be the case were the professionals to pursue a similar course.

> hesion and integration, validates hope and commitment and in short makes it work! (14, p.620)

One of the most serious (and costly) by-products of this ideology is the lack of integration of research findings into the mainstream of the clinical treatment of alcoholism. Research in the treatment of alcoholism and the actual clinic treatment of the alcoholic have been allowed to coexist, but rarely (if ever) to intersect.[4]

Research findings (especially those not in keeping with the "party line") seem to have been simply ignored by the paraprofessionals. They have died not because of contradictory research studies or polemic discussions, but simply by being disregarded. In fact, vigorous and open scientific debate rarely occurs within the alcohol treatment setting. What does occur is either personal invective directed against the speaker or writer who presents "heretical" findings (the *data* being ignored) or systematic attempts to smother debate, with the rationale that "We are all in the field to help the alcoholic so let's not argue."

Filstead and Rossi (16) have pointed out that a publication in the 1940s, "Alcohol, Science and Society" (17), dealt with issues and problems that predominated in the era of the forties and still persist in this decade. They asked why there had not been more progress.

Some answers to this question might be found by questioning directors of alcohol treatment centers as to the amount of change in the last 10 years in the functioning style of their clinic and/or the treatment offered to the alcoholic that originated not from political and community pressures but directly from published scientific findings. It might be more frightening and revealing to ask how different the psychotherapeutic clinic treatment of the alcoholic would be had there been *no* scientific research on the treatment of alcoholism published in the last 25 years. Ludwig, discussing the assets and liabilities of the mental health system's approach to the alcoholic, noted that

> there are not enough searching and probing questions or effort expended to accumulate more facts. . . . In fact the current service oriented professional mentality is such that research is not only discouraged in many treatment settings but also regarded somehow as suspicious and even immoral. (18, p.152)

An example of the blatant disregard of scientific findings in the alcoholism treatment field is the belief that one drink triggers an uncontrollable urge in an alcoholic to keep drinking. A recent article by Paredes and associates (19) reported that this hypothesis as it is commonly understood is not supported by empirical evidence. The authors underscored the results of their own research by citing similar findings in 20 previously published studies. Mendelson (20), the first chief of the National Center for the Prevention and Control of Alcoholism, reported that his own experimental work showed no evidence for such an automatic biochemical triggering effect. Despite the overwhelming weight of empirical evidence against this hypothesis and the absence of any substantiating scientific evidence, it continues to flourish in the ideology of alcoholism treatment and philosophy as if it had been confirmed time and time again.

The work of Cahalan (21) serves as a variation on the same theme. Cahalan found that many men with serious drinking problems are able to change their drinking habits and "mature out of" problem drinking without therapeutic intervention. He also found that the largest proportion of serious alcohol-related problems are in men in their early twenties, who have apparently high rates of maturing out (spontaneous remission) in their early thirties.

Cahalan's findings raise serious questions about the validity of the "progressive illness" concept of alcoholism as it is commonly understood. Pattison (22) pointed out almost 10 years ago that the well-known Jellinek chart illustrating the progressive and disastrous decline of physical and emotional health that accompanies alcoholism was based on a biased sample of Alcoholics Anonymous members who were physically and socially bankrupt. Ironically, in his original article, Jellinek (23) pointed out that many people remain "stuck" at a given level of alcoholism behavior. This point, generally unknown or ignored, is not reflected in the famous "progressive illness" chart.

It seems that scientific research in alcoholism occupies a unique position. Research is not used, as in other fields, as a springboard for developing and testing new hypotheses and abandoning old ones that are empirically disproved. Alcoholism treatment concepts in the overwhelming majority of clinic settings have developed and survive not with the aid of research but in spite of it. Clinical treatment is not the logical outgrowth of scientific discoveries but instead remains an encapsulated body of theories and shopworn slogans that are apparently immune to the outcome of scientific research.

The unencumbered flow between research and clinic treatment that Chafetz (24) has called for can occur only in an environment in which the practicing clinician is free from a personal investment in the perpetuation of a particular theory or construct. It is only in such an environment that clinicians can choose therapeutic concepts on the merits of scientific evidence. It is paradoxical that personal investment and the lack of openness to new findings and fresh conceptualizations are the hallmarks of the typical alcoholism treatment setting.

Ludwig, writing about the role of innovation and the use of research findings in the alcoholism treatment system, noted,

[4]This isolation of research from clinic realities has now been institutionalized into the annual NIAAA National Conference. Three separate "tracks" have been established—research, treatment, and prevention—all of which present papers and discussions concurrently. Interactions among individuals in these three areas are made impossible by this design.

> Despite the embarrassingly poor long-term results of most treatment and rehabilitation programs, enormous sums of money and staff time are being spent to perpetuate traditional approaches (and various mythologies) rather than being diverted to innovative demonstration projects. It is as though the underlying philosophy of these programs is based on the principle that if sufficient manpower can be deployed to employ ineffective procedures then this multiplication of ineptness will hit therapeutic paydirt. (18, p.152)

A clinical field that claims to employ scientific principles and theories in the service of helping the alcoholic while creating a climate in which research findings are ignored and disregarded aligns itself to anachronisms, sloganeering, and opinion rather than to science. Ullman and Krasner posed the question that might eventually be put to the alcoholism treatment system: "If a particular theory does not utilize data based on scientific procedures, it may well be asked whether it is a reasonable basis for the treatment of human beings" (25, p. 36).

COGNITIVE STYLES AND COMMITMENTS OF PROFESSIONALS AND PARAPROFESSIONALS

There is an obvious divergence of learned cognitive styles between professional and paraprofessional workers. Most professionals have been trained to use an analytical-objective-inductive cognitive style. Their long years of schooling have not only emphasized a respect for inductive empirical reasoning but also equipped them with the skills to interpret and critically evaluate empirical research data. The paraprofessional usually lacks this training and uses an intuitive-subjective-deductive cognitive style. Empirical data are often perceived as obscure and irrelevant, especially when they conflict with intuitive, experiential understanding.

This contrast in cognitive style is reflected in the early literature on alcoholism treatment. Much of the early research was conducted without the experimental controls vital to sound experimentation, and the literature consisted mostly of anecdotal reporting (26). The popular view of alcoholism was created largely by laymen (5) and fashioned out of their personal experience.

The intensely personal commitment the recovered-alcoholic paraprofessional may feel to the traditional model of alcoholism is quite understandable. The traditional model defines the alcoholic as the victim of a disease, entitling him to many social services for the disabled. It also saves the recovered alcoholic from the moralistic stigmata of "weak character" and "mental illness" (27). Thus, the recovered-alcoholic paraprofessional's self-esteem is often a very real part of his "stake" in sustaining the traditional views of alcoholism. The individual who, for example, has the deep conviction that his sobriety rests solely on his acceptance of the tenets of Alcoholics Anonymous or on some other traditional understanding of alcoholism is likely to feel deeply and personally threatened by any information that might call those understandings and interpretations into question.

For the professional, a challenge to traditional beliefs is often an academic issue for debate, but for the recovered alcoholic, it often becomes a threat to his sobriety, his stability, his job, and his very existence. It is quite understandable that a recovered alcoholic whose sobriety is founded on abstinence may, for example, be deeply disturbed by the idea that some alcoholics can recover and drink socially. Selzer (10) maintains,

> Perhaps another important reason for the almost reflex rejection [of normal drinking in recovered alcoholics] is that many people working in this field are alcoholics themselves and are compelled to remain abstinent. It may be especially difficult for the alcoholic who must remain dry to accept the idea that others can recover and drink socially. To hear of the "success" of others may be frustrating—and those workers prefer not to hear about it since it also upsets their treatment concepts. . . . Granted that experimentation will yield discouraging relapses in most instances, this is not sufficient excuse for prejudiced persons to vilify the truth. (p.133)

IMPLICATIONS OF THE INFLUX OF PROFESSIONALS

The professional is rather in the position of a newcomer or, in some cases, a tentatively welcome visitor in the "house" of alcoholism treatment. He may very well feel that he must, like a visitor, be on his best behavior and be sensitive to the house rules. His "gifts" and talents (clinical skills, inductive and analytic reasoning) are viewed with suspicion and/or contempt. He must tread carefully because, on a practical day-to-day level, he is the most dispensable member of the treatment team. Under these circumstances, the professional has three options.

1. He can reduce his cognitive dissonance by subscribing to traditional views. If he does this, he will eventually be accepted and find his circumstances increasingly comfortable.
2. He can attempt to use his professional training and perspective. His success will often be a function of his charisma, diplomacy, and the nature of the clinic. Often, he will find himself stymied by the passive—and sometimes active—resistance of the paraprofessional workers and those of his colleagues who have opted for tactic number 1.
3. He can flee to more comfortable and less divisive and frustrating clinical pastures (e.g., mental health). Those who choose to stay are likely to be the best and the worst (28).

PROBLEMS AND PARADOXES

Unfortunately, problems rooted in the very origin and structure of an institution are not likely to yield to

simple or painless solutions. The problems and conflicts we have discussed are deeply rooted in the history, dogma, and constituency of the alcoholism treatment enterprise. Each conceivable pragmatic revisionist strategy carries with it a paradox.

If through some administrative fiat empirical research findings were suddenly incorporated into clinical practice, a panoply of problems would result. First, it would be apparent that the vast majority of treatment personnel lack the training to apply innovative treatment methods. The paraprofessional's area of expertise would become obsolete: his credentials in the field (i.e., his personal experience) would no longer be marketable (29). The sudden shifts in power that would be produced by the application of treatment procedures and philosophies unfamiliar to the paraprofessional but within the area of expertise claimed by the professional could present an even greater problem.

We envision yet another Pandora's box of complications if the clinical field were somehow made more tolerant of healthy controversy, more responsive to empirical findings, and more receptive to new methodologies.

It is not unlikely that a training and orientation program that would inevitably tend to "professionalize" the paraprofessional would produce massive resistance, conflict, and upheaval. The process of professionalization, of increasing the paraprofessional's clinical sophistication and versatility, would produce a crisis of commitment by threatening the underlying antiprofessionalism that binds these workers to the organizational structure and validates their beliefs. Pattison warns that "A self-help group should not be professionalized or scientificized, because that would undercut the very socio-psychological matrix that makes a self-help group effective" (14, p.620).

The alcoholism treatment enterprise now finds itself in a Procrustean dilemma—to eschew the empirical, scientific orientation for the sake of intramural tranquility and therefore abandon alcohology as a scientifically oriented clinical enterprise, or to adopt the classical professional-scientific model and risk the alienation of a large segment of current treatment personnel. Ignoring this dilemma, as was done in the past, is no longer an option and would only cause further alienation and dissension within the field and even more confusion in the public about the nature of alcoholism. The alcoholism treatment field can no longer masquerade its craftlike organization under the banner of science.

Until this decision is made, no amount of funding, training, recruitment, or bureaucratic reorganization will materially alter the impact of the alcoholism treatment system. As Ludwig notes, the promise of the alcohol system "will not be realized by having more money, more staff, and more facilities but rather by qualitative changes in the nature of the activities conducted, a willingness to admit to relative scientific ignorance and a concerted effort to remedy these deficits" (18, p.156).

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Clinical Response and Plasma Levels: Effect of Dose, Dosage Schedules, and Drug Interactions on Plasma Chlorpromazine Levels

BY LEONOR RIVERA-CALIMLIM, M.D., HENRY NASRALLAH, M.D., JOHN STRAUSS, M.D., AND LOUIS LASAGNA, M.D.

Plasma chlorpromazine (CPZ) levels of 50 psychotic inpatients were measured by gas liquid chromatography; the clinical progress of 29 of these patients with acute psychoses was also assessed. CPZ levels of 50–300 ng/ml were usually associated with clinical improvement; there was also a relationship between CPZ levels and increases in certain symptoms. The 50–300 ng/ml level was best attained by doses of 400–800 mg/day. Trihexyphenidyl decreased plasma CPZ by a mean of 44.7% in 12 of 15 patients. A single 400–800-mg dose of CPZ at bedtime produced steady states equal to or better than those achieved with multiple doses. Those patients who failed to attain CPZ levels of more than 70 ng/ml despite doses of 400–1000 mg/day were receiving lithium throughout the study and had discharge diagnoses of manic-depressive psychosis, manic type, and schizo-affective schizophrenia—a finding with implications for future research.

PHENOTHIAZINES are the most effective and widely used antipsychotics despite the variable and unpredictable clinical response of psychiatric patients to these drugs. With the advent of sensitive assays for plasma chlorpromazine (CPZ), attempts have been made to correlate plasma levels with clinical improvement.

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The authors are with the University of Rochester School of Medicine and Dentistry, Rochester, N.Y. 14642, where Dr. Rivera-Calimlim is Associate Professor of Pharmacology and Toxicology, Dr. Nasrallah was a resident and fellow, Department of Psychiatry (he is now a Fellow at the National Institute of Mental Health), Dr. Strauss is Associate Professor of Psychiatry, and Dr. Lasagna is Professor of Pharmacology and Toxicology.

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Recent observations by Rivera-Calimlim and associates (1) suggest that there may be some correlation between plasma CPZ levels and both clinical improvement and toxicity. These investigators found that patients who did not improve clinically (as assessed by the Brief Psychiatric Rating Scale) generally showed very low plasma CPZ levels (less than 30 ng/ml). Those who did improve generally had plasma levels ranging from 150 to 300 ng/ml. Patients with extrapyramidal signs and convulsions had plasma levels ranging from 700 to 1000 ng/ml and more.

There were, however, some patients whose BPRS scores improved despite low plasma levels, and others who failed to improve despite adequate plasma levels. We attempted to improve the sensitivity of the BPRS by stratifying its items into four subgroups—thought disorders, paranoid behavior, anxious depression, and withdrawal-retardation. In the present study we examined correlations between responses in the various subgroups and plasma CPZ levels. We also investigated the effects of dose, dosage schedule, and drug interactions.

METHOD

The study was carried out by members of the Department of Pharmacology and Toxicology and the Department of Psychiatry at Strong Memorial Hospital and Rochester Psychiatric Center. The significance of plasma CPZ levels in acute and chronic psychotic inpatients was investigated. The clinical pharmacologist responsible for the assay of plasma CPZ had no access to the clinical records of the patients, and the rating psychiatrist did not know the plasma levels or the mode of management being used for the patient. Clinical evaluation was done only for patients with the diagnosis of acute schizophrenia. The effects of dose, dosage schedule, and drug interaction were studied in both acute and chronic patients.

Subjects

The criteria for the selection of subjects included 1) written informed consent signed by the patient or nearest relative, 2) estimated hospital stay of 3–6 weeks, 3) absence of gastrointestinal, urinary, or liver disease,

and 4) the patient's need for CPZ, either alone or with adjunctive treatment, as determined by the attending physician. The acute patients ranged in age from 18 to 37 years and the chronic patients from 40 to 65 years. There was a total of 50 subjects, 21 with diagnoses of chronic psychosis and 29 with acute psychoses.

Assay and Assessment

Plasma CPZ was assayed by the gas liquid chromatography (electron capture detector) technique of Curry (2), modified by Rivera-Calimlim and associates (1).

Response to drug therapy was measured by the BPRS, a 16-item scale of signs and symptoms. Ratings range from 0 (not present) to 6 (extremely severe). Stratification of the 16 items into the subgroups of thought disorder, anxious depression, paranoid behavior, and withdrawal-retardation was done by the method of Overall and associates (3).

The rating scale was scored by the patient's therapist and by one of the investigators (H.N.). Interrater reliability of the BPRS items, assessed by product-moment correlation, was acceptable for 11 items ($r=.70$, $p\leq.002$) and for the total BPRS score ($r=.83$, $p<.001$), moderate but still highly significant for 4 items (conceptual disorganization, guilt feelings, mannerisms and posturing, and blunted affect) ($r\geq.50$, $p=.03$), and unacceptably low for 1 item (depressive mood). We decided that validity could be maximized in the data analysis by averaging raters' scores for the patients. The lower reliability of some items would have the effect of underestimating relationships between the symptoms they reflected and CPZ levels and therefore would err on the conservative side, reducing the chance of spurious correlations.

Procedure

On the day of admission to the study, the clinical condition of the 29 patients with acute psychoses was assessed by the BPRS and blood samples were drawn from all patients for measurement of baseline values of plasma CPZ.

Dose, dosage form, and dosage schedule were left to the discretion of the attending physician, with the agreement that 400 mg/day was the lowest dose to be used and that changes in doses should not be made more than once a week (stabilization of plasma CPZ is assumed to take about a week). The effect on plasma CPZ of addition and withdrawal of trihexyphenidyl was monitored, allowing 1–2 weeks after any change of treatment for stabilization. Plasma levels at 7:00 a.m. and 6:00 p.m. of patients receiving divided doses of CPZ (at 8:00 a.m., 2:00 p.m., 4:00 p.m., and 7:00 p.m.) were compared with plasma levels of patients receiving an equivalent amount of CPZ in a single dose at 7:00 p.m. Within-subjects comparison was done for a few patients, who were given divided doses for 2 weeks and then single doses for another 2 weeks. Assay of the plasma CPZ was done 1 day each week at 7:00 a.m. and 7:00 p.m. for each patient.

Plasma CPZ levels were assayed and the BPRS was administered weekly for 3–4 weeks. Blood samples were collected before (predose level) and 2, 3, and 4 hours after the first morning dose. Patients fasted (except for water) from midnight until the end of the blood collection. Treatment records, BPRS scores, and plasma CPZ levels were matched and analyzed to assess 1) the relationship between change in clinical status and plasma CPZ levels, and 2) the effect of dose, dosage schedule, and drug interactions on plasma levels.

RESULTS

Figures 1–5 show the relationship between clinical improvement and plasma CPZ levels, expressed as the average of 3–4 weekly determinations (allowing 1 week for plasma stabilization). (Horizontal lines within each range represent the mean for patients who fall within that range.)

Figure 1 shows the distribution of plasma CPZ levels plotted against the percentage of clinical improvement as measured by the total BPRS score (change from baseline score to the score at the end of 3–4 weeks).

FIGURE 1
Percentage Global BPRS Improvement as a Function of Plasma CPZ Level

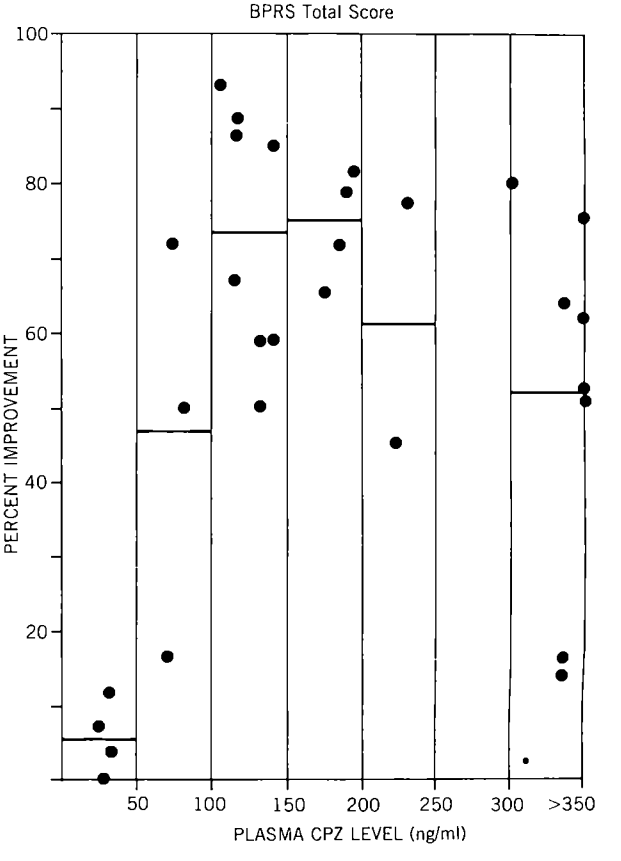
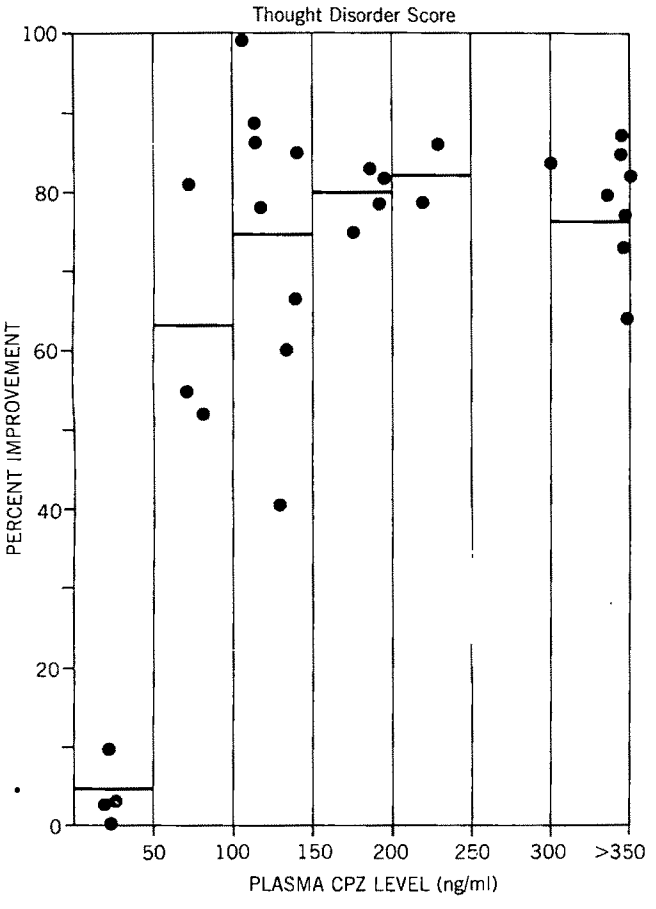


FIGURE 2
Percentage Improvement on BPRS Thought Disorder Score As a Function of Plasma CPZ Level



The 4 patients with plasma levels below 50 ng/ml showed a mean improvement of 5%. Two of the 3 patients who achieved plasma CPZ levels of 50–100 ng/ml showed improvement of 50% to 70%; the third patient had only 16% improvement. Twelve patients who achieved plasma levels of 100–200 ng/ml had a mean clinical improvement of 74%. The mean clinical improvement of the 8 patients with plasma levels of 300–500 ng/ml was lower than that of the 12 patients with levels of 100–200 ng/ml. The records indicated that the patients with the highest plasma levels had more severe baseline symptoms, which apparently necessitated larger doses of CPZ.

Figure 2 shows a clear association between plasma levels and percentage improvement on the BPRS thought disorder score. The 4 patients who did not achieve plasma CPZ levels higher than 30 ng/ml did not improve. Of the 25 patients who achieved levels above 50 ng/ml, 24 showed improvement of 50% to 82%; the 25th showed 40% improvement. As Figure 3 shows, improvement in paranoid delusions also seems to be associated with plasma CPZ levels, but the variation in paranoid delusion ratings is much greater. Figures 4 and 5 reveal no correlation between changes in depression and withdrawal-retardation and plasma CPZ levels.

Table 1 shows the plasma CPZ levels of patients on different daily doses. The results indicate a poor correlation between dose and plasma levels. Although the 3 subjects who achieved the highest levels (patients 10, 11, and 15) received relatively high doses, some patients on very high doses achieved very low plasma levels. Patients given a 400-mg daily dose achieved plasma levels comparable to some of the patients given substantially higher doses. It is interesting to note that plasma CPZ levels during the 4 hours after the dose tended to show a plateau—only patients 7, 8, 16, and 18 showed peaks in plasma CPZ levels.

Table 2 shows the morning and evening plasma CPZ levels in patients on single- and divided-dose schedules given comparable total daily dosages. Patients who were given 400–600 mg daily as a single dose in the evening achieved plasma CPZ levels of 66–264 ng/ml 12 hours after receiving their medication. Their plasma levels dropped by 12% to 50% between the 12-hour and 23-hour samples. Patients given divided doses achieved much lower plasma levels in both the morning and the evening assay than those receiving a single evening dose.

TABLE 1
Plasma CPZ Levels of Acute Psychotic Patients Given Different Doses of CPZ*

Dosage and Patient Number	Plasma CPZ (ng/ml)			
	Predose	2 hours	3 hours	4 hours
400 mg/day				
1	230	240	235	190
2	151	167	151	136
3	217	246	220	215
4	76	90	90	118
600 mg/day				
5	42	63	21	21
6	37	32	30	15
800 mg/day				
7	235	345	424	487
8	183	300	317	270
9	244	253	270	214
900 mg/day				
10	430	482	474	459
11	614	610	640	—
12	164	173	176	152
13	184	160	158	152
1000 mg/day				
14	58	58	72	79
15	539	546	612	514
1100 mg/day				
16	62	78	327	218
1150 mg/day				
17	268	284	300	359
1600 mg/day				
18	88	197	159	161
1700 mg/day				
19	242	108	134	134

* Plasma levels were obtained after 1 week of treatment without any change of dose.

TABLE 2
Morning and Evening Plasma CPZ Levels of Patients Given Single and Multiple Daily Doses of CPZ*

Dosage and Patient Number	Dosage Schedule	Plasma CPZ (ng/ml) Morning	Evening
400 mg/day			
1	Single dose at 7 p.m.	246	217
2	Single dose at 7 p.m.	66	29
3	Single dose at 7 p.m.	264	149
4	Single dose at 7 p.m.	219	144
5	100 mg 4 times daily	57	41
500 mg/day			
6	100 mg 3 times daily plus 200 mg at 7 p.m.	33	16
600 mg/day			
7	Single dose at 7 p.m.	74	41
8	Single dose at 7 p.m.	222	143
9	100 mg 4 times daily plus 200 mg at 7 p.m.	37	15

* Plasma levels were obtained after 1–2 weeks' treatment with the same dose.

TABLE 3
Effect of Addition of Trihexyphenidyl on Plasma CPZ Levels

Patient	Plasma CPZ (ng/ml)* Without Trihexyphenidyl	With Trihexyphenidyl	Percent Change**
1	983	430	(−) 57
2	662	433	(−) 32
3	277	174	(−) 38
4	66	57	(−) 14
5	57	54	(−) 6
6	222	98	(−) 56
7	98	29	(−) 72
8	390	243	(−) 38
9	66	57	(−) 14
10	538	345	(−) 36
11	119	32	(−) 74
12	51	0	(−) 100
13	132	170	(+) 36
14	100	242	(+) 142
15	48	77	(+) 60

* Plasma levels were taken after 2 weeks without trihexyphenidyl and then after 2 weeks with trihexyphenidyl. Levels are expressed as steady state for the particular treatment condition. The trihexyphenidyl dosage was 2 mg three times a day.
** Changes are expressed as percent change from baseline plasma levels; (−) indicates a decrease, (+) an increase.

Table 3 shows the plasma levels of 15 patients who were on CPZ for the first 2 weeks without trihexyphenidyl (Artane) and were then given 2 mg of trihexyphenidyl three times daily for the second 2 weeks in conjunction with CPZ. After the addition of trihexyphenidyl, there was a drop in plasma levels ranging from 6% to 100% in 12 of these 15 patients. To assess the importance of order effects, 4 patients were placed on CPZ and trihexyphenidyl for the first 2 weeks and trihexyphenidyl was withdrawn during the next 2 weeks of CPZ treatment. Table 4 shows that there was an increase ranging from 7% to 318% in plasma CPZ levels after withdrawal of trihexyphenidyl.

FIGURE 3
Percentage Improvement on BPRS Paranoid Delusion Score as a Function of Plasma CPZ Level

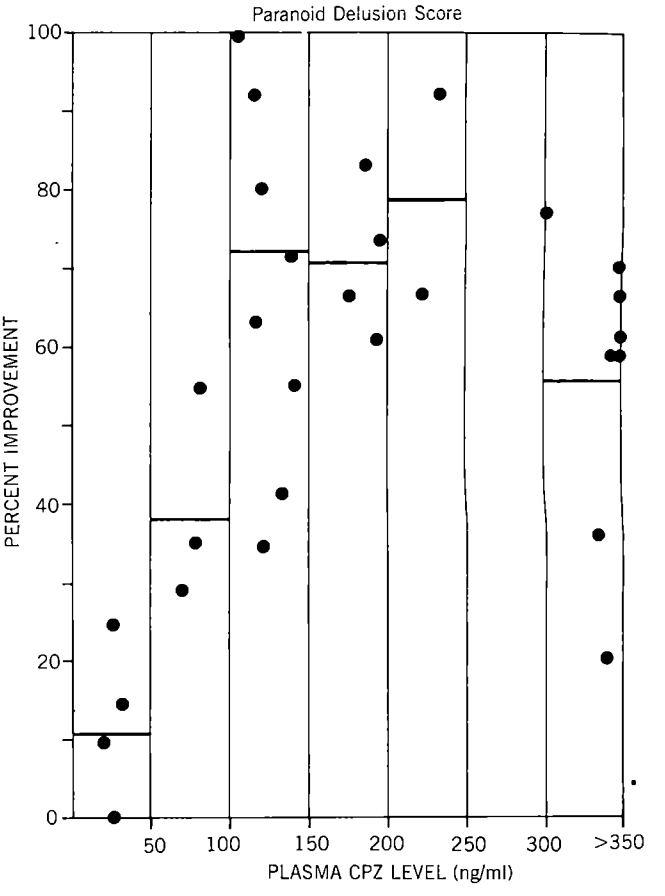


TABLE 4
Effects of Withdrawal of Trihexyphenidyl on Plasma CPZ Levels

Patient	Plasma CPZ (ng/ml)* With Trihexyphenidyl	Without Trihexyphenidyl	Percent Change**
1	164	613	273
2	1143	1225	7
3	174	202	16
4	235	983	318

* Plasma levels were taken after 2 weeks of trihexyphenidyl and then after 2 weeks without. Levels are expressed as steady state for the particular treatment condition. The trihexyphenidyl dosage was 2 mg three times daily.
** Changes in plasma levels, expressed as percent change from initial plasma levels, all represent increases.

FIGURE 4
Percentage Improvement on BPRS Depression Score as a Function of Plasma CPZ Level

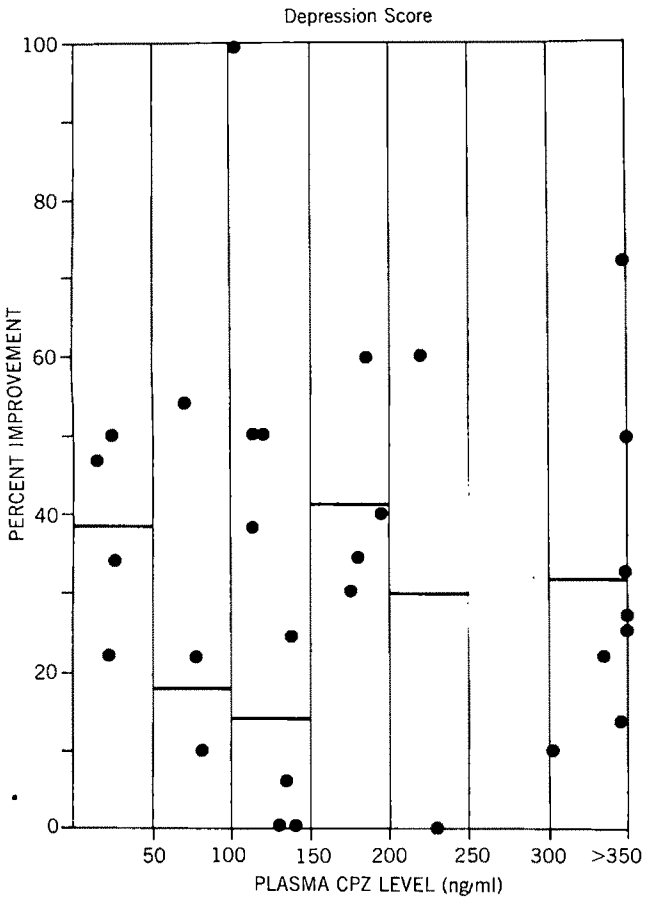
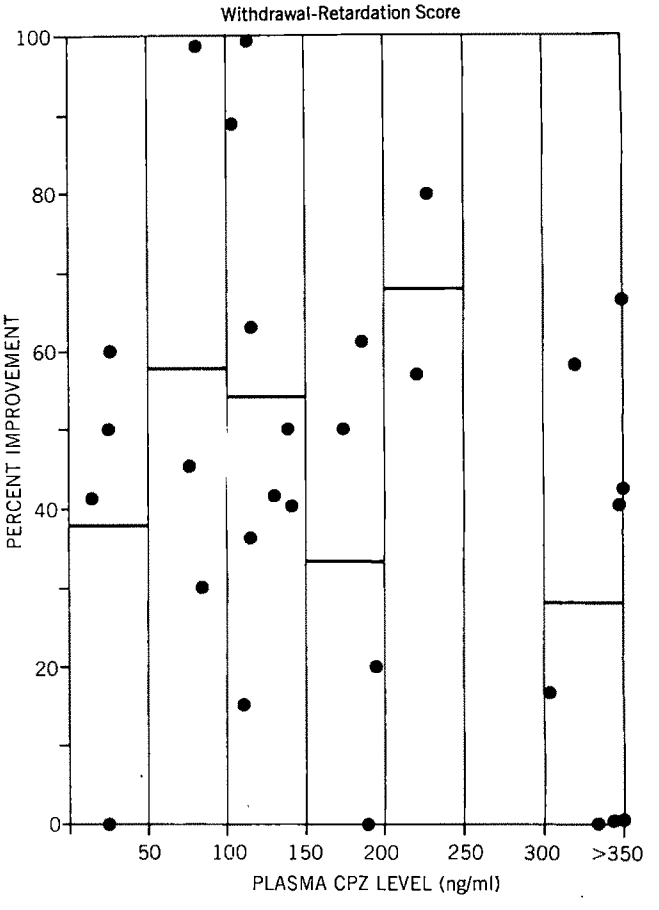


FIGURE 5
Percentage Improvement on BPRS Withdrawal-Retardation Score as a Function of Plasma CPZ Level



DISCUSSION

This study has attempted to substantiate pharmacologically some aspects of phenothiazine therapy that have been accepted for many years on the basis of clinical findings alone. Studies involving thousands of patients during the sixties (4–9) showed that phenothiazine administration produced significant clinical improvement in acute schizophrenics. Clinical relapse is far more frequent in patients for whom phenothiazines are withdrawn than in those who continue on these medications (10, 11).

The findings of this study show that some degree of clinical improvement, based on the global BPRS score, is generally associated with plasma CPZ levels of 50–300 ng/ml, as was found in a previous study in our laboratory (1). When the BPRS score was broken down into four component dimensions, the 50–300 ng/ml range in CPZ level was clearly associated with improvement in thought disorder and reasonably well associated with improvement in paranoid behavior, but there was no correlation between plasma CPZ levels and clinical improvement in depression or withdrawal-retardation. Worsening of blunted affect, motor retardation, somatic concerns, and uncooperativeness was found in several patients, especially those with very high plasma levels (700–1000 ng/ml). These data suggest that studies designed to clarify the relationships between plasma levels and clinical improvement must assess specific endpoints such as symptom groups, as well as overall changes. Evaluating clinical improvement only in a global fashion may mask either beneficial or adverse effects of the drug. Such masking may obscure the ways in which a drug has helped the patient or the possibility that adverse effects may contribute to patients' discontinuing their medication.

Clinical assessment in psychiatry is made particularly difficult by the lack of objective endpoints (e.g., blood pressure, uric acid levels, cholesterol levels, or urine output) and the extreme variation in the treatment goals of individual psychiatrists. Because of this, it is especially difficult to set rules for optimal plasma levels of CPZ or any psychotropic drug. Nevertheless, we tried to determine whether our findings suggested clinically optimal plasma levels of CPZ in the treatment of acutely psychotic patients.

Our results suggest that 400 mg daily is a good starting dose for acute schizophrenics. The data in table 1

suggest that a 400-mg dose often achieves plasma levels comparable to those found with higher doses. There is only a limited correlation between dose and plasma levels, however, so that dosage increases may be required when patients fail to improve or to achieve plasma levels of at least 50 ng/ml.

Several clinical studies have shown that therapeutic outcome does not seem to differ on the basis of single-dose versus multiple-dose schedules of antipsychotic medication (12–16). We measured the morning and evening plasma levels in patients on both schedules, and our results suggest that the single-dose regimen gives more consistent levels throughout the day. Patients who were given a single dose at night showed reasonable plasma levels even after 23 hours. The patients on multiple-dose schedules who achieved adequate plasma levels were those who received additional doses of 200–500 mg at night. Patients who were not given these additional evening doses had low plasma CPZ levels at 7:00 a.m. before the morning dose. It appears from this study that CPZ plasma kinetics may be dose dependent. Even if a single daily dose is given, 35% to 40% of it is distributed and bound in the gastrointestinal tract wall (17). Thus the gastrointestinal tract may act as a buffer to sudden increases in dosage and serve as a depot for the CPZ.

If our findings on plasma levels and dose regimen can be replicated and the superiority of the single dose regimen is confirmed, it will help patients take their medication more consistently, lessen nurses' workloads, and limit the sedative action of CPZ to nighttime, when it is beneficial (or at least not unpleasant).

Our data suggest an adverse effect on plasma CPZ levels of concomitant administration of trihexyphenidyl, an anticholinergic drug often used to combat parkinsonian side effects. In recent years the prophylactic or prolonged use of antiparkinsonian medication in patients receiving phenothiazines has been branded as needless (18–20). Twelve of our patients who had trihexyphenidyl added to the treatment regimen showed a drop of 6% to 100% (mean=44.7%) in plasma CPZ levels. When trihexyphenidyl was withdrawn from 4 patients who had already received it for 2 weeks, the plasma CPZ levels increased.

Thus, trihexyphenidyl (and conceivably other anticholinergic agents) seems to produce a decrease in plasma CPZ. Animal research in our laboratory has shown that trihexyphenidyl slows stomach emptying, thus interfering with the absorption of CPZ (21). Hence unnecessary use of antiparkinsonian medication not only may be economically unwise and cause side effects but also may result in wastage of a significant portion of the CPZ dose in the gastrointestinal tract and possibly compromise the therapeutic effect of CPZ, especially in patients with minimal plasma levels. The anticholinergics probably exert their antiparkinsonian effect at least partly by reducing the level of CPZ in plasma, which is more logically and economically achieved by reducing the dose of CPZ.

Another finding of potential significance concerns the 6 acutely psychotic patients (out of a total of 29) who had low plasma levels, ranging from 0 to 76 ng/ml, despite adequate dosage (400–1000 mg/day). These patients were the only ones who received lithium throughout the study (900–2400 mg/day). Of these 6, 4 had a discharge diagnosis of manic-depressive psychosis, manic type, and 2 had a discharge diagnosis of schizo-affective schizophrenia, excited type.

There are at least two possible explanations for this finding. Lithium may interfere with the absorption, accelerate the metabolism, or increase the excretion of CPZ, or patients in an excited, manic, or maniclike state may absorb CPZ poorly, metabolize it quickly, or excrete it rapidly. Some support for the latter hypothesis is provided by the common clinical observation that manic patients and excited schizophrenics often require huge doses of CPZ before showing a tranquilizing effect. Both of these possibilities present exciting areas for further research.

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American Board of Psychiatry and Neurology Forms Joint Steering Committee on Recertification

The American Board of Psychiatry and Neurology has formed a Joint Steering Committee on Recertification. At its first meeting in Kansas City on February 19, the committee established four subcommittees, which will report to the full steering committee at a meeting to be held in Philadelphia July 14, 1976. At this time the group will initiate plans for a national conference on continuing clinical competence and the related issue of recertification, to be held some time in 1977. The four subcommittee areas are: communications and relationship to the profession, forms of testing, curriculum content of continuing education, and administrative and legal questions.

Richard I. Shader, M.D., and Robert Senescu, M.D., were appointed by President Judd Marmor to serve as APA's representatives on the joint steering committee. The other members of the committee represent the American Board of Psychiatry and Neurology, the American Neurological Association, the American Academy of Neurology, the Child Neurology Society, and the American Academy of Child Psychiatry. The idea for the joint steering committee arose through the ABPN, which as a first step appointed an ad hoc committee. This committee held a planning meeting in Kansas City, Kansas, in October 1975, at which time it recommended that the major psychiatric and neurological organizations be invited to appoint members to a steering committee.

Can Psychiatry Police Itself Effectively? The Experience of One District Branch

BY ARTHUR ZITRIN, M.D., AND HENRIETTE KLEIN, M.D.

The growing demand by consumers and government for greater accountability from psychiatry is challenging the traditional system of self-policing by which the profession's standards of ethical conduct are established and maintained. The authors describe the operations of a district branch ethics committee, including types of complaints received, investigatory procedures, disposition of cases, and factors limiting the committee's effectiveness. They conclude that in some cases lack of funds, lack of time, and the use of peers to judge alleged misconduct make unbiased and thorough evaluation of complaints difficult. They suggest that psychiatry must take steps to improve public confidence in its ability to police itself so that the power to regulate professional conduct will remain within the profession.

MEDICINE in the United States has traditionally been largely self-policed. Society has permitted the profession to establish standards of ethical behavior and norms for quality of medical care; adherence to these standards is monitored principally by medical societies, hospital boards, and committees of professional organizations.

A recent comprehensive review by Derbyshire (1) of organized medicine's disciplinary functions concluded that they were not adequate. He also found that state boards of medical examiners were lax in carrying out regulatory duties, and warned that control of medical discipline might be taken away from the profession unless all agencies involved improved their methods and performance.

Psychiatry is subject to the same procedures and regulations with regard to ethical practice that govern other branches of medicine. *The Principles of Medical Ethics* of the American Medical Association were recently annotated by APA (2) to give them greater usefulness as guides to the acceptable practice of psychiatry. The annotations were formulated to take into account "the special ethical problems in psychiatric practice that differ in coloring and degree from ethical problems in other branches of medical practice" (2).

Data reported at a meeting in May 1974 of the APA Ethics Committee with representatives of the ethics committees of all district branches suggested that the self-policing activities of psychiatry are probably no more effective than those of the rest of medicine. Some district branches have no ethics committee; others have committees that function only as advisory bodies for local medical societies. Local procedures for investigating complaints vary. The use of legal counsel does not appear to be routine.

The recently established policy of reporting all local complaints to the APA Central Office is not uniformly followed, so it is impossible to state with precision the number of complaints received nationwide during a given period. In the year from May 1973 to May 1974, a total of 61 notices about complaints of alleged unethical conduct was sent to APA by the district branches. Sections 1, 4, and 9 of the *Principles of Medical Ethics* (2) were alleged to be violated most frequently (10, 28, and 8 complaints, respectively). These sections read as follows:

> *Section 1.* The principal objective of the medical profession is to render service to humanity with full respect for the dignity of man. Physicians should merit the confidence of patients entrusted to their care, rendering to each a full measure of service and devotion.
>
> *Section 4.* The medical profession should safeguard the public and itself against physicians deficient in moral character or professional competence. Physicians should observe all laws, uphold the dignity and honor of the profession and accept its self-imposed disciplines. They should expose, without hesitation, illegal or unethical conduct of fellow members of the profession.
>
> *Section 9.* A physician may not reveal the confidences entrusted to him in the course of medical attendance, or the deficiencies he may observe in the character of patients, unless he is required to do so by law or unless it becomes necessary in order to protect the welfare of the individual or of the community. (2, p. 1059)

Five complaints of alleged violations of section 3 were reported, 1 of section 5, 2 of section 6, 5 of section 7, and 2 of section 10; no complaints of violations of section 2 or section 8 were received.

Revised version of a paper presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Zitrin is Professor, Department of Psychiatry, New York University School of Medicine, 550 First Ave., New York, N.Y. 10016. Dr. Klein is Special Lecturer, Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, N.Y.

A DISTRICT BRANCH ETHICS COMMITTEE

This report is based on the experiences of one district branch over the past eight years. The branch currently has 1,535 members. Under the provisions of its constitution the Committee on Ethics and Discipline is composed of up to 10 members and is chaired by a past president of the branch.

The general scope of the committee's jurisdiction as set out in the branch's constitution includes "any alleged breach of the Constitution and By-Laws of the Society, or, activities or behavior alleged to be unprofessional. . . ." The language of the APA By-Laws is somewhat different; it refers to complaints "that a member has engaged in unethical conduct or has brought discredit or dishonor on the Association and/or the practice of psychiatry" (Chapter 10, Section 2, By-Laws, American Psychiatric Association).

Committee Procedures

In general, only written complaints about members are referred to the district branch's Committee on Ethics and Discipline, although matters that have come to the council's attention may be considered without a formal complaint. If the Ethics Committee as a whole feels that the alleged offense comes within its province, it refers the matter to an appointed subcommittee for exploration. In almost every case the psychiatrist about whom the complaint is made is interviewed, as is the complainant. It was felt that initial investigation by a small subcommittee would protect confidentiality, be responsive to the sensitivities of those interviewed, and facilitate an open dialogue. A final decision is made after a review of the subcommittee's findings by the entire committee. Occasionally documents are requested and/or other persons or agencies are contacted for relevant information. The Ethics Committee operates independently of disciplinary committees of the county and state medical societies and other local professional organizations.

The scope of the committee's investigatory power is not specified, and it relies wholly on the voluntary compliance of witnesses. So far in our experience no accused physician has refused to cooperate in an investigation or to meet with the Ethics Committee. In a number of instances, complainant and psychiatrist, on the recommendation of the investigating subcommittee, have met separately with the entire committee. Final committee recommendations on each case are submitted to the executive council of the district branch for approval. No final finding of unethical conduct can be made by the council without a full hearing, at which the accused person has the right to examine witnesses, produce witnesses for himself, and be represented by counsel. A vote for disciplinary action by the executive council can be appealed to the district branch membership.

An attorney specializing in psychiatry has been retained on an ad hoc basis to advise the Ethics Committee and the executive council.

Type of Complaints Received

During the period from 1970 to 1974, 44 complaints alleging improper conduct by district branch members were considered by the committee, four times as many as during the previous four years. A wide variety of offenses was charged; they can be grouped under the following headings, according to the major impropriety alleged.

Category 1: a miscellaneous category containing complaints of rudeness, suggestive sexual comments, inadequate time for examination, and delays in specific appointments (9 complaints).

Category 2: charges of undue influence, such as those relating to investments or marriage choice (6 complaints).

Category 3: alleged violations of the prohibition against sexual relations with patients (5 complaints).

Category 4: allegations about a doctor's lack of cooperation in filling out insurance forms or providing statements for tax purposes, including complaints that a doctor refused to send such forms until his bill was paid (3 complaints).

Category 5: complaints about improper advertising (3 complaints).

Category 6: complaints about excessive fees (3 complaints).

Category 7: charges of improper diagnosis resulting in loss of job (3 complaints).

There was a variety of other complaints, such as interference with ongoing psychotherapy by another psychiatrist, refusal to agree to consultation, physical assault, improper treatment, and excessive prescription of drugs.

The Ethics Committee voted for a finding of unethical conduct in only two instances. (In many other cases the committee felt the charges were justified but lacked sufficient proof to warrant a finding against the doctor.) In one case, the physician violated section 1 of the *Principles of Medical Ethics* by exerting undue influence on a patient. In the second case a physician who charged an excessive fee was found to be in violation of section 7.

Relatively few complaints were frivolous, malicious, or patently without merit. Many had been lodged only after considerable hesitation by the patient, or only after they had been urged to do so by a physician. In two cases the complainants were psychiatrists.

Difficulties Encountered by the Committee

The committee became aware quite early in the course of its work of a number of serious difficulties and ambiguities that often made discharge of its duties difficult and sometimes impossible. In several instances the accused physician was known to all members of the committee. In such cases, could the committee be viewed by the complainant and the charged psychiatrist as unbiased?

In some cases it was unclear whether a complaint came properly under the committee's jurisdiction. Was a matter of ethics involved, or a question of quality of medical care? How far could an ethics committee go in its investigation of a complaint? How much could it accomplish without a resource staff? There were a number of serious complaints in which additional evidence should have been sought, but this would have required time-consuming investigation that lack of staff precluded. In some cases patients had also filed civil suits against the physicians about whom they complained to the district branch. The advice of legal counsel was used to guide the committee on these matters.

The difficulty in determining precisely what takes place in the patient-psychiatrist encounter was underscored in cases in which the committee had to choose between two conflicting versions of a story, that of the patient and of the doctor. In one instance the committee members, all highly experienced psychiatrists and psychoanalysts, were divided as to where the truth lay. In this case a female patient accused her male physician of having had sexual relations with her over a long period of time. Lack of corroborating evidence precluded a definitive finding.

In general the committee was reluctant to widen investigations by referring complaints to legal or administrative governmental agencies. The criteria for doing so were unclear, and the district branch council never requested that the Division of Professional Conduct of the Department of Education (the state licensing and medical disciplining agency) conduct its own investigation of a complaint.

Although a clear and fair determination of whether unethical conduct had taken place was possible in most cases, in some instances no such certainty prevailed. Some members became increasingly uneasy in their committee role because they wondered whether the committee served merely as a buffer between the public and the profession, since it was without the resources, the objectivity, and (perhaps) the will to do more.

DISCUSSION

Today we are witnessing public demand for a more equitable distribution of health care, and government involvement in the delivery of such services is rapidly accelerating. Significant changes in public attitudes toward physicians are taking place. One authority on social policy and health services has warned that "the mystique that hitherto surrounded medicine is decaying, and there will be growing public support for public evaluation of the system as a whole, as well as its individual parts, and for the use of controls and sanctions" (3, p. 85).

This rise in "consumerism" is reflected by the increase in malpractice suits, the large settlements imposed by juries, and insistent demands for greater accountability of the medical profession. The nation's leading consumer advocate organization has concluded that medicine has failed to merit the public's trust that the profession will successfully regulate itself (4, p. 153).

What steps can psychiatry take to restore public confidence in its ability to police itself? The present arrangements for self-discipline will not long suffice; a system in which district branch committees attempt to monitor the actions of psychiatrists appears to be unrealistic and bound to fail. Small groups of busy volunteer psychiatrists without staff or funding cannot be expected to be investigators, prosecutors, judges, and juries in cases involving the reputations and professional careers of friends and acquaintances, even in a large psychiatric community such as ours. Even if they could do so, this system is unlikely to satisfy the demands of the rising consumer movement.

The most important factor ensuring ethical practice will always be the physician's own high standards of behavior and personal dedication to professionalism. The annotated *Principles of Medical Ethics* (2) remain excellent guidelines for psychiatric practice, and it may well be that their existence and the possibility of censure by peers are still strong deterrents to unprofessional conduct. However, additional peer review procedures need to be established if control over professional standards is not to pass out of the hands of the profession. The Professional Standards Review Organizations (PSROs) set up by section 249-F of Public Law 92-603 may well be the vehicle for a workable peer review system.

Recent reviews have commented on the difficulties of peer review in psychiatry (5–8), such as the inadequacy of psychiatric records, the limited usefulness of psychiatric diagnosis, variations in criteria for hospitalization and discharge, problems of confidentiality, and the complex nature of psychotherapy. As PSROs address these problems and formulate standards, they should also develop procedures for self-discipline that will overcome the shortcomings and failures of the present system.

Our district branch, through its Committee on Peer Review, has surveyed review practices in general hospitals in New York City and has developed some fundamental principles, methods, and strategies for peer review. The Committee on Peer Review also acts as a liaison to the PSRO conditionally designated by the Department of Health, Education, and Welfare for New York County. A joint committee composed of the chairpersons of the district branch committees on insurance, Medicaid, peer review, and ethics has recently been charged with coordinating the activities of these four groups.

It is likely that these new committees will develop more effective mechanisms for self-policing than now exist. More psychiatrists, particularly in hospitals, will soon have their professional work come under scrutiny by peer review bodies. However, unprofessional conduct will still be more difficult to deal with than professional incompetence. Reporting by physicians of un-

ethical conduct by colleagues is quite rare, and it will be difficult to provide incentives to alter this fact.

CONCLUSIONS

We believe that investigation of complaints against a physician should not be made by colleagues who know the accused, as is now often the case. The establishment of a statewide panel of psychiatrists, perhaps by the New York State Council of District Branches, would provide panelists who could investigate cases in cities other than their own. Staff and expense funds could be provided on an ad hoc basis. Although expensive and time-consuming, such a procedure would be a step toward demonstrating to the public the profession's willingness and determination to take care of its own house, and it might also help still demands for consumer participation in the policing procedure. What is now lacking is not a suitable definition of standards of professional psychiatric conduct but public confidence that psychiatry has the will to enforce these standards.

At this time in the history of psychiatry and the APA, with the current concern about organizational improvement and internal democratization, it seems as if the patient is sometimes forgotten. The goal of all our deliberations—about organization, training, research, and continuing education—must be improvement of the care of patients. Increased accountability, to ourselves and to the public, including better procedures for self-regulation, will be an important step toward that goal.

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Cultural Perspectives on the Impact of Women's Changing Roles on Psychiatry

BY VIRGINIA ABERNETHY, PH.D.

The author examines sex role stereotypes in the United States and their impact on the self-concept of women. She suggests that the fact that women's roles are changing may have a positive impact on general mental health because both men and women will profit from increased flexibility in gender role specifications. She emphasizes the fact that therapists as well as their patients have been socialized into accepting the values and assumptions of our society; there is thus a high probability that not only the presenting complaints but also the treatment goals for women in therapy are culturally determined.

ALTHOUGH THE IDEAS of the women's movement are still difficult to understand and alien to many, the social pattern embodied in renewed agitation for women's rights can be seen as a fundamentally creative effort to innovate and grow that is in harmony with the underlying tenets of the mental health professions. The objectives of this paper are to show ways in which the new directions taken by women today are constructive and, in view of the charges of sexist bias that have been leveled against some mental health professionals, to suggest that the source of conflict is culture change rather than a war of patients against therapists or, still more destructive, women against men.

CULTURAL DETERMINISM

A premise of anthropology is that all of us are bound by our culture. The deeply held ideas, values, or beliefs of our society inevitably represent reality for us. This phenomenological sense of authenticity may be so great that we remain unconscious of the extent to which our cultural assumptions are mental constructs. Moreover, as long as we remain within a single stable culture our certainty of its assumptions is continually reinforced because culture is the shared contents of minds. Both therapists and patients are products of our culture; therefore, they are similarly determined, or bound, by our society's attitudes and behavior patterns.

Dr. Abernethy is Director, Division of Human Behavior, Department of Psychiatry, Vanderbilt University School of Medicine, Nashville, Tenn. 37232.

When there is no free choice, responsibility and recrimination are irrelevant. However, freedom from responsibility coupled with absence of free will is not a condition in which many might wish to remain. Just as psychoanalysis has the goal of exposing and thereby freeing an individual from the determinations of his or her developmental history, so an awareness of cultural forces can enlarge an individual's range of free choice.

Awareness of how women are constrained by our culture can be focused by considering sex role stereotypes. Research suggests that sex role stereotypes in our culture are pervasive, that they exert a major influence on self-concept and behavior, and that they may present an obstacle to the growth and mental health of both men and women (although they are more particularly damaging to women). Health care professionals necessarily share the stereotypes of their culture. Therefore, therapeutic goals are sometimes limited by culture-bound preconceptions of what is ideal or adaptive male or female behavior.

SEX ROLE STEREOTYPES

A number of research teams in the United States have reported congruent findings on ideas of what is archetypically male and female (1–5). Especially impressive is the work of Broverman and associates (6), who developed a sex role questionnaire consisting of 41 empirically derived items that distinguish between popular conceptions of the average man and the average woman at highly significant levels. Broverman and associates tested nearly 1,000 subjects in samples that covered a broad spectrum of religious, socioeconomic, sex, and age categories; they were consistently able to replicate their findings. The stereotypically masculine traits they identified clustered around the concept of competency; the stereotypically female traits clustered around the dimensions of warmth and expressiveness.

Social Desirability

The isolation of sex role stereotypes is in itself not so striking as Broverman and associates' discovery that the masculine cluster of attributes was more socially desirable than the female cluster: 29 of the 41 stereotypic traits at the masculine pole of the scale were judged to be not only more desirable but also more descriptive of a healthy adult; by comparison, only 12 of

the 41 traits at the feminine pole were judged to be more desirable and part of the healthy adult profile.

A sampling of the socially desirable items that clustered at the masculine pole follows: aggressive, independent, not emotional, hides emotions, objective, not easily influenced, dominant, active, competitive, logical, and worldly. The complete list of the socially desirable items that clustered at the feminine pole is as follows: doesn't use harsh language, talkative, tactful, gentle, aware of feelings of others, religious, interested in own appearance, neat, quiet, needs security, enjoys art and literature, and easily expresses tender feelings.

Self-Concept

The data of Broverman and associates further suggest that the skewed social desirability of the stereotypes impinges in a negative way on women's self-concept. Not only were there fewer positively valued feminine traits, but women, unlike men, appeared to incorporate the devalued characteristics attributed to their sex along with the positive characteristics. It appears that women saw themselves not only as sensitive, gentle, and expressive but also as subjective, easily influenced, passive, irrational, and not independent. Men, however, described themselves as possessing not only the positively valued competency attributes that were judged stereotypic for the average man, but also the traits of the feminine cluster that were positively valued for the average woman.

It is not known to what extent poor self-concept may be crippling. It seems a reasonable hypothesis, however, that a woman's internalization of a negative evaluation of female capabilities promotes her withdrawal from competition. This would then be a factor in the well-known tendency for girls' school performances to begin to deteriorate around high school age and in the associated downgrading of achievement goals among adolescent girls (2, 3).

Innate or Learned?

Justification for the stereotypes usually hinges on the protest that they represent the natural order and, therefore, nothing can be done. Thus it is necessary to confront a different order of question, i.e., whether the stereotypes are in fact an accurate reflection of male and female potentials independent of socialization pressure. In other words, are the differences between men and women innate or learned? Furthermore, if they are innate, do they exist in a small degree or in the magnitude suggested by the stereotypes?

Some assert that innate differences in any of the stereotypic traits must be negligible because observable variations within a sex are of greater magnitude than the difference between the averages of each sex, i.e., there is enormous overlap of male and female distributions. Clearly, the evidence that will resolve this hard question is not and may never be in. However, the facts that girls perform academically at least as well as boys up to the age when career choices begin to be made and that only after adolescence do discrepancies in male-female achievements favor the male (3) must present a problem for those who believe in innate differences in competence.

Similarly, statistics on the prevalence of various mental disorders in childhood show that boys greatly outnumber girls in every category, be it learning difficulties, behavior disorders, or childhood psychoses. However, among adults the prevalence of almost all categories of mental illness is reversed: women are sicker than men (4).

What causes this shift? Are adult, but not developing, women weaker than men? Is labeling applied differentially to men and women? Is the environment perhaps more pathogenic for women than for men? It is at least a plausible explanation of these related phenomena that the dawning realization of the culturally determined expectations for women and the choices closed to them causes young women to constrict their horizons, thus increasing their susceptibility to psychological disorders.

The issue of innate differences was directly addressed by Mead in *Sex and Temperament* (7), which described a New Guinea society in which women assume responsibility for the management and productive functions of the economy. These women exhibit characteristics associated with what our society judges to be competence to a greater degree than men, who are passive, narcissistic, and, to the extent that they dwell on jealous intriguing for the women's attentions, could be seen as masochistic. These findings suggest that differences between men and women along the specific dimensions of our cultural stereotypes are not so great as to be immutable. Role reversal is possible.

Finally, although ongoing investigation of innate male and female behavior is identifying some differences in predispositions between the sexes, these appear to be tangential to the stereotypic attributes. The apparent differences are that the male's activity level is higher (8, 9) and that from the embryonic stage onward the female's tolerance for stress is greater (10, 11). In addition, the cyclicity of female hormonal production probably exaggerates and inhibits certain traits in a rhythmic way; there is some evidence that there is an increase in sexual drive and in sense of competence and well-being around the time of ovulation (9, 12, 13).

The feminist position is that the issue of innate differences is a red herring. Because it is indubitably true that socialization accounts for at least some of the differences between male and female personalities, is it not a truer form of justice and a straighter road to mental health to endeavor to equalize the self-concept, training, and opportunities available to men and women (5, 14, 15)?

PSYCHOTHERAPEUTIC PRACTICE

The evidence is mixed as to whether the average mental health professional provides constructive and

appropriate support to female patients. The findings of Haan and Livson (5) suggest that female therapists are more critical of close adherence to sexual stereotypes by either male or female patients but that male therapists are more concerned by deviance from sexual stereotypes. However, other data (6) suggest that both male and female therapists evaluate patients against an idealization of these stereotypes.

The Broverman team (6) found not only that male and female mental health professionals agreed with each other on descriptions of characteristic differences between men and women but also that a comparison of clinicians' profiles of the average man and the average woman revealed a negative evaluation of the female character:

> The clinicians' ratings of a healthy adult and a healthy man did not differ from each other. However, a significant difference did exist between the ratings of the healthy adult and the healthy woman. Our hypothesis that a double standard of health exists for men and women was thus confirmed: the general standard of health (adult, sex-unspecified) is actually applied to men only, while healthy women are perceived as significantly *less* healthy by adult standards. (p. 71)

From these data it appears that some clinicians share the general cultural sex role stereotypes. The further inference is that these stereotypes color their perception of patients, their clinical judgments about prognosis, and, most vitally, their interactions with female patients. The net effect is probably that they promote women patients' choosing the traditional female roles and self-concept. The less healthy aspects of the feminine personality may be reinforced because so much of what is healthy is considered to be specifically masculine. It is difficult to allay the suspicion that in therapy just as in the culture generally a woman is rewarded for unaggressiveness, irrationality, dependence, passivity, and emotionality and that she may be attacked for exhibiting the opposite traits, which are judged by clinicians to be healthy in the socially competent adult person.

There are limits to inferences that can be drawn from therapists' responses to questionnaires or to hypothetical patient profiles. Clinical evidence that treatment varies systematically with the sex of therapist and patient is usually anecdotal. However, those who wish to be sensitized to this area of physician-patient interaction will find documentation in Asher's report on the American Psychological Association's casebook on sex bias and sex role stereotyping in psychotherapeutic practice (14).

According to Asher (14), patients' complaints focused on the male therapist's tendency to view women's problems in terms of unfeminine assertiveness, lack of compliance, inattentiveness to a husband's emotional requirements, and failure to put the needs of the marriage above personal satisfactions. The patient's career or aspirations may be depreciated, and the therapeutic goals might be set in terms of acceptance and adjustment to the roles of wife and mother. In addition, a woman's assertiveness is not infrequently interpreted as penis envy, and her attitudes toward childbearing or child rearing may be used as indices of emotional maturity.

THE RELEVANCE OF PSYCHOTHERAPY

Value judgments based on our society's traditional view of men and women carry a therapist a vast distance from the currents of the present cultural trend, which is gradually legitimizing a broad range of roles for women. Many women no longer docilely accept the idea that their psychological difficulties lie within themselves and that they must therefore strive to adapt to difficult environmental situations. Instead of trying to change themselves, their newly legitimized objective is to change or reject the difficult social environments (16).

Specifically, to view "feminine assertiveness as a neurotic phenomenon . . . aimed at overcompensation for and denial of the lack of a penis" (15, p. 90) is beginning to conflict with the ideas of our culture. On the contrary, large numbers of women accept the idea that women envy men for their social power and for the cultural permission given them to be competitive, rational, competent, and sometimes just plain ornery. Moreover, many women are asking why a mature woman should want a penis as part of her anatomy. Consider a contemporary feminist joke: A mother consoles her young daughter who feels outdone by a playmate's external anatomy, "Honey, with what you've got, you can get all you want of what he's got."

In order to be relevant to women today, therapists are asked to be creative, open, and nondirective. The fact that the therapist as an individual is a product of the traditional culture does not prevent him or her as a professional from recognizing that attitudes toward women have been culturally determined and do not necessarily represent truth. In the service of promoting mental health, a therapist can support a woman's endeavor to think and feel better about herself, to keep from becoming (in a self-fulfilling prophecy) a dependent, passive, and ineffective nonadult.

It should be recognized, however, that some women find that the best adjustment for them lies within the dependent stereotypic role model; increasing numbers, however, are questioning, distressed, or raging at the confines in which their lifestyle places them. The constructive therapist can provide support or insight for the change compatible with each patient's strengths and wishes.

In this context, it may be useful to repeat that many patients are as unaware as any traditional therapist that their treatment goals might be culturally prescribed and sex specific. Because of gender stereotyping, which until recently virtually all women accepted, both patient and therapist may misinterpret the

presenting complaints. Thus treatment may fall short of its potential to stimulate growth.

MARRIAGE AND FEMINISM

A further issue for the therapist is the impact of women's new attitudes and roles on a male-female relationship and, by extension, what the therapeutic goals for this dyad should be. This is an area in which there has been pessimism because of the prevailing view that a marriage necessarily suffers if a woman puts her own needs first. I wish to suggest on the contrary that the effect of the new trend is positive and that this will be increasingly evident as equality is more fully accepted and women are no longer pushed into positions of feminist militancy.

The basis for this healthier male-female relationship is that women who acquire social power for themselves in a career or professional setting are able to empathize in a new way with men who have always had this power and responsibility. Not only do the couple share new areas of common experience but the woman now has a sense of independence and competence and is therefore not threatened by the man's occasional needs to be passive, emotional, and taken care of.

The traditional culture denies men permission to express passivity and vulnerability. This is inevitable as long as husbands bear the sole responsibility for the security of dependent, stereotypically feminine wives. The artificial and rigid rules for masculine behavior can relax, however, as new rules for feminine behavior apply. A reprieve for women is a reprieve for men because as the culture comes to allow women greater scope for exercise of competence men will benefit from a complementary permission to be more expressive and less determinedly, exhaustingly invulnerable (16).

The prediction that male and female stereotypes will become increasingly blurred, to the benefit of both men and women, does not carry the implication that there will be role reversal in intimate dyadic relationships. Logically, it should be clear that for a woman to have an independent power base because of her societal role in no way determines the balance of power between her and any particular man: an increase in the overall competence and independence of women carries no intrinsic threat to male control of individual intimate relationships.

There is no evidence that a marriage is well served by the husband's being the weaker partner, but there is evidence to the contrary (17). Some have speculated that a cause of the currently high divorce rate is that as the traditional marriage develops, the woman becomes the more powerful personality. This imbalance occurs because of the cultural obstacles put in the way of a young woman's realizing her potential at a reasonable age. In the present pattern, a woman limits her self-concept and marries what seems to be a paragon of strength relative to her culturally prescribed weakness; the woman's later, unanticipated growth upsets the balance of the marriage.

I suggest that the trend should be toward more careful mate selection and that marriage should be delayed until both men and women have found occupational identities for themselves and have come to terms with who they are and can be. It is worth noting, in this context, that fostering achievement goals in young women is compatible with delayed marriage. Indeed, it might be sufficient cause for development of a delayed marriage pattern.

CONCLUSIONS

There is inherent contradiction in a health care tradition that systematically forces women into a stereotypic role that is judged by both clinicians and public opinion to be inferior to a healthy adult level of functioning.

It also appears that our culture is changing: new expectations, behaviors, and choices are opening to women. If the medical and mental health professions are to remain relevant, they need to be open in dealing with the changes. Conflicts and problems will inevitably arise; the opportunity for creativity lies in viewing these as manifestations of culture change rather than as the wrongheadedness of men or women.

The greatest challenge and field for innovation may lie in developing a model for marriage that is habitable by two fully adult persons. Men as well as women can derive greater satisfaction from a marriage in which both partners are allowed a full range of human expression—from competence and rationality to passivity and tenderness. Both men and women are, by turns, tough and soft. By accepting these needs in each other, they can be friends.

Therapists are participants in and products of their culture. Therefore, when the evolution in sex roles is completed, that transition will have been made in the mental health professions as well. However, why proceed in lockstep with the culture? By understanding the forces that shape our minds it is possible to be objective about the different and conflicting currents that are swirling around us today and to use our knowledge and self-knowledge creatively.

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Journal Policy on Multiple Authorship

It has come to our attention that there has been some misunderstanding regarding the *Journal*'s policy on polyauthorship. The number of authors listed on a paper is not a factor in the review and acceptance/rejection procedure. However, once a decision has been made to publish a paper, we do apply *Journal* policy regarding the number of authors in the by-line: only principal researchers and writers of articles are listed in the by-line; collaborators who have provided technical and other assistance are acknowledged in a footnote. In papers that list more than 4 people as principal authors, we query the first author to verify that those individuals were substantial contributors to the research or ideas reported. Since certain types of psychiatric research may require multiple workers, we will of course include all of the names in these cases.

The Behavior of Hypersexual Delinquent Girls

BY JEROME D. GOODMAN, M.D.

The author presents case reports of seven girls with histories of hypersexual behavior associated with delinquency, aggressive behavior, and hypomania. Six of the girls had been exposed to virilizing hormones in utero or in childhood. Most of the girls in the sample were hirsute, tended toward mesomorphic build, and had been young at menarche. The author postulates that exposure to virilizing hormones can result in the kind of hypersexuality and delinquent behavior observed in his sample; he points out the need for adequate medical attention to this type of patient.

THE ASSOCIATION of problematic sexual behavior and juvenile delinquency in adolescent girls is commonly seen in juvenile courts. The pathway to the court for girls has traditionally involved charges of incorrigibility and running away, both of which carry implications of sexual behavior beyond parental sanction. Characterizing such sexual behavior as nurture-seeking rather than mature sexuality, Russell (1) found that the behavior of these girls had largely been allowed to run a chronic course before legal action was considered. Gianturco (2) has described problem sexual behavior in girls who had high scores on the Minnesota Multiphasic Personality Inventory scales for psychopathic deviate and hypomania.

Part of the upsurge in problem sexual behavior among adolescent girls is culturally related; contraceptives are more available now than formerly and there is greater acceptance of sexual behavior. Commenting on the changes in the sexual behavior of adolescents in the 1960s, Schmidt and Sigusch (3) cited as one factor the lower age at which the initial sexual experience takes place. There are probably biological determinants as well. The modern-day compression of childhood is a real phenomenon. The average age at menarche has dropped 3 or 4 years in the century since statistics have been recorded (4).

Early sexuality in adolescent girls can also reflect psychodynamic factors (5). Allen (6) described cases of hypersexuality as psychogenic in etiology. The surface appearance of hypersexuality, however, with a history of poor impulse control, rebellion against societal constraints and parental authority, and self-defeating patterns of promiscuity, does not preclude some true precocious priming or excess of libido. Freud, in discussing what he termed a powerful masculinity complex wherein some young girls identify with a phallic mother or father, asked, "What can it be that decides in favor of this outcome?" He answered, "We can only suppose that it is a constitutional factor, a greater amount of activity, such as is ordinarily characteristic of a male." (7, p.594).

Studies and clinical observations over the last 20 years have supported the thesis that biological libido, that is, the driving sexual force, is directly correlated with androgen levels. As Stoller (8) has written "Libido is clearly not dependent on estrogens in women but is probably the result of minute amounts of androgens normally produced in the adrenals . . .." In the case of adrenocortical carcinoma, for example, women can become rapidly virilized, with sudden onset of increased libido (9). The administration of methandrostenolone, an anabolic agent with known virilizing potential, can also result in hypersexuality.

There are other possible determinants of very active sexual behavior in young women. Manic episodes are often associated with hypersexuality, as are hyperthyroidism, certain lesions of the central nervous system, and other physical abnormalities. It is also true that some quite sexually active young women simply represent one extreme on the bell-shaped curve of biological variation.

Over the last 7 years I have examined a large number of adolescents for the Court of Juvenile and Domestic Relations in northern New Jersey. In a combined private and hospital-based practice, I have seen more than 400 adolescent girls at the direction of the court. Of these, 7 girls presented histories of unusual hypersexuality associated with delinquent behavior. These 7 girls underwent psychiatric examination, complete psychological test batteries, and a social investigation by probation officers. All of them were later followed in psychotherapy. Table 1 summarizes the behavioral and physical characteristics of these girls.

Revised version of a paper presented at the 128th annual meeting of the American Psychiatric Association, Anaheim, Calif., May 5–9, 1975.

Dr. Goodman is Assistant Clinical Professor of Psychiatry, Columbia University College of Physicians and Surgeons, New York, N.Y. Address reprint requests to him at 45 West Saddle River Rd., Saddle River, N.J. 07458.

TABLE 1
Physical and Behavioral Characteristics of 7 Hypersexual Girls

Item	Number
PHYSICAL CHARACTERISTICS	
Hirsutism	6
Acne	5
Voice	
Low	4
Normal	3
Prenatal exposure to progestins	4
Age at menarche	
8–9	3
11–12	4
Age at onset of hypersexuality	
9–10	2
11–12	2
13–14	3
IQ	
111–120	4
121–130	1
131–140	2
BEHAVIORAL CHARACTERISTICS	
History of tomboy behavior	3
Running away	7
Suicide attempts	4
Stealing	5
Homosexual activity	5
Hypomanic episodes	7
Assaultive behavior	4

CASE REPORTS

Case 1. A 15-year-old girl was seen in emergency psychiatric interview because of her agitated condition. She had made two suicide attempts by slashing her arms, and she carried scars of the 13 sutures required to close the wounds. There had been brief psychiatric intervention previously, but the major therapeutic effort was directed toward environmental measures (e.g., change in educational programs, different housing, and monitoring her association with peers) and family counseling. This young girl admitted having used LSD, methamphetamine (Methedrine), and marijuana.

When first seen in psychiatric examination, the girl appeared older than her years. Her facial acne and hirsutism were masked with cosmetics. She spoke in an unbroken torrent, pouring out historical material with seeming unconcern for censorship. This unguarded outpouring was perhaps the most striking characteristic of the initial interview. She told many family secrets and stated that she had only recently learned that her mother and father were not married at the time of her birth. Her affect was euphoric and she spoke very rapidly in a hypomanic fashion. She told of disturbed family relationships and described both parents as alcoholics.

The girl told about the separation of her parents, which lasted from her infancy until she was 9 years old. During these early years she was influenced considerably by the attitudes of her maternal grandmother, with whom she was placed periodically. When she rejoined her parents at the age of 9, she had pubic and axillary hair and had begun to menstruate. When she was between the ages of 13 and 15 her father began to make sexual advances toward her, culminating in intercourse shortly before her first suicide attempt. She had welcomed these advances and considered the evening rendezvous a special bond with a father who had deserted her as an infant.

She found that her sexual longings were intense and began to seek outlets more frequently. Approximately 6 months before her first suicide attempt, when she was 14 and her father was becoming increasingly bold sexually, she started dating one boy in particular. She was aggressive in securing this boy's attention. In addition, she often attended dances to pick up boys, and would perform multiple fellatio in order to demonstrate that she could entice a large number of boys in rapid succession. She also had a short-lived homosexual affair in which she acted the part of the masculine tribade.

At this time her breasts—which had been full—began to regress in size. She began to envy other girls and was particularly attracted to their breasts. Her account of this included "riding up on top and finger banging" another, more passive girl. Her entire fantasy life, dreams, and many of her activities were devoted to proving herself as a female. She was concurrently battling with hirsutism, diminishing breast size, irregular menses, and other physical signs of dwindling femininity. As her physical femininity seemed to be deserting her, her libido increased, and she began to engage in sexual activities of a polymorphous, perverse nature. During the 2 years prior to the suicide attempt she was in counseling, but her parents, especially her father, did not follow through in any consistent plan of psychiatric treatment. The girl made both of her suicide attempts after arguments with her father. Both attempts involved lacerations inflicted by razor blade. The first took place in her home, the second in a women's rest room in a public park. Both were serious attempts involving arterial bleeding. In the second attempt, she was discovered by a young man who "had the nerve to show me his prick. . . . I tried to cut it off. . . . I really would have." After she had recuperated from the second suicide effort she became more rebellious, impulsive, and provocative. It was at this time that she was separated from her parents and surroundings.

This 15-year-old girl was initially very unpredictable. Her ego strength was variable. She used rapid-fire, intense language with coprolalic expressions woven into her vocabulary. She filled the first six sessions with sexual material. She seemed to enjoy any shock value she could create, relating almost as though she were a young man bragging of his unending conquests. Her mood fluctuated between euphoria and depres-

TABLE 2
Serial Laboratory Assay of a 15-Year-Old Girl with Hyperadrenocorticism

Substance	Time of Assay							
	Initially	First Month	Second Month	Third Month	Fourth Month	Fifth Month	Sixth Month	Seventh Month
17-Ketosteroids (mg/24 hours)	16.50	27.10	25.00	15.80	7.28	17.97	6.45	5.45
17-Ketogenic steroids (mg/24 hours)	17.91	63.33	50.40	20.40	7.50	18.58	13.50	10.30
17-OH Corticosteroids (mg/24 hours)	9.90	34.50	19.25	8.23	2.28	15.35	3.80	2.40
Testosterone (ng/24 hours)	85.80	87.80	—	25.30	—	—	—	—
Pregnanetriol (mg/24 hours)	2.07	4.57	—	0.60	—	—	3.21	—
Prednisone (mg/day, orally)	—	—	7.50	7.50	5.00	—*	5.00	5.00

* The subject stopped taking prednisone during the fifth month.

sion. It was at this time that consultation for endocrinological studies was begun. The girl's long-winded descriptions of her sexual dreams and memories seemed nonending. She began bragging about clandestine meetings with young men in the early morning hours. Her grandmother brought the matter to the attention of the juvenile court, and the girl was hospitalized; this provided the opportunity for more intensive medical investigation. Sexual material continued to occupy most of the clinical hours at this time.

Endocrinologists and clinical pathologists diagnosed her condition as acquired adrenal hyperplasia. Serial laboratory determinations are summarized in table 2. After treatment with steroids, bioassay revealed the expected decrease in adrenal substances, including the androgenic substances testosterone and pregnanetriol. There was a noticeable change in her attitude and thought processes only a week or two after the onset of steroid therapy. She became much more interested in schoolwork and became "feminized" in that her vocabulary, interests, and entire manner of presenting herself were vastly softened. Her mood was generally elevated and sustained from the previous intermittent dysphoria. Over a period of 3 months she became more interested in personal grooming, grew her hair to nearly shoulder length, and began dating again. Her sexual drive moderated. She accepted probationary status and was compliant with the rules of probation. She was no longer driven to prove herself as aggressive and sexually desirable.

Then suddenly, and almost as before, she again became aggressive, rebellious, and very sexually active. She again spoke about sexual matters in a poorly modulated manner. Her aggression became prominent, both in physical fighting with peers and general rebelliousness in school. She began courting neighborhood boys and keeping surreptitious rendezvous. She began to engage in more daredevil activities and to use coprolalic expressions during the treatment sessions. This behavior continued for a period of 4 weeks; then she admitted that she was not taking the prescribed doses of steroids. When she returned to regular steroid maintenance therapy, her behavior once again changed dramatically. Eventually, almost all sexual material disappeared from the clinical sessions. Even her repetitive dreams of a violent and usually sexual nature again abated. She remained on steroid therapy, graduated from high school, and married at age 19. There had been no recurrence of the behavioral difficulties.

The girl's unauthorized cessation of steroid therapy had caused the reappearance of the biological drives of aggression and hypersexuality. This variant of congenital adrenal hyperplasia, which is often milder and appears later, is also termed "postpubertal adrenal virilism." Grayzel (10) noted that clinical signs vary according to the age when excessive androgens affect the target organs. Acne, poor breast development, and low voice are more common than a large clitoris or pronounced hirsutism.

Case 2. This 16-year-old girl was seen by court order because of her possession of marijuana and stolen identity cards. She had earlier come to the court's attention due to her runaway and assaultive behavior. Her early childhood had been characterized by precocity, with high energy levels and excellent school performance. She preferred to play with boys and was agile and athletic. She had an IQ of 147 when she was 10 years of age. During our study this patient scored a full-scale IQ of 120 and appeared indifferent to higher achievement. In her words, "I used to hear how smart I was, that I could do anything I wanted . . . and I guess I rebelled against all of that and started to play the ignorant game." The girl had been persistently enuretic until her menarche at age 11. Her sexual adventures began in earnest at age 13. At age 14 she made a serious suicide attempt by lacerating her arm.

She had identified with "bikers" (motorcycle clubs)

and varieties of "freaks" and had been seriously burned during a fight with another girl who was vying for her position as "the old lady" to one of the "bikers." Shoplifting had become a way of life for her, although she had only been apprehended once. She had held a series of jobs in gas stations as a mechanic and customarily dressed in men's clothing (overalls, hat, boots). Starting at age 15 she was employed as a go-go dancer on the weekends. She freely admitted that she was bisexual, and her sexual identity was fluid and adaptable to the circumstances. In her last shoplifting venture she stole a nutcracker, a wrench set, and a timer (auto repair equipment) from a large store. Safely outside with this equipment, she returned to steal a hair curler, lipstick, and eye liner and was apprehended. When later asked what possible meaning was underscored by this shoplifting incident, she could only reply, "They're better set up for busting chicks in the makeup department."

Her hypersexual behavior took the form of marathons, frequently involving casual meetings. She occasionally had group sex and often had sex with men she met while hitchhiking. In one case, two men "worked me over with vibrators and oils." She was proud of her ability to have multiple orgasms. In her initial interview, when asked what she would like changed about herself, she replied, "A cock would come in handy." She had had several lesbian encounters but had no real preference in variety or form of sexual activity. This patient's manner of relating included large gestures with the hands, some pressured speech, and what appeared to be candor. She thought "as a man" and felt driven to be the pursuer, the aggressor, the initiator of sexual encounters. Her speech was filled with active vernacular expressions; she said she was the one who would "get it on . . . rip it off . . . blow his mind . . . or ball that chick." Basically, she saw herself as a misfit who had to conceal her intelligence and deny her need for security.

Her only medication was an oral contraceptive that she had taken since she was 15 years old. With therapy, there was some improvement in her delinquent behavior but little change in her sexual orientation.

Case 3. This 16-year-old girl was seen at the direction of the court because of a history of running away and incorrigible behavior. The girl's mother had been given injections of "hormones" during the first trimester of pregnancy to combat the threat of miscarriage. In addition, as a 12-month-old infant the girl was given "hormone shots" because of her small size; after that, she was said to have grown rapidly. Both series of hormones were administered in Europe. In addition to being of small stature in early childhood, she was quite hirsute and required extensive electrolysis for cosmetic purposes. She was restricted and very overprotected by her parents. During puberty she engaged in frequent masturbation, which was psychically painful but irresistible. Menarche at age 11 was uneventful but was presaged by full breast development and hirsutism from the age of 9.

Dating began as surreptitious adventures when she was 12. She saw older boys, and at age 14 began to run away with men in their twenties. She regarded sexuality as repugnant and anxiety provoking, but also as an irresistible force. She had a repetitive dream of being shot by different men. She became pregnant at age 15 and underwent a therapeutic abortion. This pregnancy resulted from a sexual liaison that went beyond her control; her abiding anxiety was "going out of control." When she first began working, her greater exposure to men led to increased sexual activity. She diverted most of her energy to juggling dates, and when she could no longer hide her activities from her father, she would run away with one of her boyfriends. These runaways led to police and court involvement.

Her affect was characterized by mood swings, with depression usually following sexual relations. She was overly verbal, highly energetic, slept poorly, and maintained stable weight despite eating excessively. With therapy, the patient's social behavior became moderated; she became engaged and was married at age 18.

Case 4. This small, chunky girl was first seen at age 16 when she was a senior in high school. She was the only child of professional parents and had been born after two miscarriages. Her mother had received progestational agents because of early signs of miscarriage during the first trimester of pregnancy. The girl's childhood had been marked by intellectual and sexual precocity. She entered first grade at age 5 and skipped the second grade. She was a prodigious early reader and was interested in solitary pursuits rather than peer companionship. Her mother recalled that the girl had masturbated frequently during early childhood. Onset of menses, at age 9, was uneventful.

Furtive running away and sexual intercourse began at age 12. The girl was musically talented and used music practice rooms in a conservatory for numerous rendezvous with boys. She entered a private school at age 13 and began liaisons with male teachers. She was moderately hirsute and underwent electrolysis at age 14. She was expelled from the first private school for stealing examination questions. Running away became more frequent at age 15; at that time her use of tranquilizers brought her to the attention of the court. When she was 15 she was simultaneously involved with men who were aged 22, 35, and 44. She was an avid reader of science fiction and attended conventions of science fiction buffs, where she formed more sexual liaisons. At 15 she started using oral contraceptives and also had her first homosexual relationship, finding it "interesting but not satisfying." She was expelled from a second private school for selling drugs. She kept a small pharmacy of medications for herself and was particularly concerned about having antibiotics on hand to take after sexual relations to prevent venereal disease. She ran away shortly after her mother's hospital-

ization for depression and was apprehended by police while hitchhiking.

In therapy she was restless and paced about the room. Her speech was studiedly profane. She challenged psychiatric theory and spoke of having read textbooks on abnormal psychology. During therapy she did attempt to cut down on her use of drugs. However, her hypersexuality continued, especially when nocturnal masturbation no longer helped her get to sleep.

Case 5. This 15-year-old girl, the older of two children in an intact family, was referred because of her continuing incorrigibility. Her mother's pregnancy was marked by repeated threats of miscarriage, and after bed rest and sedation, progestins were prescribed. As an infant, this girl was described as "totally active—always on the go." She had a strong masculine identification and was very jealous and demanding after her brother was born. She continued to out-run, out-swim, and overpower the brother, even when he was 14 and she 15. Menarche, at age 12, was uneventful.

She started stealing and lying at the age of 11. At 13, in the seventh grade, she began meeting older boys for sexual adventures. She began to roam farther from home, ran away, and became a burglar's accomplice.

In therapy she remained manipulative and deceptive about her sexual encounters but her stealing and conjoint burglary ventures became less frequent. She was quite muscular and prided herself on being able to "punch like a man." She was involved in several fights in bars. She enjoyed playing football, diving, and gymnastics, and rebelled against wearing clothing other than shirts and blue jeans. She was impatient with studies, usually restless in a classroom, and often took drugs to "relax." After she had terminated probation and therapy she confided that she had settled down sexually at age 18 and was living with two lovers who were brothers. "One is never enough, but I don't have to run out to find it the way I used to."

Case 6. This 14-year-old girl was the oldest of three children; she had two younger brothers. Although her parents' marriage was intact, her mother had been episodically depressed, requiring hospitalization when this patient was 9 years old. Her pregnancy and delivery were said to be uneventful. She had been a precocious child, walking at 7 months and talking in sentences at 1 year. She had a history of glomerulonephritis and had been hospitalized for 1 month and had received steroid injections at age 7. Menarche occurred at age 11 and was followed by stealing and meeting boys on the sly.

Her involvement with the juvenile court began at age 12 after she ran away many times. At age 13, using forged identity, she accompanied an 18-year-old man on a number of journeys and eventually married him. She was apprehended and brought to a detention home where a psychiatric examination was ordered. She escaped a number of times from relatively secure detention facilities.

In early psychiatric interviews she alternated between noncompliance with withdrawal and angry depression. When seen 3 years later she was extremely verbal and quick moving. She had been assaultive on occasion and made it her business to become a leader of other young female prisoners whenever she was incarcerated. She made several suicide attempts by cutting herself and taking an overdose of drugs. She was proud to be a tomboy but admitted to enormous sexual needs. Thioridazine (prescribed during two incarcerations) had blunted her sexual drive. She ran away many times for the express purpose of sexual adventure and noted "there's no high that can beat getting it on." She enjoyed sex and did not care whether she knew her partners, whether they cared for her or she for them, or whether liaisons were heterosexual or homosexual. What depressed her was confinement and the restriction of motor activity. She maintained contact with her childhood mate after the marriage was annulled but became attached to another, older man who became her primary interest. At age 18 she was single and maintained liaisons with one man and one woman. At the same time she was having sexual relations with others several times a week.

Case 7. This 17-year-old girl was seen in psychiatric consultation for the chief complaints of depression and shoplifting. Her mother had received a series of injections in midpregnancy due to the threat of miscarriage. Her family was intact; she was the youngest of three girls. She described a relatively happy early childhood during which she played as "one of the boys" and often recalled street games with boys as "carefree days." This happy period ended with precocious development and menarche at age 8.

Her classmates in grade school were "in awe" of her, and although she recalled being told about sexual matters early, she thought she was "bleeding to death" with her first menses. She was not treated for possible sexual precocity, but at age 15 she was discovered to have a benign thyroid goiter with nodules. She was treated with whole grain thyroid hormone, usually from 2 to 4 grains daily. She had been masturbating "as long as I can remember." She was intermittently enuretic from age 11 to 13. Her first sexual relations, at age 10, were with a girlfriend who slept over; this was followed by other similar encounters. At age 14 she attempted to hang herself with a blanket and was hospitalized briefly. She had repetitive dreams of being chased by monsters. Her first intercourse was at age 15 because "it was high time to get broken in." She spoke of numerous sexual encounters in a bragging fashion, with persistent cynical remarks about the inadequacies of her partners. She often would "wear out" male partners and alternated male and female lovers. She often masturbated as frequently as eight times daily. She used pseudonyms and ran away for "thrills."

Her court involvement began at age 15 after her apprehension for shoplifting. She had a pregnancy terminated by abortion at age 16 and was referred for psychiatric consultation by the court at age 17 after another series of shoplifting incidents. She regarded her hypersexual behavior as "tension relieving" and was "proud of my appetite." Her depression alternated with the more usual hypomanic energy and nonstop recounting of her many adventures. Shoplifting provided her with only another variety of thrill. She was bright and creative and managed to curb her shoplifting while on probation. When she turned 18, she left home to pursue a modeling career.

DISCUSSION

These seven girls shared several characteristics. All of them were of above-average intelligence, with an average IQ of 122. Their age at menarche was usually quite young, most of them were hirsute, four of them had low voices, and they tended toward mesomorphic build. Three of the girls had marked acne when I saw them. Their breast size was perhaps smaller than average. None of the girls had frank gynecological evidence of clitoral hypertrophy or other evidence of possible hermaphroditism. The mothers of four of the girls had received hormones during pregnancy because of the threat of miscarriage. Two of the remaining three girls had a history of excessive levels of adrenal hormones, one endogenous and one exogenous. The exposure to potentially virilizing hormones forms the common denominator of the group, with only one exception.

In 1958 Wilkins and associates (11) first described the masculinization of the female fetus associated with the ingestion of progestins during pregnancy. From 1950 to 1975 progestin therapy was commonly used in cases of threatened miscarriage and also for pregnant diabetic women. Only recently has the Food and Drug Administration (12) warned of its poor efficacy and problems with its aftereffects. Following up on a study of IQ and psychosexual identity in progestin-affected girls, Ehrhardt and Money (13) found significantly higher IQs and tomboy behavior. One of their positive findings was the girls' great interest and participation in muscular exercise and expenditure of energy. In a later study, Ehrhardt (14) found that girls with fetal masculinization had a significantly higher interest in a career and a lower interest in the traditional maternal role. These girls preferred boys' playtoys and had little interest in dolls.

A Russian study (15) of women who were treated later in life for a virilizing adrenogenital syndrome found that they had a significantly higher than average general educational level. This study, which employed anamnestic interviews, found elevated sexuality in these women, with increased libido and a high frequency of sexual dreams and masturbation. Discussing innate masculine-feminine differences, Gadpaille (16) described girls who were reared as females and exposed to the cultural stereotypes of femininity but were still masculine in their preferences. Conversely, Yalom and associates (17) have shown that prenatal exposure to an estrogen-progesterone combination has a demonstrable effect on the masculinity of boys and has been shown to reduce expected levels of aggression, assertiveness, and athletic ability.

When we turn toward a summary of the behavior of the seven girls whose delinquency was associated with hypersexuality, we note that there was a high incidence of running away and serious suicide attempts. Several of the girls were quite aggressive and assaultive, and stealing was a common antisocial activity. Clinically, all of the girls had definite signs of hypomanic ideation. High energy levels, rapid and overinclusive speech, and labile mood swings were characteristic of the group. None of the girls was frankly psychotic.

It is not possible to conclude from this small group which cultural, psychodynamic, and biological factors are critical. It is postulated that girls who have been exposed to virilizing hormones in utero or later in childhood can be characterized by hypersexuality, hypomania, high intelligence, and aggressiveness. Some of the delinquent manifestations of these girls and young women are seen as extensions of their "psychovirilization." These girls were therefore delinquent by default and as a consequence of their hypersexuality.

Psychotherapy combined with legal probation had some effect on the delinquent behavior but was less effective in reducing hypersexual activity. Physicians should be on the alert for young men and women born in the 1950s and 1960s who may have been exposed to feminizing or virilizing agents. Perhaps in the future some of the clinical problems of these at-risk patients who come to the psychiatrist's attention can be ameliorated by hormonal therapy, combined with the psychotherapist's appreciation of the role of these neuroendocrinological factors in certain behavioral manifestations. Hypersexuality may then be conceived of in terms of a biological drive that may have been primed by exposure to virilizing agents and is expressed in the force field of psychodynamic considerations. The association of hypersexuality with delinquency in these adolescent girls is thought to represent a defect of behavior engendered by the biological priming.

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A General Systems Approach to Severe Family Pathology

BY STEPHEN FLECK, M.D.

The author proposes a general systems approach to the examination and understanding of family functioning. Scrutinizing the family as an open system with 1) evolutionary goals and tangible tasks, 2) a need for "semipermeable" boundaries, 3) cultural and subcultural communication modes, 4) leadership effectiveness, and 5) the nature and age-appropriateness of the affective bonds will enable clinical judgments about family functioning that indicate general system competencies and defects. Such a framework may also lead to a typology of family process because the major functions of the family as a system (creation, nurturance, enculturation, and guidance of the young into adulthood) can be evaluated regardless of symptoms or diagnoses.

TOLSTOY'S *Anna Karenina* begins with the statement that all happy families are similar and that all unhappy families are unhappy in their own fashion (1). A general systems approach to family study and assessment may help, not to disprove Tolstoy (because family clinicians must agree with him) but to bring some measure of order into the chaotic world of family pathology. The clinical participant-observer needs a superordinate framework and referential system to categorize and order his observations of family behavior. Like other systems the family can be examined and evaluated on five criteria: 1) its central functions and goals, 2) its leadership, 3) the regulation of the boundaries among system components and between the group and the outside, 4) managerial links and affective bondages, and 5) the effectiveness and relevancy of communication inside the system and between it and the outside world (2–4).

These five system constituents and forces are distinguishable in any functioning complex but are also interdependent, and malfunction of one sector affects all others. For instance, leadership declines if there is communication disturbance, and boundary diffusion and affective imbalances can interfere with task pursuit. It is the circularity of these attributes and forces that must be appreciated, as well as the cumulative effects of dysfunction or deficiencies. One defect passed on along an assembly line may render the end product unworkable, and developmental failure at a particular stage in the human organism can interfere with the emergence of an internally and socially integrated adult (5).

Biosocial and cultural givens require that the family humanize the young by teaching them not only about themselves and how to live within the family but also about the mores and cultural values of the society. In addition to these evolutionary goals of the family as the keystone of society and the aforementioned system functions, this group must also accomplish tangible and specific tasks related to self-sustenance.

The evolutionary tasks are as follows: effecting a marital coalition capable of forming a triad of parents and infant, nurturing and weaning the infant to enable it to exist as a body apart from the mother, and imparting language and intrafamilial behavior modes epito-

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Dr. Fleck is Professor of Psychiatry and Public Health, Department of Psychiatry, Yale University School of Medicine, 25 Park St., New Haven, Conn. 06519.

The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

mized by the resolution of the oedipal phase. Furthermore the family must introduce the child into society in school and through peer relationships, which provides familiarization with society's tools and values. Tools include formal learning, communication mastery, and competence in abstract and symbolic thinking and expression. After puberty the child turns again more toward relationship issues, establishing not only his/her sexual identity but also undertaking more lasting and intimate relationships with peers. The family system is involved in all these stages; malfunction may burden and handicap offspring severely, particularly in the process of emancipation from the family to become a full-fledged adult member of society (5–7).

As a system, however, the family is more than an evolutionary production line. Parents must not only function as gender typical adults in their society but also tend to the other system parameters. Boundaries must be managed so that within the group each member is afforded a sense of "I" as well as "we." Boundary is a difficult concept but is essential for a sense of integrity for the individual and the family. Boundary has two dimensions, time and space, each with tangible and intangible aspects. For instance, boundary management between the family and its community must be appropriate to subcultural norms (e.g., when a child enters school). Later, the child must cross the boundary comfortably for other purposes and for longer periods without losing the sense of family belonging, which must persist through life lest a person lose contact with his past. Another crucial boundary within the family is generational, distinguishing the parents as leaders and teachers and the children as followers and learners.

Affect flow in the family is another basic function, also determined by the parents' behavior and leadership. In brief, the maintenance over time of a positive balance of good feelings and mutual fondness is essential. The initial physical closeness between parents and child must be reduced so that a more abstract deeroticized sense of closeness evolves, with sexual intimacy limited to the parents. The resolution of conflicts and the modulation of feeling intensity through example are the leaders' tasks in maintaining a harmonious and functioning group (6).

Communication distinguishes human behavior from all other. Animals exchange signals and can even recognize feeling states in another being (8), but only humans can make feelings explicit, learn the history of their species, and mentally project future action through language-derived symbols and abstract ideation before implementing it. Linguistic competence in the family consonant with communication styles of the society is therefore as crucial to the evolvement of individual integrity as it is to effective social participation (9–11). Observing familial communication, therefore, is also a major examination tool of family functioning, just as dyadic interviews and participant observation constitute the psychotherapist's matrix for evaluating intrapersonal processes.

CLINICAL ILLUSTRATIONS

The following is an opening vignette from a meeting of a family and two hospital staff members:

Mother: It's nice to have such lovely weather. [It is pouring outside the window.]
Daughter: I am Nikita Khrushchev.
Father: [Shakes head mildly and lights a cigarette.]
Therapist: Daughter seems to be responding to mother's nonfactual statement.
Mother: It is raining, but how are you, doctor? You look well.
Father: We had a good visit.
Daughter: Can I have a cigarette?
Therapist: A good visit?
Father: Yes, of course we didn't go out but I had some good talks with daughter.
Therapist: Yes?
Daughter: [Leaves room.]

One need hardly be a skilled observer or student of family process to conclude that this is indeed a very disturbed group of people, leaving one guessing as to who might be the patient. It is also not difficult to draw inferences about the individual malfunctionings exhibited, through the use of the referential systems of psychoanalysis and psychodynamics. General systems principles also seem useful to categorize the behavior and dynamics of this unhappy group.

One can say that in one sense no communication was taking place—these people did not even greet each other and no one validated anyone else's statement or behavior. The comment about the weather is a classic opening gambit for strangers seeking to begin an agreeable discourse. In this vignette it was also an idiosyncratic distancing gambit; it sought no agreement because it contradicted the obvious. However, it was not lost as a stimulus, eliciting a crazy response from the daughter and a kind of nonverbal "let's ignore it" from the father. The father's first statement was equally inconsistent with his own attitudes and wishes. Another conventional gambit, "We had a good visit," is nice to say if one wants to elicit agreement, but it was nonfactual; indeed, the good visit he had wanted, even demanded, was that his daughter should be allowed to be with the family away from the hospital. His wish was not granted, and everyone in the room knew this.

Thus communication in this group was earmarked by misstatements about observable or known facts and mutual nonvalidation arousing a high level of anxiety and anger in the participants. After the daughter's departure, communication between the parents continued in a challenging and disagreeable manner, and neither seemed attentive to the other's intent or discomfort (11, 12). Communication could also be categorized as fragmenting with regard not only to meaning but to the individuals involved. This gave the family therapists at least an opportunity to try to reduce fragmentation, which in this instance did not suc-

ceed. But we can agree with Wynne and Singer (12) that this type and style of communicating is indeed damaging to and pathognomonic of at least one schizophrenic member in the family.

Family behavior can also be assessed in the framework of task performance—by immediate tasks in a therapy session and by examining longitudinally the general and basic evolutionary family task performance. In this instance the family had been informed in previous sessions that the task was to converse with each other, to improve contact with each other, and to understand their difficulties better. Task performance in the vignette was nil if not negativistic, arousing the observer's suspicion that the broader spectrum of evolutionary task achievements may have been equally deficient if not destructive. Indeed, the family history revealed that there was not one phase during which this triad functioned adequately or in a manner appropriate to the daughter's age and developmental needs.

The mother suffered recurrent illnesses requiring lengthy hospitalizations almost from the time of her daughter's birth; an effective mother-daughter relationship never developed because, even when physically present, the mother was rarely emotionally available to her daughter. There was no consistent mother substitute, this role having been filled by a succession of maids, two older sisters, and the father to a considerable extent. Yet it was not a typical single-parent family. The maternal behavior on the part of the father interfered with the emergence of any real oedipal situation, let alone any resolution, and this particular father's maternal attention to the daughter extended through puberty. During this period there was no real family unit, in part because of the wide age spread among the children but also because of the mother's recurrent but unacknowledged difficulties.

At about puberty the daughter began to show psychotic symptoms and behavior and became the identified patient for the next 6 or 7 years. On the other hand, the family had not identified the mother as a psychiatric patient and still referred to her illnesses as infections in spite of her having been on antipsychotic medications for years. In terms of evolutionary family tasks, the psychotic daughter never approached any emancipation phase and faltered in early adolescence, thus requiring psychiatric hospitalization. She has claimed to function on an age level of 4.

One can discern the extreme degree of failure in every phase of system goals, representing almost in caricature our formulation, advanced some 12 years ago, of schizophrenia as a deficiency disease of family functioning and task performance (6, 9, 13).

In this chaotic family, boundary management was equally pathological. In the session, the family members exhibited rigidly drawn noncontiguous boundaries, each withdrawn from the others in his idiosyncratic stance. However, we observed also extreme intrusiveness, a violation of ego and intergenerational boundaries in the form of inappropriate physical contact between father and daughter. She has also perceived her mother as being sexually interested in her.

Children are usually expected to negotiate family boundaries without undue discomfort when entering school, which presupposes some degree of separation mastery within the family. The children in this family were tutored at home because of the father's fear of his children suffering contamination and infections in school. Boundaries were overcontrolled and rigid instead of being permeable and transgressible in line with children's age-appropriate needs. The isolation of the identified patient almost from birth illustrates further boundary mismanagement, punctured by inconsistent intrusions almost literally into her body space.

The parental role reversal was blatant, and obvious role model defects in both parents constituted leadership failure evident even in this brief vignette. Considering this family from the standpoint of system integrity, we see a system that mismanages boundaries, is not led properly, and in which affective bondages and interactions (communication in particular) interfere with self-definition and task performance. Such a system can neither function nor achieve goals, whether these are givens or explicitly defined as in the family meeting.

A brief description of contact (or noncontact) with the family of another hospitalized patient may illustrate further boundary mismanagement and other system defects. According to the social worker's record, "This family met on common ground in their wish not to be involved with the hospital or in treatment. Family meetings were difficult for all of them, particularly for the mother and the son (the identified patient). They collaborated in trying to keep discussions as irrelevant as possible and had a variety of maneuvers to avoid engaging themselves. The father used a style of legal interrogation with his son and was apt to get into lengthy debates about the details of an event. He also claimed total ignorance of anything having to do with emotions, interpersonal relations, or treatment. He became quite irritated with his wife when she occasionally revealed that he had been angry with his son, with the social worker, or with the way a family meeting had gone, but usually denied having said any such thing."

The mother's tactics included attempts to discuss the social worker's personal life, other patients, current events, and psychological theories. When the session became more personal, she tried to force her son to confess that he had a happy childhood; she was quite angry at the hospital staff for stimulating him to think about his life and his feelings. As she became more disturbed and vulnerable, her intense conflict between her feelings of hostility and rejection toward the patient and her enormous guilt about these feelings were increasingly apparent.

The identified patient was the second child and only son in a family of four and was clearly not wanted by the mother. Her then 3-year-old daughter already was in a day nursery when the second pregnancy occurred.

She had at this point turned her interest and energy toward various community activities and was also very much involved with another man. Shortly after being sent to nursery school at age 3, the son developed poliomyelitis, from which he eventually recovered completely. Because his prolonged illness had produced regression and developmental stagnation, he was not even ready for nursery school by the time he was 4; his mother, however, ignored such messages from teachers and insisted on continuing with the out-of-home program so that she could pursue and nurture her community interests and ties, which interfered with the family's needs at the time and with family life. In this instance, boundary management failed because it was not congruent with this child's emotional needs or with engendering a sense of family unity and bondage.

Movements of the young across the family boundaries are predicated on separation mastery and on the child's inner certainty that a person out of sight is not lost ("object constancy"), neither of which can occur in the context of pretense or sham, which characterized the parents' affective and communicative behavior with each other and with their children. The son's psychosocial immaturity, evidenced by separation difficulties, was ignored.

This boundary mismanagement is different from that of the first example. In both families, contact is avoided so that there is little affect flow, and everyone remains encapsulated. However, in the first family distance alternates with intrusive excursion into each other's affective space, and encapsulation, even autism, is achieved through symbolic distortion and idiosyncratic, nonvalidating communication (7, 12). In the second family, communication sounds and seems rational but is not truthful or expressive. Feelings are avoided and denied but not distorted, projected, and/or fantasized. The second family spawned a psychopath, the identified patient, who became a master in the family's instrumental, manipulative, and nonexpressive communication style. "Manipulative communication" means that the speaker focuses on whatever reaction he wants to elicit in the listener, whether the intent is pleasing, duping, or angering the other.

In this family, with a very disturbed, intermittently depressed, but mostly sociopathic patient, more than one essential systems paradigm was violated. Boundaries were mismanaged intrafamilially by imposing emotional distance and extrafamilially through pushing the younger members beyond the family sphere before adequate individuation and separation mastery had been achieved. The generation boundary, although too wide, was not violated; however, nurturant tasks were accomplished marginally at best. Goal pursuit in general was inadequate and deceitful, both in the therapy setting and in the life of the family; for instance, no attention was paid to the patient's misconduct and deceitfulness unless evidence was furnished from outside by schools or police.

Finally, emancipation was never joined as an issue at the usual age because of the antecedent evolutionary task failures in mastering separation, although the patient was physically out of the home in various boarding schools from early adolescence. Yet his misbehavior, leading to repeated expulsions, was never acknowledged as such, let alone as a veiled message of his unpreparedness to live without parental care.

There was a good deal of verbal emphasis on love, but both the record and the session behavior were replete with indications of mutual rejection and hostility between the spouses and between mother and son. Communication in sessions was often disputatious, especially when unacceptable affects had to be denied. Whereas interparental communication was superficial, rational, and seemingly logical, it was neither affective nor truly responsive, and the patient often talked in fragmentary fashion like a child unready to think in sentences. Communication was substantively deficient in that it lacked expressiveness, and affective exchange was not taught or practiced; instead, communication was manipulative. Leadership was perfunctory and stereotypic but not grossly aberrant. It was deceitful because of the emphasis on appearances; i.e., whatever looks all right is all right. Thus we find communication and leadership marginally acceptable but affective forces, boundary regulation, and some family tasks grossly deficient.

DISCUSSION

I have attempted to demonstrate briefly one method of how to order initially confusing, disturbed, and disturbing family behaviors. These two examples derive from very aberrant families, each with at least one member in need of psychiatric hospitalization.

The information obtained both from direct participant observation and from historical exploration can be ordered by examining the basic characteristics of any general system—task and goal pursuits, leadership, boundary regulation, affective bondages, and effective communication. Since the central mission of the modern nuclear family is the nurturance, enculturation, and guidance of the younger generation into adulthood, the history must be relied on for the most part to provide relevant data (6, 9, 13, 14). However, leadership, communication, boundary management, and the affective bonds are also observable and available for inferences from behavior in family meetings (7, 11, 15). These procedures have only been indicated here and should be described in greater detail in the future.

It can be pointed out that this approach may serve not only to conceptualize family functioning and malfunctioning along two axes, historical and cross-sectional, but also to establish a family typology, specifying normal and abnormal system functioning and performance according to the aforementioned principal system paradigms.

Families as severely disturbed as those I have described demonstrate multiple (and in one instance

global) system deficiencies; many more families have system deficiencies that are more discrete and do not affect all parameters of system integrity and all evolutionary stages of family task performances (7, 12, 16).

As far as schizophrenia is concerned, this illustration and like considerations confirm our earlier thesis of pervasive family system deficiencies in the background of young schizophrenic patients (13). In the first example the parents were severely disturbed, rendering the formation of an effective parental coalition and leadership impossible. The parent-patient triad seems deficient in all evolutionary phases, and there are boundary violations of personal space and defects in family-community relationships. Emotional bonds are inconsistent, probably incestuous in quality, and anxiety laden. Communication was ineffective in both families—bizarre, fragmented, and fragmenting to the speakers in the first and amorphous and dissimulating in the second (12, 13, 16).

The second family as a system was different from the first. Although both parents could be considered neurotic (the mother suffered various psychosomatic symptoms, the father showed obsessive-compulsive traits), neither was grossly disturbed, and they effected a workable albeit superficial marital coalition. However, the coalition did not extend to the parental tasks, and leadership and affective assertions in the family were a sham. Crises and family failures were denied instead of coped with, and boundaries were kept noncontiguous within the group and were almost nonexistent between the family and the outside. Communication was earmarked by its exclusively instrumental usage (an emphasis on its sounding right whether or not it was true and meaningful). The sham behavior extended to the family's task performances—there was nurturance without warmth, enculturation without substance (the principle that as long as behavior looks all right, it is all right). There was no family togetherness in leisure or crisis, and emancipation was also a sham in that the patient's signals of his inability to cope with separations were ignored.

CONCLUSIONS

The general systems approach of defining a family typology seems useful in ordering family behavior data and may also correlate with certain of the major clinical entities, a prospect reinforced by other family records so analyzed. This correlation may be more quantitative or cumulative than qualitative. The data so far suggest that the number and severity of systems defects may distinguish pathological outcomes rather than any particular intrinsic defect.

In summary, scrutinizing families as an open system allows us to arrive at sequential judgments and categorizations that differ from such simple polarities as normal versus abnormal or healthy versus pathogenic. General systems theory permits cross-sectional and longitudinal examination of the group not only in terms of the special group that is the family but also as it pertains to any collective of interacting forces. Judgments about the system's behavior and past performance in terms of the family's role as the keystone of society can then be made independent of presenting disturbances, symptoms, or traditional clinical classifications.

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The Fatal Myth of Death in the Family

BY E. MANSELL PATTISON, M.D.

The author hypothesizes that the observed pathogenic effects on a child of the death of a parent are the result of the family's culture-bound inability to integrate death as a natural part of the process of living. The family tries to deal with death by the avoidance mechanisms of myth and family mystification; it is this process which is pathogenic rather than the experience with death itself. To illustrate his thesis he presents the case history of a man who was fatally affected by the family myth and mystification process.

THIS PAPER EXPLORES the function of the family as a psychosocial system in the management of experiences with death. In particular, I shall examine how the family system copes with the death of a parent.

Psychiatric opinion is divided on the questions of how traumatic the death of a parent is to a child, how irreversible the effects of such a loss are, and how these effects may be meliorated for the child. At the pessimistic pole there are those who view the death of a parent as a major irreversible trauma. Moriarty (1), for example, stated, "Those who suffer from exposure to death in early childhood may be haunted for the rest of their lives by these memories" (p. 120).

A more mediating position holds that experiences with death are a severe childhood stress but not necessarily a dire trauma. For example, Laufer (2) stated, "While itself not pathogenic, object loss can become the nucleus around which earlier conflicts and the latent pathogenic elements are organized" (p. 290). Similarly, Furman (3) stated,

> When a child's parent dies, each surviving member of the family faces so complex and difficult a situation that no form of assistance may seem adequate to the task. Yet, many children and parents . . . can master the stress. (p. 296)

Finally, the nonpathological view holds that death has become pathogenic because sociocultural taboos prevent child and family from appropriately coping with death (4). In Victorian times cultural denials and taboos centered on sexuality; therefore, sexual experience generated psychopathology. Today cultural denial and taboos that center on death result in death's generating psychopathology (5).

The following constitute the basic theses of my paper: 1) much of the observed psychopathology of experiences with death during childhood results from a sociocultural failure to appropriately integrate death as part of natural life experience; 2) the family system incorporates and embodies this cultural denial of death; 3) the family management of death is dysfunctional for both the family and the child because it fails to appropriately integrate death within the family; 4) the family deals with death through the avoidance mechanisms of family myth and family mystification; and 5) the family myth and mystification processes are the pathogenic elements in the death of a family member rather than the death per se.

CASE STUDY

Harry, a 40-year-old physician, came for psychotherapy with the chief complaint of alcoholism that had started four years earlier after the sudden death of his wife Florence. She had had undiagnosed leukemia that erupted in fulminating symptoms and sudden death following four days of illness. Harry expressed profound and unrelenting guilt for her death, which he tried to drown in alcohol. He stated that his marriage to Florence had been idyllic: she had been the perfect woman for him as well as acclaimed as the epitome of femininity and motherhood by all who knew her.

After three months of therapy, Harry's intense guilt was relieved, his reactive depression was resolved, and his drinking was controlled. He was able to return to his medical practice and function at a socially competent level. An intelligent, articulate, psychologically sophisticated, and introspective man, he recognized that his neurotic grief reaction and alcoholism were the symptoms of long-standing life conflicts. The next period of psychotherapy revealed Harry's history.

Harry was an orphan who had no knowledge of his biological parents because he had been left on the doorstep of two unmarried sisters as an infant. These two women, Aunt Millie and Aunt Mary, reared him to adolescence. Harry perceived the younger sister, Aunt Millie, as kind, sweet, and gentle; he lived with her until he started kindergarten. Then, for reasons unclear to him then and when I knew him, he had been moved to live with the older sister, Aunt Mary, whom he perceived as stern, severe, and businesslike. He mourned his separation from Aunt Millie, who had been like a mother to him. Although he saw Aunt Millie on holidays, he was nev-

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Dr. Pattison is Associate Professor and Vice-Chairman, Department of Psychiatry and Human Behavior, University of California, Irvine, and Deputy Director of Training, Consultation, and Education, Orange County Department of Mental Health, Orange, Calif. Address reprint requests to him at the Department of Psychiatry, Orange County Medical Center, 101 City Drive South, Orange, Calif. 92668.

er again allowed to live with her. During therapy, Harry expressed affection for both women, and his visits with them several times a year constituted family reunions for him.

Aunt Millie had had a boyfriend for many years, Mr. Brown. Mr. Brown had always taken a kind, fatherly interest in Harry and had even provided some financial support for him. Harry was told that Aunt Millie and Mr. Brown planned to get married after the death of Mr. Brown's sickly wife and that they would then legally adopt him; Harry would then inherit Mr. Brown's business. However, when Harry was 14 Mr. Brown rather than his sickly wife died. After that it seemed to Harry that he was even less welcome in Aunt Mary's home. He felt dejected, rejected, and alone, so he ran away. He lived in a series of friends' homes until he could work full-time and live alone. An able student with a winsome personality, he worked his way through college and medical school with the help of scholarships. He met his wife Florence in college, she helped him through medical school, they had three children, and he embarked on a medical career that was successful for 10 years—until the sudden death of Florence.

At this point it became clear that Harry had not developed his severe guilt, depression, and alcoholism immediately after the death of Florence. After an initial period of mourning he seemed to have made a good adjustment. Within six months he married his second wife, Sarah. It was after this second marriage that his disturbing emotions and symptoms began to develop. Because the second marriage was happy, mutually rewarding, and successful, Harry could not account for his increasing inability to function.

Sarah had been Florence's roommate in college. The two women presented contrasting personalities. Florence was kind, sweet, gentle, and unassuming, a contented and loving wife and mother. Sarah, who had never married, was rather brusque, businesslike, and capable. She had pursued a successful career as a psychiatric social worker and had continued to remain friends with Florence and Harry during the years. As a matter of fact, Harry and Sarah had had intermittent sexual liaisons during the 15 years of his first marriage. Neither considered this a threat to his or her relationship with Florence, who ostensibly was unaware of the liaisons. After Florence's death Sarah immediately volunteered to help with the children; shortly thereafter she and Harry were married.

During this period in therapy Harry continued to function well in his medical practice, his social life, and his marriage; he also remained abstinent from alcohol. However, as the history of his life unfolded, he began to experience new and disturbing affects. He reported a sense of foreboding, as if his existence were to be snuffed out, and an inchoate stirring in his stomach, as if his insides were being wrenched. Then he told of a sense of panic and dark anxiety, as if he must jump and run. He was unable to locate himself and felt bewilderment, confusion, and diffuse ennui. This amorphous state of profound panic was more than he could endure. When he remembered that he had felt this way when he left Aunt Millie he shuddered. He felt this way when Mr. Brown died. He felt this way after his first marriage. He felt this way when Florence died. However, he had endured all of these crises.

The only times he felt he could endure now—the only times when he could emerge from this engulfing lostness—was when he talked to me; there did not seem to be anyone else who could really be with him. He felt that his wives were an attempt to create a mother, but they were not his mother. His aunts were an attempt to create a mother, but they were not his mother.

"Where, oh where is my mother?" he sobbed. "If my mother is dead then let her be dead. But maybe she is alive. Sometimes I think Aunt Millie is my mother, and I think Mr. Brown was my dad. I know it's not true, but I wish it were so. It wouldn't have to make any difference to them. But just to know that I came from somewhere, that I belong to somebody, that I'm not just a nothing. God! How can I be? I'm nothing, nothing, nothing. Mother, mother, mother, where are you? Dead? All right. Give me your grave. Let me put flowers on it. But be, damn it, be! You know, sometimes I know Millie is my mother. I don't want anything from her. I just want to know. I have to have a mother! God, oh God, have mercy on me!"

The next day Harry went to see Aunt Millie and Aunt Mary. He was armed with the resolute conviction that they held some knowledge of his parentage. He returned crestfallen to report that there was nothing to report. Aunt Millie and Aunt Mary said they knew nothing about his origins and that he was certainly not the son of Millie and Mr. Brown.

At 2:00 a.m. the next morning Sarah called me: Harry was dead.

Harry's epitaph is not a night of whiskey drinking that failed to drown out his senses and then the need for a few barbiturates to bring on sleep. Nor is it the will that Harry left: he requested that his ashes be strewn over the sea. Rather, it is a prosaic bit of historical fact. Two weeks after Harry's death, Sarah went to visit Aunt Millie and Aunt Mary to offer her condolences. Shaken by Harry's death, they said they could finally say what could not be said while he was alive. Aunt Millie was his mother, and Mr. Brown was his father. To protect Millie's family as well as Mr. Brown's, Millie, Mary, and Mr. Brown had made a pact that Harry should never be told. When Millie and Mr. Brown had resumed their relationship they had protected themselves from Harry by shipping him off to Aunt Mary.

Harry was right all along. He knew he could not know what was known.

THE PSYCHODYNAMICS OF DEATH IN THE FAMILY

The story of Harry is the story of his several families. To begin with, Harry was never provided an unambiguous mother object. He was first reared by two sisters, neither of whom assumed primary responsibility. Although he lived with Millie, she did not claim him as her own child, as a foster child, or as an adopted child. The basic ambivalence of the infant toward the mother was reinforced by this ambiguous status. The splitting of the mother object into "good mother" and "bad mother" was reinforced by the presence of two mother objects, one of whom assumed a good, accepting role while the other assumed a bad, rejecting role. Harry was given no opportunity to resolve his ambivalence and to attain a constant mother object. He had no basic nurturant mother object to internalize and use to build his constant object self.

A second step in object identification was also blocked. Harry was offered a tentative identification with Mr. Brown. However, when Mr. Brown reentered the scene Harry was just at the onset of the oedipal stage. The desexualized good mother object, Aunt Millie, was transformed into a sexualized wife with

Mr. Brown. Harry was not offered identification with Mr. Brown or allowed to retain a desexualized relationship with his good mother. Rather, he was sent away to his bad mother, Aunt Mary. He was not told why. Bad mother was better than no mother at all at this point, and Harry was still given the hope of adoption by Mr. Brown and the inheritance of his business. The opportunity for identification with Mr. Brown made life with bad mother tolerable, but Mr. Brown died, and with that death the tentative identification with a father was lost. Harry could no longer tolerate life with bad mother, so his only alternative was to run away.

As Harry reached adulthood he recapitulated his search for a mother. He married a desexualized good mother object, but the sexual bad mother was present from the beginning of the courtship and throughout the first marriage. Eventually, he was left with the bad mother. Just as the two aunts had colluded in their mother role, so the two roommates seem to have colluded in the wife role. The death of the first wife evoked bewilderment and guilt, just as the loss of the first mother had evoked bewilderment. Florence's sudden and mysterious leukemia was like the unexplained loss of Aunt Millie.

It is noteworthy that Harry's depressive and alcoholic symptoms emerged only after he married Sarah. In this case, Harry again found temporary hope in identification with a male father object—the psychotherapist. The threat from the bad mother (his second wife) was attenuated, and Harry was able to attain symptom relief. The psychodynamic analysis in therapy uncovered the projections into his current life, thus releasing his wives and myself at least partially from the transference roles. On the other hand, this placed Harry right back at the earliest stage of object relations: the search for a mother object and a profound primordial panic. He could not be a person without a mother object on which to ground his sense of self. A dead mother would release him from the search and allow him to recathect living objects, a rejecting mother would give him a reality to work with, but the third alternative was unbearable—a mother who gave cues recognizable in preconscious form that declared he was her son and verbal cues that declared he was not.

The family myth declared that he had no known parent, but the mystification of the myth subtly implied that the myth was myth. Psychotherapy dispelled the clouds of mystification. Harry attacked the myth. When it was thrown back into his face, he knew he had lost: he could not have the truth about himself. If your mother does not acknowledge your existence, how then do you exist? Harry could not.[1]

[1]I acknowledge that this cursory case summary may appear to provide too pat an analysis of the object identifications, transferences, and dynamics in the interest of stressing formulations pertinent to the theses of this paper. I am also aware that continued exploration in psychotherapy may have stirred up previously repressed material leading to Harry's suicide. Mea culpa. Becker (6) touched on this dilemma for all therapists: he said we should have a sign on our door that says, "Danger, real probability of the awakening of terror and dread, from which there is no turning back" (p. 271).

OBJECT CONSTANCY AND LOSS

The family matrix provides the basic critical objects for the infant. The young child must have constant objects to accept, love, and nurture him. This matrix of constant love objects provides the young child with a sense of identity (7, 8).

A major task of the family is to provide the young child with constant objects of nurturance. The loss of a parent object can be tolerated only if the family provides unambiguous definitions of the loss as well as unambiguous object replacements. (In the case of Harry there was both ambiguous definition of the loss and ambiguous replacement of the parental object.)

Schoenberg and associates (9) pointed out that loss per se is not the psychological issue but the effectiveness of the process of working through the grief. Failure to deal with psychological separations and losses in the family may lead to subsequent family pathology (10). Paul (11), for example, found family pathology not only in loss by death but in the failure to deal with the loss of the primary parent object. When children fail to resolve their symbiotic dependence on the family matrix of objects and then fail to work through the grief of loss of the symbiotic dependence, they fail to achieve mature individuation.

The process of maturation requires a movement from object constancy provided by a loving, accepting parent object to object constancy based on the internalized parent object and transformed into the self. This is necessary in order to love oneself and maintain one's own object constancy and can occur only through the gradual severance of the symbiotic object relationship; the parent must reject the dependence of the child (8).

The child learns to love himself because of rejection by the symbiotic parent object. The child must suffer this loss to recathect himself. It seems to me that the family management of parental loss is critical to the family nurturance of both self-individuation and the management of the physical loss of death. Thus the family management of death is grounded in the family management of object separation and object loss.

We may consider object separation and loss as necessary growth and individuation experiences. Heretofore we have considered object loss as an undesirable stress, whereas it may be a necessary precursor to the development of a self object. Hofling and Joy (12) reported favorable growth consequences of loss.

My first reinterpretation of death is, therefore, that object separation and object loss are necessary growth experiences that provide the anlage for later successful management of experiences with death. This thesis is consonant with the theoretical work on object splitting by Fairbairn (13). The exciting-frustrating symbiotic object can be split by the child into good object and bad object. When the child is unable to reconcile this ambivalence, he may retain the good-bad internal object split. Indeed, people may exist in reality as counterparts of the internal dichotomy, and a person may

continue to make active unconscious attempts to force and change close relationships to fit the internal role models.

If the child does not resolve the basic object ambivalance and achieve separation and autonomy apart from the symbiotic object parent, he may continue into adulthood striving to prevent the loss of the ambivalent symbiotic object. Framo (7) summarized the dynamic involved here nicely:

> Projective transferences, externalization, vindictive fantasies, vicarious participations, all serve the function of recapturing the symbolically retained old love objects who have their representation in current real family members, thus delaying the pain of loss and mourning. Object possession . . . helps prevent individuation which can result in the catastrophe of separation, the old dread of abandonment, and facing of the fact that one has irretrievably lost one's mother or father. (p. 135)

Thus observations based on ego object theory support the contention that loss of the parent is necessary to individuation and that physical death of the parent may well be considered a variant in the normal process of loss of the parent that every family must work through.

I suggest a second reinterpretation: the concept that the physical death of a parent is a pathological trauma is a myth that precludes awareness of the necessary and inevitable loss of the infantile symbiotic parent object. (In the case of Harry, there was no opportunity to work through the ambivalent symbiotic relationship with his two wives. Harry could not grieve the loss of the mother object because there was no defined object to lose. He could not achieve individuation because he did not have a concrete mother object to internalize and then separate from.)

THE ROLE OF MYTH AND MYSTIFICATION

It is clear from family therapy experience that family psychopathology often centers around family myths (14–17). Ferreira (18) defined the family myth as

> a series of well-integrated beliefs, myth-like, which members of a family entertain about each other and their relationship. . . . *The family myth is to the family what the defense is to the individual. . . . The family myth is a group defense against disturbances of changes in the relationship*. (p. 187)

The family myth is a neurotic mechanism for family coping and homeostasis in the face of unresolvable conflict. The process of mystification is the secondary mechanism that maintains the myth. Mystification, as Laing (19) explained it,

> is to befuddle, cloud, obscure, mask whatever is going on, whether this be experience, action, or process. . . . The state of mystification . . . is . . . a feeling of being muddled or confused. (p. 344)

Mystification was described by Wynne and associates (20) as "pseudomutuality" and by Searles (21) as a mode of "driving the other person crazy" by techniques that tend to "undermine the other person's confidence in his own emotional reactions and his own sense of reality."

Elsewhere (4) I have documented the evidence that our culture has developed enormous myths about death and that as a culture we engage in massive mystification mechanisms about our myths of death. I have selected the case of Harry to present because it so clearly demonstrates the process of family myth and family mystification in regard to the death of a parent. It was the myth and mystification of his mother's existence or death that drove Harry to his death. Therefore, my third reinterpretation is that the death of a family member is not pathogenic; myth and mystification of death are pathogenic.

THE FAMILY AS MEDIATOR OF MYTHS

The family is the bridge and mediator between the social matrix and the individual. It is through the medium of the family that the child learns to participate in the larger society's perceptions and constructions of reality (22). We live in a social matrix that defines death as unnatural, pathological, and even pornographic. Our society has elaborated myths and mystifications about death; it is therefore not unexpected that families transmit and participate in these myths and mystifications of death in the family (23). It should not be surprising that clinical data reveal that children as well as adults have difficulty coping with death. My fourth reinterpretation, therefore, is that children can cope with death but will have difficulty coping with family myths and mystifications of death.

Two conceptual distinctions must be noted here. I am not suggesting that the death of a parent is not stressful. The distinction is that successful family coping may enable the child to cope with stress so that it is not converted into trauma. The other distinction is between levels of anxiety about death. The first level is the experiential relationship with an object. We might term this "psychological death anxiety." The second level is the relationship of the self with the world. We might term this "existential death anxiety." Ernest Becker (6) brilliantly showed that we confuse these two levels. He suggested that only when we come to grips with the existential dilemma of death do we equip ourselves to deal with psychological loss. I concur with Becker and suggest that mystification of psychological loss is a maneuver to deal with the existential dilemma of death.

Our existential view of death is also our view of life. If we cannot admit that death is part of life, it may be that we cannot face the fundamental psychological issues of separation and individuation that contain the kernels of loss. Therefore, I suggest that the management of death in the family is rooted in the basic man-

agement of object relation separation and loss in the family, which in turn is a reflection of the existential death position of the family.

Perhaps a personal vignette can best summarize this last point. When our children were young we discussed the death of an elderly neighbor one night at supper. After some speculative questions about death, the kids got right to the gut of it. They asked, "Mom, Dad, are you going to die?" Our answer was something like this:

"Yes, we will. We are in good health and do not expect to die while you are young and need our help. You can do some things for yourself now, and when you grow older you will not need us to help you. Even if we die when we are old, we will miss each other. It will be sad, but it will be all right because you will be able to take care of yourselves. And if we happen to die while you are growing up, we have made arrangements with Aunt and Uncle for them to become your parents in our place. They cannot replace us, but they can help you as we would want to help you. We would be sorry to die now and not be here to help you and enjoy you. But you will always have our love and that is the most important thing. And because we love you, we have made plans with Aunt and Uncle, who love you too. We cannot predict or prevent our death. We will die someday in your lives. But we can enjoy our family now. And when we die you will have these good days to remember and always be a part of your lives."

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A New Concept and Finding in Morbid Jealousy

BY JOHN P. DOCHERTY, M.D., AND JEAN ELLIS, R.N.

The authors describe an unexpected coincidental finding in three couples who sought therapy because the husband was pathologically jealous. In the course of treatment they found that during early adolescence each of these men had witnessed his mother engaged in extramarital sexual activity. The authors discuss the implications of this finding for further understanding of the etiology of the syndrome of pathological jealousy, its transactional dimensions, and possible psychotherapeutic approaches. They also present a typology of morbid jealousy that consists of excessive, obsessional-delusional, and ego dysfunctional forms.

PATHOLOGICAL JEALOUSY is a clinical problem that can appear in widely varying forms, contexts, and intensities (1–4). In the course of our clinical work with three couples during the past two years, a provocative and unexpected finding emerged that we think sheds light on the etiology and structure of at least one form of pathological jealousy as well as possible psychotherapeutic approaches to the syndrome.

It is somewhat difficult to designate this form of jealousy because, although many classifications of the syndrome have been proposed (5–12) (see table 1), none has gained wide consensus. We prefer to divide the pathological disturbances of jealousy into three categories: excessive, obsessive-delusional, and ego dysfunctional. All three of the patients we are reporting on here fell into the obsessive-delusional group; i.e., these patients had no obvious severe disturbance of ego function but obsessively or delusionally harbored the persisting notion that their spouses were sexually unfaithful.

CLINICAL FEATURES

The clinical form that obsessive-delusional jealousy takes is quite consistent. The central dominating symptom is a preoccupation with the conviction that the spouse is sexually unfaithful. Two accessory symptom patterns are generated from this source.

First, there is an almost constant harassment of the spouse, which takes the form of meticulous observation and reflects a watchful vigilance for any sign of the suspected infidelity. This observation may extend to the most absurd trivia of the spouse's personal life.

Second, there is an alternating cycle of rage and remorse. Actual physical assault is not unusual, homicide is a real risk, and physical destructiveness of one form or another tends to be the rule. These periods of rage seem to cycle with periods of despondency and, often, abject remorse. The patient is contrite and sorrowful and begs forgiveness. It is during this period that suicide becomes a risk.

FORMULATIONS OF PSYCHOPATHOLOGY

Studies of pathological jealousy have been either descriptions of a variety of precipitating, releasing, or circumstantial factors thought to be associated with the onset or exacerbation of pathological jealousy or elucidations of psychodynamic processes thought to be involved in the genesis of the disorder.

The first line of investigation has implicated a wide variety of factors, such as normative life crises (13), a vaguely defined jealous predisposition (2, 3), impotence (14), feelings of inadequacy and insecurity (15), and several others (16, 17).

Along the second line of investigation, a number of intrapsychic mental processes have been implicated (18). The major formulations have been the displacement of oedipal wishes (19–22), the projection of homoerotic or heteroerotic impulses (6), and oral sadistic fixation (23–26).

However, despite the overwhelming social and interpersonal consequences of this syndrome (4, 13, 16), almost no formulations have been made that systematically address transactional aspects of the disorder. In general, our present understanding of the psychopathology of this syndrome is limited.

NEW FINDING

For the last two years we have been treating three couples whose chief complaint was obsessive-delusional pathological jealousy in the husbands. A striking coincidental finding has emerged in these couples

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Dr. Docherty is Clinical Associate, Section on Neuropsychopharmacology, Adult Psychiatry Branch, National Institute of Mental Health, Bethesda, Md., and Ms. Ellis is Staff Nurse, Psychiatry Service, Veterans Administration Hospital, West Haven, Conn. Address reprint requests to Dr. Docherty at Bldg. 10, Rm. 4N214, National Institute of Mental Health, 9000 Rockville Pike, Bethesda, Md. 20014.

TABLE 1
Typologies of Pathological Jealousy

Study	Types of Jealousy		
	Type 1	Type 2	Type 3
Jaspers (5)	Personality development	Process occurrence	—
Freud (6)	Projected	Delusional	—
Ey (7)	Emotionally jealous reactions	Delusions of jealousy	—
Mairet (8)	Hyperesthetique	Monomanie	Folie
Lagache (10)	Reactive	Personality development	Process (psychic, organic, and toxic)
Revitch (11, 12)	Emotionally insecure	Conjugal paranoia	Psychotic syndrome
Mooney (9)	Excessive	Obsessive	Delusional
Docherty and Ellis*	Excessive	Obsessive-delusional	Ego dysfunctional (functional, toxic, and dementiform conditions)

* Reported in this paper.

during the course of treatment. We feel that this finding helps advance our understanding of the etiology, psychodynamics, and transactional structure of this syndrome. The jealous husbands' accusations formed a coherent pattern, but one that diverged noticeably at points from a realistic picture of their wives. In fact, the accusations more appropriately fitted the patients' mothers. In all three cases, it emerged that the husband had witnessed his mother engaged in extramarital sexual relations during his early adolescence.

CASE REPORTS

Case 1. Mr. and Mrs. A had been married for 27 years. Mr. A was 47 years old, his wife was 41, and they had three children aged 10 to 15. They came for treatment at the insistence of Mrs. A, who, having finally established a rewarding life outside the home, found the marital situation more and more intolerable.

They initially presented as a family in avalanching disarray. Both Mr. and Mrs. A had begun to drink heavily, and their sexual relationship, which was never very good, had terminated entirely. All of the children were doing poorly in their schoolwork, and the couple's fights had become more frequent and more violent. A core issue in these fights was Mr. A's condemnation of Mrs. A for alleged sexual infidelities.

Before they were married, Mr. A regarded his wife-to-be as a saintly virgin. Shortly after they were married, however, he began to accuse her of being unfaithful to him. These accusations greatly intensified during her first pregnancy and were repeated with each subsequent pregnancy. During each of the pregnancies Mr. A was convinced the child was not his. As time went by, his accusations and restrictions of Mrs. A increased: he monitored her telephone calls and examined her purse, and he would berate her until she would kneel at his feet and beg him to believe and trust her. At times, however, he would become contrite and self-accusatory and plead with her not to be angry with him. Then he would perceive some flirtation and the cycle would begin again.

During the course of treatment we began to see that some of the recurrent accusations Mr. A made about his wife simply did not fit. For example, he accused her of drinking, but when we examined this we found that, although she drank episodically (usually when she had a severe altercation with Mr. A), there was no evidence to support the belief that she was a daily drinker. Mr. A's mother, however, was well-known for her alcoholism; on more than one occasion Mr. A had had to go to town to pick up his intoxicated mother.

Mr. A also accused his wife of sexually consorting in an indiscriminate way with all sorts of undesirable characters. When we examined this we found that the only reasonably suspicious partners for Mrs. A's alleged infidelity were all respectable men. Mr. A's mother, however, had taken a job as a waitress when he was a young adolescent and had fraternized with various unsavory customers.

Having noticed these discrepancies, we pursued the matter further with Mr. A until he recalled a memory of great emotional charge. When he was 12 years old, he returned home unexpectedly early one day and found his mother engaged in sexual intercourse with a strange man. He was enraged at what he regarded as her betrayal of him and felt tremendously divided loyalties between his mother and his father. He resolved to say nothing even though his mother's affairs became the subject of violent arguments between his parents. This left him feeling bitter resentment toward his mother and guilty disloyalty toward his father.

Case 2. Mr. and Mrs. B had been married for 2 years; they had lived together for approximately 1 year before they were married. Mr. B was 36 years old; his wife was 31. This couple came for therapy after an incident in which Mr. B had become enraged at his wife for suspected infidelities. He had been drinking heavily and became physically abusive.

Almost from its beginning, this couple's relationship was plagued with Mr. B's suspiciousness. There were indications of his jealousy even before their marriage, but Mrs. B was not fully aware of the extent of his preoccupation and concern. She soon learned, however, to be extremely cautious in her interactions with other men. Mr. B demanded, for example, that at a party Mrs. B should be with him or with other women but never with other men. He needed to know her whereabouts at all times, and any unexpected change in her plans could throw him into a jealous rage. At those times, he would begin to drink, which led to even more bitter haranguing and persistent interrogation of his wife.

During treatment, we noticed a phenomenon in Mr. B similar to the one that occurred with Mr. A. Mr. B accused his wife of being grossly negligent in her household duties, but when we examined this matter in concrete detail we found that this was a highly inaccurate appraisal. It did, however, fit Mr. B's memory of his mother. This led us to pursue the question of his mother's sexual activities. With confusion and angry resentment, Mr. B recounted that when he was a young teenager his mother began to frequent a neighborhood tavern. He recalled calls from the bartender to take her home and remembered seeing her there with other men. He also recalled with bitterness a time when he came home from school and found her engaged in sexual activity with one of

the men from the tavern. Like Mr. A, Mr. B did not divulge this information to his father, although he felt terribly conflicted. From that time on he tried to cut himself off emotionally from his mother.

Case 3. Mr. and Mrs. C were both 42 years old. They had been married for 23 years and had five children. Through most of the years of their marriage, jealousy posed little problem. In fact, Mr. C paid little attention at all to Mrs. C. Three years before they came for treatment, however, things changed. Mrs. C joined a weight-reducing program, changed her style of dress, and began to look considerably more attractive. Mr. C showed signs of increased interest in her, but it took the form of mockery and growing jealous preoccupation.

Mr. C struck up an acquaintance with a man whom he began to refer to as his "best friend." He began to bring this friend home and leave him alone with his wife. Finally, he planned a trip to Florida with his wife but at the last moment stated he could not go and insisted that this friend go with her instead. Notably, the friend had recently been released from jail following a conviction for a sexual morals offense. On their return from Florida the man informed Mr. C that Mrs. C and he had had sexual relations while on the trip. Mr. C was beside himself with anger and hurt. He began to verbally attack his wife in the most vile and derogatory way. He was convinced that she was having affairs with other men. He followed her, had the telephone disconnected, and took away her car. He also continually questioned her, wanting her to tell him why she had slept with his friend. No explanation satisfied him. He began to drive his wife to distraction and himself to desperation.

We asked Mr. C about infidelity on the part of his mother. He reported that his mother had had extramarital affairs and that he became aware of this in a singularly dramatic and disturbing way when he was 14. One day he returned home after having been sent shopping by his mother and found her engaged in sexual intercourse with a man he regarded as his father's best friend. He was shocked and disgusted, but he never told anyone about the incident until he recalled it with us in therapy.

DISCUSSION

We feel that the striking and unexpected finding that three men with pathological jealousy had seen their mothers engaged in sexual infidelity when they were young adolescents is important in furthering our understanding of the obsessive-delusional form of pathological jealousy. We therefore examined the relevance of this finding to the etiology of the syndrome, the intrapsychic and transactional aspects of its structure, and possible psychotherapeutic interventions.

Etiology

The phenomenologic work on pathological jealousy has tended to implicate a wide variety of releasing or facilitative states in the genesis of this syndrome, but it has not revealed any specific factors. Psychodynamic formulations have also been broadly general. Our finding, however, holds the possibility of providing a specific etiologic factor for at least some cases of obsessive-delusional jealousy. It provides clear continuity between the patient's symptoms and his life experience.

Our speculation is that the patient is unable to integrate the specific categorical trauma of the mother's sexual infidelity. Under provocative or exciting circumstances, therefore, he deals with the trauma by means of the syndrome of obsessive-delusional jealousy. We refer to the mother's infidelity as a categorical trauma because we suppose that actually witnessing it is probably not necessary and that one isolated event is probably not sufficient; strong fantasies of maternal infidelity elicited by a seductive or flirtatious parent may also be effective causes.

Intrapsychic Aspects

It is not hard to imagine why a mother's sexual infidelity would have a pathological outcome in her son. Even on the most superficial level this event might be expected to be sufficiently discordant and to produce sufficient anxiety to foster dissociation from the major stream of mental life. This behavior is extraordinarily threatening to the integrity of the family group and introduces numerous survival anxieties. Much more to the point, however, are the probable harmful effects of such an event on the young adolescent boy's oedipal complex. This would be expected to be particularly powerful because early adolescence is normatively characterized by an oedipal resurgence. We might therefore expect a marked intensification of the fantasies of and drive toward oedipal fulfillment because the mother has demonstrated that she can be sexually available to someone other than the father. The traumatic event gains even greater power by fitting almost precisely the form usually taken by the family romance fantasies during this developmental epoch (27, 28).

We believe that one source of the aggression that characterizes the pathological jealousy syndrome is derived from two other vicissitudes of the same traumatic event. First, although the mother appears to be more available sexually to the patient, she is not actually. This constitutes in effect a tease. Second, but perhaps more important, the rage that the son feels at being second best in the oedipal situation is exacerbated in a more serious, profound, and damaging way. Now he is not only second best to his father but to strange men who have no valid claim on his mother at all. By cuckolding the father the mother makes him second best. Thus the son is unable to use an identification with the father to achieve preeminence. He is doomed unalterably to second-rate status.

We would expect the rage from this source to be enormous; we would also expect a strong drive to undo such a situation; and we hypothesize that it is this drive which lies behind the compulsive, baseless accusations of these patients and their curious avoidance of situations that might provide positive proof for their suspicions (15, 16). Thus they are able to introduce doubt and undo in some measure that terrible reality. Our observations strongly support this line of deduction, although our clinical material does not permit

confirmation in complete detail.

In addition to the issues already mentioned, there is yet another interesting speculation concerning the effects of our finding on the oedipal complex. Noteworthy in our clinical material is the very similar, almost stereotyped description each of the three men gave of his father. The fathers were almost uniformly described as passive, hardworking, long-suffering, and nurturant. The patients said their fathers were the parent with whom they had the greater conscious affectionate ties. This might well suggest a strengthening of the negative oedipal complex in these men, which might help explain the presence of an important homosexual component in patients with obsessive-delusional jealousy, albeit a less malignant component than that found in some cases of ego dysfunctional jealousy.

Transactional Aspects

Our finding suggests the operation of projective transference distortion in obsessive-delusional jealousy. One of the most cogent formulations of this process has been given by Framo (29), who described it as involving an intimate relationship in which a split-off or disavowed part of an internalized object relationship is imposed on another person. The subject relates to the other person as the projected internal object and attempts to force the other person into becoming that object (30–32).

This process stood out in bold relief in the attempt by the three husbands we treated to live out with their wives the previously unexpressed aspects of their highly conflicted oedipal relationships with their mothers.

This formulation has important implications for our clinical understanding of this disorder. It brings more obscure aspects of the syndrome into better focus. For example, the curious and remarkable endurance of the wives in the face of the husbands' fanatical jealousy becomes understandable. Laing (32) noted the compelling, almost ineluctable force of projective transference distortion. Its effect is to produce what Framo termed a "transference fix" (33). The reason for this force has not been made clear by psychological investigation. We speculate that the projective transference distortion acts as a narcissistic trap. It severely wounds the self-esteem of the recipient and elicits the assumption that repair is possible only by changing the projection. This acts as a kind of Chinese handcuffs. The more the recipients struggle to free themselves, the more deeply enmeshed in the transference relationship they become.

Implications for Psychotherapy

We feel that recognizing the presence of projective transference distortion in the structure of the pathological jealousy syndrome represents a major new perspective for psychotherapy. We agree with Lloyd and Paulson (34) that unrecognized projective transference distortion between a married couple poses an almost insurmountable resistance to therapy and may account for the therapeutic nihilism that has been expressed about this syndrome.

More optimistically, recognizing the presence of this process is a positive indication for certain obvious therapeutic interventions. The therapist should carefully develop an understanding of the distorted images the spouses hold of one another. The demonstrable differences between the actuality of the mate and the projected images should then be clearly and repeatedly identified. Further, the therapist should be aware and make use of the fact that as the therapeutic relationship grows the therapist's perceptions of the husband and wife will serve increasingly as a countervailing force against the pull to conformity with the transference distortion. This seems to allow the partners to display novel behavior within the therapy session that is at variance with the transference expectations. Much therapeutic gain can be made by attending to the emergence of discrepant behavior. This formulation also insists that therapists attend to the details of the accusations of the jealous husbands because these details will allow more effective and accurate reconstruction of the disavowed relationship and thus enhance its reinternalization.

Finally, our analysis suggests a rational basis for the difficult question of the simultaneous use of multiple modes of treatment. A clear but interdigitating difference in the tasks of individual and couples treatment is indicated. Couples treatment would seem to be the preferable mode for the analysis of the projective transference distortion and the reciprocally interlocking circuit that gets set up around it. Individual therapy would seem a more efficient and effective mode for a full exploration and resolution of the conflicted oedipal relationship of the husbands.

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Treatment for the Parents of Feminine Boys

BY LAWRENCE E. NEWMAN, M.D.

Because extreme boyhood femininity is often a precursor of adult transsexualism, transvestism, and homosexuality, the author recommends early intervention for boys who meet specific behavioral criteria of gender disturbance. It is necessary to involve the parents in such treatment and to deal with the resistances they may have to recognizing and working on the problem as well as problems within the marital relationship that may affect the child's behavior. These boys are remarkably responsive to treatment given between the ages of 5 and 12, becoming more masculine in behavior and more comfortable with their identity as males.

YOUNG BOYS who behave like girls have been the subject of increased clinical concern in recent years. There is evidence that boys with pronounced feminine behavioral patterns do not necessarily become masculine with time. In many cases boyhood femininity is a precursor of transsexualism, transvestism, and homosexuality. This was first suspected because almost all adult male transsexuals and many homosexuals and transvestites gave histories of dressing up as girls and pretending to be women in their play as children (1–4).

Prospective studies lend support to these clinical reports. Green (5) conducted longitudinal studies of 5 feminine boys. As adults, 3 had become homosexual, 1 refused an interview but was reported by his family to collect photographs of nude males, and 1 reported sexual arousal when putting on feminine underpants, although he had not cross-dressed since age 6. Lebovitz (6) found that of 16 adult males who had been considered feminine in childhood, 3 were transsexuals, 2 were homosexuals, and 1 was a transvestite. Zuger's follow-up study of 6 feminine boys into adolesence indicated that 3 were homosexual and 1 was transsexual (7). Thus, evidence from recent studies indicates that feminine boys are a high-risk group for adult gender identity disorders.

Dr. Newman is Adjunct Assistant Clinical Professor, Department of Psychiatry, University of California School of Medicine, 760 Westwood Plaza, Los Angeles, Calif. 90024.

POSTPUBERTAL GENDER DISORDERS

Gender identity aberrations seem to crystallize during puberty, becoming essentially irreversible (8). Despite the efforts of therapists working with various methods, reports of real change in adolescent or adult transsexualism are very rare (9–11). Barlow and associates (12) reported one case of gender reorientation by behavior therapy in an adolescent transsexual. Other reports of gender reorientation of transsexuals, however, describe individuals who began treatment with evidence of significant preexisting masculinity. We recently described a number of men who initially insisted on reassignment surgery but gave up this wish after psychotherapy (13). These men did not manifest exclusively feminine lifelong interests, nor was there a total absence of masculinity in their adult lives. They had suddenly seized upon the idea of sex change as a magical solution after a crisis such as loss of a homosexual lover or schizophrenic decompensation. Truly transsexual males, in contrast, feel like women continuously. They have given up any desire to be accepted as males and have no motivation for treatment to alter their feminine orientation. However, many do recall wishing for acceptance as boys in childhood; they often report that this desire was then as strong as their feminine feelings. During adolescence, they give up the wish to be accepted as a male by others. Sex reassignment for these males as adults is a palliative treatment in which the body is altered to fit the mind because the individual is no longer able or willing to change his mind to fit his body. Therapeutic results with other gender disorders, such as transvestism and effeminate homosexuality, is also poor, suggesting that these less severe gender misorientation patterns also become fixed after puberty.

RESPONSIVENESS TO EARLY INTERVENTION

Feminine boys, unlike men with postpubertal gender identity disorders, seem remarkably responsive to treatment. Feminine boys treated between the ages of 5 and 12 gradually become less feminine and more masculine (14–17). Cross-dressing and feminine fantasy play diminish rapidly and the boys become more verbally and physically aggressive toward their mothers in almost all reported cases. Teasing and social rejection by male peers decreases and is replaced by acceptance. During the initial 12–24 months of treatment, these patients begin to enjoy being accepted as boys, and their acceptance is a strong, continuing reinforcer. Improvement has been achieved with a variety of different approaches, including daily psychoanalytic play therapy (14, 17), behavioral therapies (15), and individual therapy for the child combined with counseling for the parents, either individually or in groups (5). We reported the results of 4 years of treatment of 5 extremely feminine boys. Treatment consisted of once or twice weekly individual play therapy with a male therapist and parent counseling (18). All of these boys became more masculine, more aggressive, and discontinued their specifically feminine behaviors (e.g., cross-dressing).

The dramatic results that can be achieved are noted in Rekers and Lovaas' discussion of the case of a very feminine little boy treated with a behavioral approach.

> When we first saw him, the extent of his feminine identification was so profound (his mannerisms, gestures, fantasies, flirtations, etc., as shown in his "swishing" around the home and the clinic, fully dressed as a woman with a long dress, wig, nail polish, high screechy voice, slatternly, seductive eyes) that it suggested irreversible neurological and biochemical determinants. After 26 months follow-up, he looked and acted like any other boy. People who viewed the video taped recordings of him before and after treatment talk of him as "two different boys." (16)

Some of the boys treated in childhood have now become adolescents and remain masculine. None are reported to have reverted to cross-dressing. Transsexualism no longer seems likely for any of them. However, their future choice of sexual objects remains unknown.

Acceptance by same-sex peers in childhood replaces ostracism with a happier existence. They no longer need to withdraw into the feminine fantasy world and are capable of giving up this world voluntarily.

It seems evident that the treatment of choice for gender identity disorders is primary prevention in childhood. No matter how dramatic the femininity of a prepubertal child may be, the evidence indicates that his gender orientation has not yet crystallized. These patterns are not fixed by the third year of life (19).

FAMILY PATHOLOGY

It is fortunate that very feminine boys have behavioral patterns that are easily identified. However, these obvious signs do not always lead the families of boys at high risk to seek help. It is not unusual for parents to shield, tolerate, and rationalize the boy's cross-dressing for years. Mothers generally fear losing the son's companionship as he becomes more masculine and are therefore reluctant to begin a treatment program. The fathers generally resent and avoid their feminine sons.

Both parents tend to find rationalizations for avoiding professional help. The most common are: 1) the problem is being exaggerated and is actually much milder than the person who has pressured the parents to seek help believes it to be, 2) it will go away with time, 3) evaluation or treatment might in some way damage the child's sensitive nature or paradoxically make matters worse by "making the child think about it more," and 4) the feminine son is "destined" to become a homosexual (or other very feminine male) regardless of any intervention, so why bother? Fathers

who employ such rationalizations avoid their sons, which compounds and sustains the problem. The mother's "tolerance" really means insulating her son from the insults of siblings, father, or schoolmates by building a tight cocoon in which he can indulge his feminine play. She is being tolerant not of her son but of his pathology, and is indulging her need for his dependency and companionship.

In evaluating an extremely feminine boy, one should anticipate such parental rationalizations and search for them even if they are not openly expressed at first. They must be effectively countered during treatment. In the rare case in which there is no covert parental resistance, therapeutic change may occur with extraordinary speed (18).

THE EVALUATION PHASE: DECIDING WHETHER TO TREAT

Critical assessment of the parental description of the boy's gender role behavior, past and present, precedes direct assessment of the child in the playroom. I believe that it is useful to divide these behaviors into two categories and that treatment is not indicated for a complaint of boyhood femininity unless one or more behaviors from the first category are described. These first-category behaviors are those which, if continued into adult life, would make up the clinical picture of adult transsexualism.

1. Cross-gender clothing preferences (wearing girl's dresses, putting on the mother's shoes, wigs, makeup, or jewelry, or creating dresses or skirts out of towels or other materials).
2. Verbal statements expressing the wish to become a girl or to grow up to become a woman.
3. Taking female roles in fantasy games such as that of actress, mother, grandmother, or witch, while protesting if given the role of a boy or man.
4. Imitating the gestures and mannerisms of a woman, including exaggerated swaying of the hips, effeminate gait, feminine inflection of speech, etc.

These major (pretranssexual) behaviors generally are found together with the following "minor criteria" behaviors. However, if no major criteria behavior is present, significant gender pathology is probably not present.

1. Dislike and avoidance of rough, competitive boys' games.
2. Dislike of mechanical toys such as trucks, metal airplanes, construction equipment, etc.
3. Preferences for artistic activities (painting or listening to music) or for other sedentary activities.
4. Enjoyment of girls as playmates.
5. Gracefulness in bodily movements and gestures (to be contrasted with the exaggerated imitation and mimicking of the behavior of women that is a major behavioral criterion).
6. Reports of being teased as a "sissy" by other boys.

Without the presence of the first-category behaviors, these behaviors do not reflect gender pathology. Each reported behavior should be evaluated for intensity (emotional importance to the child) and duration; a behavior that occurs only once or twice and is willingly given up does not fulfill a criterion. The description earlier in this paper of the boy treated by Rekers and Lovaas (16) is typical of the truly feminine boys who meet at least one and perhaps all of the major criteria described previously.

Not infrequently, evaluation is requested by parents for boys who meet none of the major behavioral criteria. These parents may require only a careful hearing and reassurance. Such reassurance relieves the parents' anxiety and helps to prevent the negative impact that their doubts about the child's masculinity can cause. For example, the parent who believes that his/her son is not masculine solely because he avoids rough games and prefers reading or painting may be subtly harming the boy's sense of worth.

The parents' reports of the boy's behavior are essential to making the diagnosis of boyhood femininity and determining whether treatment is necessary. These reports must then be followed by playroom evaluation of the child, in which his gestures, choice of play materials, and fantasies expressed in play are observed and assessed (5).

THE TREATMENT PHASE

It is essential that the therapist enlist parents' support and commitment to the lengthy treatment endeavor. The therapist has to bring into the open and fight the nihilism of the parent (usually the father) who feels that his son is already a homosexual or worse and wants nothing to do with him. He must also counter the ambivalence of the mother, who may express the fear that treatment will upset her son and make him unhappy. Mothers may feel that because the son has so little else (no boy friends, girl friends whose parents dislike him, ostracism at school), it would be cruel to deprive him of his feminine activities. They may covertly supply him with feminine play materials after treatment starts because they do not want him to be sad. They are also satisfying their own need to keep the boy emotionally close by such activity.

The case of a very feminine 10-year-old boy is illustrative.

CASE REPORT

The parents brought the child for evaluation only after the pastor of their church observed the boy's feminine ways and love of makeup when he was in a church play. The boy had cross-dressed for at least six years with his mother's knowledge. The father expressed the feeling that his son was a "fag," which drew a startled and angry look from the mother but no protest. The father loved to hunt and fish and was therefore away from home almost every weekend. He said

he had tried to take his son along but "gave up" because the boy did not seem to enjoy the activity. The boy felt that his father hated him. He usually stayed in his room when his father was at home to avoid encountering him. The boy's symptoms included frequent cross-dressing, effeminate posturing, and the wish to become a woman.

Throughout treatment, sessions with the mother were necessary to help her accept the emotional loss of her son as he became more outwardly aggressive and began to express hostility toward her for the first time. The mother was a lonely woman who had tolerated the emotional and physical absence of her husband much of her married life. Shopping with her son was one of the few high points in her day, and in the course of the treatment he turned against this activity. She disliked giving up the companionship of her son on visits to the beauty parlor, an outing he had previously loved.

Sessions with the mother sometimes dealt with the meaning of masculinity and femininity. The mother secretly hated what she then thought masculinity was—a mixture of brutality, sexual license, and boorishness. These had been qualities of her own hated father, and she felt they were also characteristic of her husband. She preferred boys who were "gentle" because she feared that the "other kind" would grow up to become "selfish, mean men." She had discouraged any signs of assertiveness or aggressiveness in her son and often expressed to him her hatred for the "nasty" boys in the neighborhood. Her son loved her more than he loved his father, she reported with some pride, as if it were a contest.

After 15 months of treatment the boy, who had become much less effeminate in his mannerisms and had drawn away from his mother, seemed to regress, and the mother seemed to be hiding something. She resented my asking her what was making her uneasy, and angrily replied that nothing was wrong. Shortly thereafter the son revealed to me that he had for the last two months been wearing a pair of his mother's high-heeled shoes in the house when his father was not home. When confronted with this, the mother admitted in her session that she had given the shoes to her son because "I was all through with them anyway and he always used to admire them so. I felt, well—I'm going to be throwing them away anyway, so why shouldn't he have them?" With help, she was able to see that her behavior had been an effort to sabotage the treatment. She had difficulty facing this fact when it was openly stated and felt very guilty. This enabled her to begin to work more seriously on her fears of losing her son's companionship and on her hatred toward her husband and father and her fear that her son was going to become "just like them." The son rapidly became more assertive and less dependent on his endless solitary hours of feminine fantasy play. He began to make friends with other boys for the first time in his life, but preferred sedentary activities.

During this phase of the treatment it was possible to enlist the father's help. Up to that point he had not been able to "find the time" to come see me. Encouraged by his son's clinical improvement, the father again offered to take him on fishing trips, and the boy now accepted. The shift in family dynamics, with the mother no longer demanding (or needing) the son's total involvement and the father's new acceptance of him enabled his gender role behavior to move rapidly in a masculine direction.

This case report is illustrative of several issues that the psychiatrist should anticipate in approaching the parents of a very feminine boy. Of greatest importance is the mother's covert dependence on the unique emotional relationship with her feminine son, which makes her ambivalent toward the goals of treatment. In addition, she may be genuinely confused about the "definition" of masculinity and femininity and associate masculinity with ugly and insensitive behavior. She needs help in tolerating her son's initial unhappiness when he is told not to cross-dress. She will also need help in recognizing and reinforcing specifically masculine behaviors while negatively reinforcing specifically feminine behaviors such as cross-dressing. Treatment in which the boy is secretly allowed to cross-dress at home is only a training in deception, yet it does occur.

Sometimes the parent's uncertainty about the definitions of masculine and feminine behaviors comes from confusion about his/her own gender role. When this problem is pronounced, psychotherapy for the parents is indicated.

A further important goal in working with the parents of feminine boys is to improve the marital relationship—schisms between the parents are apparent in almost all cases. Gender aberration is extremely unlikely when love and sexual pleasure are present in the parents' marriage. Treatment for the parents aimed at overcoming their distance from each other is a powerful tool in freeing the child from the family scapegoat role in which he has been trapped.

THE POSTTREATMENT PHASE

When the boy has become normal in his behavior for a period of at least six months, continued psychiatric treatment is no longer necessary. However, contact with the parents should be maintained continuously thereafter in order to ensure that the pathological family dynamics do not reassert themselves, bringing about a regression.

How is it possible for dramatic changes in gender orientation to occur as a result of treatment in childhood? Extremely feminine boys do not seem to develop normal oedipal conflict prior to treatment (19). Evidences of oedipal tension, especially incestuous feelings, castration anxiety, and identification with the parent of the same sex, are not seen. Without treatment, the preoedipal feminine orientation of the boy develops in an undistorted form and in adolescence he has a growing conviction that he must become female, thus creating transsexualism.

With treatment (i.e., the intervention of a masculine therapist who is respected and perhaps feared by the mother), oedipal conflict becomes evident. Pride in being a boy appears for the first time along with increased aggressiveness toward the mother, breaking their symbiotic tie. This is the same kind of behavior observable earlier in normal masculine boys during the oedipal phase. The male therapist initially substitutes as a friendly role model for the patient's own father, who is, almost invariably, a distant, unavailable, and often hostile figure.

ETHICS OF TREATMENT FOR FEMININE BOYS

Is it right to attempt to alter the behavior of the very feminine boy? Some who do not view homosexuality or even transvestism and transsexualism as illnesses argue that it is wrong to intervene. Others may argue that treatment for very feminine boys, although perhaps successful in replacing femininity with masculinity, does not necessarily alter future choice of sexual objects. The very feminine boy may not become a transsexual (as some do without treatment), but may instead be homosexual. Is one clinical state better than the other?

I believe that treatment *is* indicated for the very feminine boy. The preference for feminine behaviors seems to be based upon a deficit in learning masculine behaviors from an appropriate model. Once the therapist provides this model, identification occurs, new behaviors are learned, and the feminine behaviors are gradually given up. Even if the boy does not then become completely masculine, the reduction in overtly feminine gesture and play preference makes him acceptable to boys who previously teased and avoided him. Experiences of being ostracized and ridiculed may play a more important role than has been recognized in the total abandonment of the male role at a later time. With treatment, behavioral change, and a new acceptance by his male peers, the child gains in self-worth, which seems to influence the long-term outcome. Treatment should be directed not at turning the feminine boy into an athlete or suppressing his aesthetic yearnings, but rather at developing his pride in being male. A happier childhood as well as primary prevention of an adult gender identity disorder are two related goals of treatment.

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ECT in the Treatment of Mania: A Controlled Study

BY MICHAEL S. MCCABE, M.D.

In a chart review study of manic patients, the author found that ECT remarkably improved outcome variables of condition at discharge, duration of hospitalization, and social recovery in comparison with an untreated matched control group. More conclusive evidence would depend on a prospective trial with random assignment of manic patients to ECT, lithium carbonate, and major tranquilizers and blind evaluation of outcome.

RECENT LEGISLATIVE, social, and legal developments have resulted in serious constraints being placed on the use of electroconvulsive therapy in psychiatric practice. *Psychiatric News* has covered legal developments in California and Alabama in detail (1, 2). When major constraints are placed on the use of specific forms of treatment, it is imperative that claims for the effectiveness of these treatments be based on firm scientific ground.

Most reports on ECT as a treatment for manic-depressive psychosis deal with depression alone (3–6) or are anecdotal with regard to the treatment of the manic phase of bipolar (manic-depressive) psychosis (7–10). Two early studies (3, 11) employed controls. The study by Ziskind and associates (11) included manic episodes but used 14 treated patients and only 6 untreated controls. Treatment results were excellent (86% recovered at follow-up), but the number of controls was too small for statistical analysis. The careful study by Huston and Locher (3) used an untreated control group but specifically excluded patients with manic episodes. The paucity of controlled studies of ECT in the treatment of mania suggested the need for a companion report to the investigation by Huston and Locher. Consequently, I am presenting the results of a chart review study of ECT in the treatment of mania with untreated controls.

Dr. McCabe is Assistant Professor, Department of Psychiatry, State Psychopathic Hospital, University of Iowa, 500 Newton Rd., Iowa City, Iowa 52242.

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METHOD

Treatment Group

ECT patients were selected from consecutive admissions to the Iowa Psychopathic Hospital during the years 1945 through 1949 with a hospital diagnosis of manic-depressive:manic or manic-depressive:mixed. Symptoms of mania required by the "Diagnostic Criteria for Use in Psychiatric Research" (12) had to be documented in the hospital record. These diagnostic criteria are euphoric and/or irritable mood and at least three of the following symptoms: hyperactivity (including motor, social, and sexual activity), pressured speech, flight of ideas, grandiosity, decreased sleep, and distractibility. The syndrome had to be of at least 2 weeks' duration. Patients with a prior diagnosis of organic brain syndrome, mental retardation, schizophrenia, alcoholism, anxiety neurosis, obsessive-compulsive neurosis, hysteria, drug dependency, or antisocial personality were excluded. Thirty-two patients in the specified time period met the diagnostic criteria for mania: the 28 who were treated with ECT constituted the treatment group.

Control Group

Controls were selected from consecutive admissions to the Iowa Psychopathic Hospital from the period 1935–1941 and were matched to the ECT group on the basis of age and sex. The age difference between ECT patients and their matched controls did not exceed 3 years. The selection of controls was blind as to outcome. Data on a larger group of manic patients from which the control group was selected were reported in "The Iowa 500" (13). Diagnostic criteria in the control group were the same as those cited for the ECT group.

Procedure

Data collected for both ECT patients and controls included age, sex, marital status, length of episode at admission, prior admission, duration of hospitalization, type of discharge, condition at discharge, social recovery, subsequent episodes, family history, number of treatments, and clinical symptomatology. The quality of follow-up information has been discussed previously (13) and is based on letters to other hospitals, families, or private physicians, and interviews by social workers. Follow-up information for at least 6 months was available for all subjects except 3 control cases who were transferred to other hospitals. Statistical analyses were done by Fisher's exact test, except duration of stay, which was compared by an independent t test. A probability not exceeding .05 was required for statistical significance.

RESULTS

Demographic Data and Prior History

The ECT and control groups consisted of 17 women and 11 men each. Mean age was 33.5 years in the ECT group (range, 17–57) and 33.6 years in the control group (range, 18–58). The mean number of ECT treatments was 17, with a range of 6–32. Eleven of the patients required more than one series of treatments. Seventeen of the ECT group were married at the time of index admission compared with 15 of the controls (p=n.s.).

Symptomatology

Table 1 presents a comparison of the frequency with which different symptoms were noted in the two groups. As would be expected in patients selected by identical diagnostic criteria, differences are small and statistically nonsignificant. The most frequent symptoms were those required by the diagnosis.

Outcome

Follow-up information for at least 6 months after discharge was available for all subjects except the 3 controls who were transferred to other hospitals. Follow-up covered a mean of 5.2 years in the ECT group and 3.1 years in the control group. This difference appeared to reflect a greater tendency for patients to return to the same facility for treatment of subsequent episodes of illness during the years when ECT was available than was observed before the availability of ECT.

Results for category of discharge, duration of hospitalization, and last known outcome are given in table 2. Discharge condition, taken from the physician's discharge summary, was rated worse, unimproved or unchanged, mild or slight improvement, marked improvement, or well. (These ratings were collapsed for the statistical analyses.) "Worse" and "unimproved" are considered self-explanatory. "Mild improvement" was applied to those patients who experienced diminution of intensity of symptoms but who could be characterized as hypomanic and not delusional. "Marked improvement" was applied to patients who demonstrated no manic symptomatology at discharge. The term "well" was erratically applied. In the ECT group the breakdown was as follows: unimproved, N=1; mild improvement, N=4; marked improvement, N=18; and well, N=5. The breakdown in the control group was as follows: unimproved, N=15; mild improvement, N=3; marked improvement, N=9; and well, N=1. The principal difference between the two groups centers on the numbers of patients who were considered markedly improved or well: 23 patients from the ECT group (82.1%) were in these categories compared with 10 (35.7%) in the control group. Duration of hospitalization was defined as the total period of hospitalization for the index episode, including the time after transfer to another facility. The category "last known ill" includes 4 deaths in the control group; all occurred during the index manic attack. In 3 cases the description appeared to indicate fatal exhaustion. The fourth patient died of pulmonary embolism following a venous thrombosis in the thigh due to an improperly placed binder. No patient in the ECT group died dur-

TABLE 1
Symptomatology in 56 Manic Patients, in Percents

Symptom	ECT Patients (N=28)	Controls (N=28)
Mania		
Euphoric affect	96	93
Hyperactivity	100	96
Agitation	75	50
Excessive spending	36	17
Increased libido	32	39
Increased interests	25	—
Pressured speech	96	93
Flight of ideas	82	75
Grandiosity	57	42
Distractibility	71	82
Sleep disturbance	82	86
Depression		
Depressed affect	4	11
Anorexia	18	18
Weight loss	4	11
Decreased energy	7	4
Retardation	0	0
Suicidal thinking	7	11
Diurnal mood variation	11	11
Feelings of guilt	7	11
Delusions and hallucinations		
Passivity	0	4
Depersonalization	7	4
Symbolic disturbance	4	7
Changed perception	0	11
Persecutory delusions	32	21
Auditory hallucinations	11	18
Visual hallucinations	4	14
Haptic hallucinations	4	11

TABLE 2
Outcome Factors for 56 Manic Patients

Factor	ECT Patients (N=28)	Controls (N=28)	Significance*
Duration of hospitalization (weeks)			
Range	3–13	2–52	
Mean	6.5±2.13	15.3±11.3	p<.001
Discharge condition (percents)			
Improved	96	44	
Unimproved	4	56	p<.0001
Discharge category (percents)			
Home	96	44	
Transfer	4	56	p<.001
Social recovery (percents)			
Never socially recovered	0	30	
Recovered ever	100	70	p<.002
Last known recovered	78	56	
Last known ill	22	44	p<.05

* All p values are based on Fisher's exact test except duration of hospitalization (t=3.82, df=51). The number of control subjects in that comparison was 25.

ing a manic episode. The numbers of deaths in the two groups were not significantly different (p=.056).

DISCUSSION

This investigation used an untreated control group to study ECT in the manic phase of bipolar affective disorder. With the development of effective treatment methods such as lithium carbonate and major tranquilizers, it has become increasingly difficult to justify ethically the use of a placebo control group. This investigation circumvented that ethical problem by selecting controls from a period in which there was no effective treatment available for mania. Such an investigation can be performed only where records reflect standards of excellence over a period of many years; the Iowa Psychopathic Hospital has maintained such a tradition.

Although ECT patients and controls were selected from different time periods, illness variables that might have been expected to affect outcome proved to be remarkably similar. Age, sex, and diagnosis were controlled. Marital status, prior episodes, and duration of episode prior to admission were almost the same in the two groups (no comparison was statistically significant). Similarly, symptoms used in diagnosing mania were virtually the same in the two groups. There were no significant differences in the various types of hallucinations and delusions recorded for the two groups. Treatment and control groups were comparable in severity of illness and premorbid functioning at the time of their index admissions. Only four patients who met diagnostic criteria for mania did not receive ECT. These patients all had milder forms of the illness, and this selection bias tended to favor the control group.

The principal defect in design was that the subjects were selected from different time periods. Although all outcome measures favored the ECT group, differing expectations of physicians regarding the prognosis of the disease during these time periods might have affected any or all of the outcome variables. Had this been the case, few of the patients in the 1935–1941 period should have been described as markedly improved or well. In fact, 36% were so described. Similar arguments could also be presented for the discharge category. If the expectations were uniformly gloomy, the discharge to home of nearly half (44%) of the control group would be indeed remarkable. Discharge data from the two periods provide further information. The mean duration of stay at the Iowa Psychopathic Hospital during 1935–1941 was 37.0 days compared with 46.1 days in the period 1945–1949. This is a reversal of trends found in the current investigation for manic patients. In addition, the magnitude of the difference in mean duration of stay of patients and controls would be difficult to explain entirely on the basis of physician expectations. The most definitive way to deal with this question would be to randomly assign patients to treatment and control groups, using blind evaluation of results.

Differences in final outcome are difficult to explain on the basis of physician expectations. Social recovery was based on the report by the Social Service Department that the patient had returned to his/her premorbid level of functioning. Most important here is the finding concerning death during the index episode. Four untreated manic patients died during the index episode. With the variety of treatments available today—lithium carbonate, phenothiazines, butyrophenones, and ECT—the thought that untreated mania can be lethal arises infrequently.

The finding that 96% of the ECT group were sufficiently improved to be discharged to home suggests that ECT may be a valuable tool in the treatment of the manic episode. Previous reports dealing with the efficacy of ECT in manic-depressive psychosis dealt only with the depressive phase (3–6) or were anecdotal (7–10) and had no controls. This may reflect in part diagnostic concepts that did not distinguish between unipolar (depression only) and bipolar (mania and depression) affective illness. This report partially fills the void in the literature concerning ECT in the treatment of the manic phase of bipolar affective disorder. More conclusive evidence concerning the efficacy of ECT would have to come from a prospective trial with random assignment of subjects to ECT, lithium carbonate, or a major tranquilizer and blind evaluation of outcome.

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Psychiatric Illness in Homicide Victims

BY MARIJAN HERJANIC, M.D., AND DAVID A. MEYER, M.D.

The authors studied the demographic characteristics and history of psychiatric treatment of 214 homicide victims and 67 suicide victims. Thirty-two (15%) of the homicide victims and 12 (18%) of the suicide victims had a record of psychiatric care; these rates were higher than the treatment rate of the general population. The authors suggest that there may be a connection between the occurrence of homicide and the increased frequency of psychiatric disorders among victims of this crime.

THE INCREASE in the number of homicides in urban areas has recently attracted a great deal of public attention and concern, and the voluminous literature on homicide indicates that social scientists as well as the public have a great interest in this crime. In St. Louis, the homicide rate increased from 8 per 100,000 in 1942 (1) to 33 per 100,000 in 1973, and other large cities have experienced similar or larger increases.

Early literature on murder focused on the offender, but Von Hentig (2) shifted the emphasis to the victim-offender relationship, and Wolfgang (3) pointed out that one-fourth of homicide victims had clearly precipitated their own deaths. Houts recently went even further by stating that "in a great many cases, the murderers are rather reasonable normal people, whereas the victims were the discordant individuals—at least, they possessed strange quirks of personality and character that contributed mightily to their demise" (4, p. 230).

This suggestion of psychological aberrations in homicide victims raises the issues of psychiatric illness among victims and the potential for a contribution by mental health workers to the prevention of crimes. Because we were unable to find any systematic studies of psychiatric illness in homicide victims, we decided to examine the circumstances in which homicides occur and the general characteristics of victims, with special emphasis on victims who had received psychiatric care. We contrasted our findings about homicide victims with the characteristics of a well-studied group of individuals who also suffer violent deaths—suicide victims.

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Dr. Herjanic is Associate Professor, Department of Psychiatry, Washington University School of Medicine, St. Louis, Mo., and Assistant Superintendent, Malcolm Bliss Mental Health Center, 1420 Grattan St., St. Louis, Mo. 63104. When this work was done, Dr. Meyer was a medical student, Washington University School of Medicine; he is now a resident in psychiatry, Washington University School of Medicine and Malcolm Bliss Mental Health Center, St. Louis, Mo.

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METHOD

There were 220 homicide victims in the city of St. Louis in 1973. Since we were primarily concerned with studying psychiatric treatment of homicide victims, 4

victims whose names were unknown were dropped from the study. Two other victims, children 18 months and 9 years old, were also dropped; thus the final study group contained 214 victims.

The circumstances of each crime were studied and coded. To find out about psychiatric care received by homicide victims, a list containing the name, sex, race, and date of birth of each victim was compared with a list of patients treated at two large state-supported institutions (Malcolm Bliss Mental Health Center and St. Louis State Hospital) that provide psychiatric care for the city of St. Louis. Victims' medical records discovered by this method were reviewed systematically and data were recorded.

Data were gathered by a similar method on all persons (N=67) who committed suicide in St. Louis in 1973. This group was used for comparison purposes.

RESULTS

Table 1 gives a summary of the study results. Men predominated in both the homicide and suicide victim groups. There were significantly more black homicide victims than suicides ($p<.001$). Homicide victims were significantly younger (average age=35 years) than suicide victims (average age=49 years) ($p<.001$, by t test); blacks were younger than white victims in both groups. Approximately the same proportion of both groups had a history of psychiatric treatment.

To determine how many former patients one could expect to find among homicide and suicide victims, we used data from one center (Malcolm Bliss Mental Health Center) that provides psychiatric care for approximately two-thirds of the total population (622,000) of St. Louis. Only homicide and suicide victims with home addresses in the catchment area of this center were used for comparison. Although only 4% (N=15,848) of the people living in the catchment area had a record of patient care at the center, 12% (22 of 183) of the homicide victims and 22% (10 of 44) of the suicide victims had been treated. The differences between the treatment rates observed for the homicide and suicide groups and the rate in the general population were highly significant ($\chi^2=28.13$ for homicide victims, 35.05 for suicide victims; $p<.001$). No significant difference was found between the rates of psychiatric care in the homicide and suicide groups.

Diagnoses

The diagnosis of each subject was taken from hospital records; no attempt was made to reach an independent research diagnosis. Alcoholism, sociopathy, and drug addiction accounted for 72% of the diagnoses of all homicide victims, with alcoholism the most frequent diagnosis (see table 2). If secondary diagnoses had been taken into consideration, 2 more victims would have been included under alcoholism, 1 more under sociopathy, and 1 more under drug addiction. All patients with a diagnosis of drug addiction had been addicted to opiates. Of the 4 homicide victims listed in table 2 under the diagnostic category "other," 1 had a diagnosis of hysteria, 1 situational reaction of adolescence, 1 inadequate personality, and 1 drug intoxication. Among the suicide victims, the patient listed under "other" had a diagnosis of hysteria.

It is interesting to note the complete absence of affective disorders among homicide victims and the rarity of other serious psychiatric illness such as schizophrenia, organic brain syndromes, or mental retardation. Since there were only 4 victims with these diagnoses, it was impossible to examine them as a separate group. However, some features distinguished them from the other homicide victims—they were considerably older (average age 53 years), and the circumstances under which they were killed were different (the two schizophrenic victims were killed by strangers in their own homes and the patient with organic brain syndrome was killed during a robbery, whereas a typical victim was killed by a friend or relative after an argument).

Suicide victims showed a different pattern of diagnoses. Fifty percent of the group suffered from affective disorders. The diagnostic distribution of suicide

TABLE 1
Characteristics of 214 Homicide Victims and 67 Suicide Victims

Item	Homicide Victims		Suicide Victims	
	Number	Percent	Number	Percent
Sex				
Male	182	85	50	75
Female	32	15	17	25
Race				
Black	174	81	17	25
White	40	19	50	75
History of psychiatric treatment	32	15	12	18
No psychiatric treatment	182	85	55	82

TABLE 2
Diagnoses of 32 Homicide Victims and 12 Suicide Victims Who Received Psychiatric Treatment Prior to Their Deaths

Diagnosis	Homicide Victims		Suicide Victims	
	Number	Percent	Number	Percent
Affective disorder	0	—	6	50
Schizophrenia	2	6	0	—
Organic brain syndrome or retardation	2	6	3	25
Alcoholism	11	34	2	17
Drug addiction	5	16	0	—
Sociopathy	7	22	0	—
Other	4	13	1	8
No diagnosis	1	3	0	—

victims in our study who had received psychiatric treatment was similar to the distribution found by Robins and associates (5) in a study of 134 suicides committed during 1956 and 1957 in the St. Louis area. Robins and associates (5) also found that, although almost all persons who committed suicide suffered from a psychiatric illness, only 19% were actually treated by a psychiatrist during the year prior to their deaths; in our study, 12% of the suicide victims had received psychiatric treatment during the year before their deaths.

DISCUSSION

Our method of determining the rate of psychiatric treatment of homicide and suicide victims is open to challenge, since we checked only the records of two public psychiatric institutions; other victims may have been treated instead by private practitioners or in private hospitals. A more accurate method would have been to interview the relatives and friends of the deceased victims to obtain a full psychological autopsy, for the important question is not only how many victims received psychiatric care but also how many displayed symptoms indicating a possible psychiatric disturbance and need for treatment.

We contrasted the group of homicide victims with a history of treatment for psychiatric illness with the rest of the homicide victims with regard to the following variables: sex, race, age, relationship with assailant, location of crime, drinking at the time of death, role of victim in precipitating the crime (3), place of death, weapon used, victim-offender relationship by sex, victim-offender relationship by race, and the time of day of the crime. The two groups of homicide victims appeared very similar with respect to these variables. Statistically significant differences were found for only 2 of the 12 characteristics examined—fewer victims with a history of psychiatric care had been killed by members of their own family and more were killed on the street or in their cars than victims without previous psychiatric treatment. However, in view of the small numbers in each subgroup and the low levels of significance obtained, these findings should be viewed as trends at best.[1]

CONCLUSIONS

Based on the data we gathered, the typical homicide victim was a young black male who was killed with a gun in an argument with a relative, friend, or acquaintance. In 91% of the homicides studied, victims and murderers were of the same race, and in 74% of the cases they were of the same sex. History of psychiatric treatment or absence of it did not divide homicide victims into two distinct groups.

The number of homicide and suicide victims with a history of psychiatric care was significantly higher than would be expected from study of the general population. There was a qualitative difference in the diagnostic distributions found for the homicide and suicide groups.

Although it is assumed that almost all suicide victims are psychiatrically disturbed (5), only a small proportion (18%) of suicides in our study had been treated by psychiatrists. Since the proportion of suicide and homicide victims treated by psychiatrists was similar, we wonder if we could conclude that most homicide victims are also disturbed.

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[1]A detailed table of the results can be obtained from Dr. Herjanic.

Forensic Psychiatry: Profiles of Two Types of Sex Offenders

BY FRITZ A. HENN, PH.D., M.D., MARIJAN HERJANIC, M.D., AND ROBERT H. VANDERPEARL, M.D.

The authors examined records of 239 individuals charged with sexual offenses and referred by the courts to a forensic service. Defendants charged with rape were typically under 30 with histories of antisocial behavior that included other types of violence. Major mental illness was rare in this group. Child molesters in the sample were of no particular age, usually had no history of violent behavior, and had a low incidence of psychosis. The most common secondary diagnosis in both groups was alcohol or drug abuse.

THIS PAPER reports an empirical examination of the characteristics of a group of defendants charged with sexual crimes and referred to the Malcolm Bliss Mental Health Center Forensic Service for psychiatric evaluation. The study was designed to answer two principal questions: Are there distinguishing characteristics between groups of defendants charged with different sexual offenses? What role does psychiatric illness, particularly psychosis, play in sexual offenses? The results of our study of 239 defendants can be most clearly presented by reporting on the individuals charged with child molestation and rape, which were the most common charges against defendants admitted to the service.

A wide variety of viewpoints has been brought to the study of sexual offenders. To some extent, this is due to the nature of the subject itself. Sexual offenses are, in part, culturally determined and therefore value laden, and responses to them become imbedded in the legal framework. The legal responses to these offenses often reflect the fears or values of the culture rather than the characteristics of the offender. Because the offenses are related to cultural values, opinions are strong and facts are sometimes difficult to ascertain. As Guttmacher and Weihofen (1) stated over 20 years ago, "There is doubtless no subject on which we can obtain more definite opinions and less definite knowledge." However, there have been advances since that statement was made. A book by Mohr and associates (2) on child molesters and exhibitionists provided a careful evaluation of these offenders. MacDonald (3) examined many aspects of rape. Thus, comparisons of different cohorts of sexual offenders have become possible, and the facts concerning sexual offenders can be established and made available not only to psychiatrists but to all concerned with these problems.

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Dr. Henn is Assistant Professor, Department of Psychiatry, State Psychopathic Hospital, University of Iowa, 500 Newton Rd., Iowa City, Iowa 52242. Dr. Herjanic is Assistant Superintendent and Dr. Vanderpearl is Director, Forensic Service, Malcolm Bliss Mental Health Center, St. Louis, Mo.

METHOD

We reviewed records of all defendants charged with sexual crimes and referred for evaluation to the Malcolm Bliss Mental Health Center Forensic Service, St. Louis, Mo., during the years 1952–1973. Out of a total of 1,195 cases, 239 involved defendants charged with at least one sexual crime. These cases were reviewed and coded for demographic variables, diagnosis, crime, and outcome. Crimes represent the charges against the defendant on admission to the service, and diagnoses are those given in the report to the court. The Forensic Service is responsible for all patients referred by court in the city of St. Louis and some patients (less than 10% of the total) from surrounding counties. In more than 90% of the cases, the defendant was admitted for a pretrial examination.

RESULTS

The cohort of 239 defendants had a total of 273 charges of sexual offenses. There were 69 charges of rape and attempted rape, 116 of child molestation, and 43 of sodomy. We will focus on rape and child molestation, since sodomy consists of a variety of different offenses. The other classes of offense, such as exhibitionism, were seen in only a small proportion of these defendants.

Demographic Variables

The demographic variable that most strikingly separated defendants charged with rape from those charged with child molestation was age. Those implicated in rape were much younger on the average—over 75% were under 30. Defendants involved in cases of child molestation were distributed over the entire age range—only 28% were under 30 and almost as

TABLE 1
Primary Diagnoses of 239 Sexual Offenders

Primary Diagnosis	Principal Sexual Offense							
	Rape and Attempted Rape (N=67)		Child Molestation (N=111)		Sodomy (N=42)		Other (N=19)	
	N	%	N	%	N	%	N	%
Schizo-affective disorder	2	2.9	2	1.8	0	0.0	0	0.0
Schizophrenia	6	9.0	10	9.0	3	7.1	4	21.0
Affective disorder	0	0.0	2	1.8	2	4.8	0	0.0
Antisocial personality	32	48.0	7	6.4	7	16.7	1	5.3
Other personality disorder	14	21.0	9	8.1	12	28.6	8	42.1
Drug and alcohol abuse	3	4.3	8	7.2	4	9.5	4	21.0
Organic brain syndrome	2	2.9	16	14.4	3	7.1	0	0.0
Mental retardation	2	2.9	15	13.5	5	11.9	1	5.3
Other	0	0.0	34	30.6	1	2.4	1	5.3
Not ill	6	9.0	8	7.2	5	11.9	0	0.0

many were over 50. The difference in mean age of the two groups was statistically significant ($p<.01$).

Concurrent Charges

We tabulated concurrent charges against defendants that involved violence or potential violence (i.e., homicide, robbery, and assault). Defendants involved in rape or attempted rape (N=67) had a total of 15 offenses against persons, while there were only 2 such concurrent charges in individuals charged with child molestation (N=111). Individuals charged with sodomy (N=42) accounted for 1 charge of murder, 1 of robbery, and 2 of assault. Our small sample of exhibitionists (N=7) had no concurrent charges of crimes against persons.

Psychiatric Illness

We examined the possibility of an association between types of sexual offenses and psychiatric disorders. Since our sample of defendants referred for psychiatric evaluation is biased toward the finding of psychiatric illness, we felt that any association between mental disorders and deviant sexual behavior should be evident. Table 1 gives a breakdown of psychiatric diagnoses for several groups of offenders. The predominant diagnostic grouping for those charged with rape is personality disorder; antisocial personality was found in 48% of this group. Psychosis (affective disorder, organic brain syndrome, or schizophrenic process) was reported for less than 15% of the defendants charged with rape or attempted rape. Over three-fourths of the rape defendants fell in the categories of personality disorders or no psychiatric illness.

A different picture emerges for those charged with child molestation: personality disorders are not predominant, and only 6% of these defendants were found to be antisocial. The dominant diagnosis is sexual deviant (pedophilia) without other disorder, which was given to 29% of this group (32 of the 34 cases in the "other" category). This group, combined with those categorized as having no psychiatric illness, constitutes over a third of all the child molesters seen. Other categories that contribute heavily to the diagnostic distribution in this group include organic brain syndrome, mental retardation, and schizophrenia, which were found in almost two-fifths of the cohort charged with child molestation. Alcoholism and drug abuse were the predominant secondary diagnoses in both rape and child molestation, constituting one-third of the secondary diagnoses for these two groups of defendants.

We examined the influence of major psychiatric disorders in defendants charged with sexual crimes by looking at the incidence of schizophrenia and affective disorders among defendants charged with such offenses. Police records from the years 1961 through 1973 for all rapists and other sexual offenders (excluding offenses involving commercial vice) were obtained. Unfortunately, child molestation was not kept as a separate category by the police. We then reviewed records of those in this group charged with rape and other sexual offenses who were seen on the Forensic Service between 1961 and 1973 for the presence of either illness. Since the Forensic Service is the only such service in St. Louis and receives more than 90% of its defendants from St. Louis City Courts, we assumed that the defendants with diagnoses of either schizophrenia or affective disorder would be in the group of defendants seen on the Forensic Service. This assumption is supported by the referral of a large proportion of defendants without severe psychiatric disorders, indicating a high index of suspicion of psychiatric illness on the part of the court. This makes it unlikely that the courts would overlook a significant number of defendants with serious psychiatric problems. Further, a study of convicted felons in Missouri found little psychosis in the prison population (4).

Schizophrenia, including cases of schizo-affective disorder, was found in 0.3% of all rapists (8 of 2657 cases) and 0.22% of other sexual offenders (11 of 4793

cases). No affective disorders were found among persons charged with rape and only 0.08% of other sexual offenders received this diagnosis.

The report to the court indicated that out of 104 requests for sanity evaluations in defendants charged with child molestation, 79 were found to be sane, and out of 105 competency evaluations, 86 were considered competent. For those charged with rape, 38 out of 40 evaluated for sanity were found sane, and 40 of 43 evaluated for competency were found competent. These figures indicate that insanity plays almost no role in rape, especially since this sample represents cases in which there is a potential psychiatric illness.

DISCUSSION

The findings we have presented allow us to suggest a typical profile for defendants charged with child molestation and rape. The rapists seem to be males under 30 with histories of antisocial behavior. They might also have a history of violent behavior, and their antisocial tendencies would not, for the most part, be confined to sexual areas. Major mental illness (psychosis, thought disorder, and/or delusions) would be unlikely in these individuals. This profile is comparable to that of the offenders studied by MacDonald (3)—75% of his sample were under 30, the vast majority had committed previous crimes, and 15% had a concurrent charge of robbery. He also found a disproportionate incidence of blacks among his offenders, and 41 of the 67 accused rapists we studied were black. No estimate of concurrent mental illness was given in MacDonald's study, but his impression was that it was very low. McCaldon (5), in a study of 30 convicted rapists, found that half had antisocial personalities. Gebhard and associates (6) estimated that less than 1% of rapists have psychotic illness. Amir (7) also found that young black antisocial males were the largest group among defendants charged with rape.

These data are consistent in suggesting that youth and antisocial tendencies are the most common features of rapists. More interestingly, they suggest that psychosis in no way predisposes an individual to sexual assault. Clinical impressions and data from the St. Louis Forensic Service suggest that the incidence of psychosis in rapists is lower than that among the general population. The myth of the "crazy rapist" seems to apply to the extreme exception, and from our data it could even be suggested that psychosis protects individuals from the possibility of committing rape.

The profile of child molesters suggests no particular age and, most likely, no psychiatric diagnosis other than sexual deviation. These individuals are unlikely to evidence other criminal behavior, and violence is not a feature of their activities. In our sample, mental retardation and organic brain syndromes were not uncommon, and 11% of those charged with child molestation were diagnosed as schizophrenic. Mohr and associates (2), in a study of 68 pedophiles, found a trimodal age distribution and an IQ range only slightly skewed to the lower end of the scale. They also found little violent or other criminal behavior in their sample. The incidence of psychosis was very low in their study and those they reviewed. The relatively high proportions of defendants with mental retardation, organic brain syndromes, and schizophrenia in our study reflect the fact that our subjects had been referred for a psychiatric evaluation, which would be expected to maximize psychopathology in the sample. Again, crude calculations suggest that the incidence of psychosis among pedophiles in general is very low.

The most common secondary diagnosis in both groups of offenders was alcohol or drug abuse. Alcohol, even without alcoholism, plays a major role in these offenses. This was suggested by Rada (8), who found that 50% of convicted rapists had used alcohol before committing their offense. The role of alcohol and secobarbital in violent assaults was also emphasized in a recent report by Tinklenberg and associates (9).

CONCLUSIONS

In summary, we found two distinct populations among sexual offenders referred for a psychiatric evaluation. The first, young and antisocial with a tendency toward violent crime, was involved in rape. The second, of varying ages with no marked antisocial tendency but some chance of suffering from organic brain syndromes, mental retardation, or schizophrenia, was pedophiliac. Psychosis does not appear to be associated with either of these sexual offenses.

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Peer Review of Prescribing Patterns in a CMHC

BY HERBERT DIAMOND, M.D., RICHARD TISLOW, M.D., THOBURN SNYDER, JR., M.D., AND KARL RICKELS, M.D.

Monitoring the prescribing patterns of psychiatrists is a challenging and now necessary professional responsibility. The authors describe a peer review and feedback system in a decentralized CMHC, linked to continuing education in psychopharmacology, that made staff psychiatrists familiar with the state of the art and accountable for practice within its guidelines. Data are presented that confirm shifts from polypharmacy toward more appropriate use of psychotropic medications.

THE MODERN ERA of psychopharmacology has evolved over the last two decades. In this relatively short period of time a new armamentarium of effective psychoactive agents has been developed, studied, and introduced into clinical psychiatric practice. For the first time psychiatric clinicians have available some uniquely therapeutic medications. Out of a massive clinical experience a number of authors have identified some ubiquitous problems in the use of these medications that point to new directions prescription practices must take in order to dovetail more appropriately with the state of the art (1–3).

The major problems highlighted in the literature include drug overuse and polypharmacy—combining multiple antipsychotic or antidepressant agents, excessive pairing of antipsychotic with antidepressant agents, and overuse of antiparkinson drugs. DiMascio (4) examined prescription practices in hospital settings and discussed the "folly of polypharmacy," as well as many instances of inappropriate use of individual drugs. Merlis and associates (5) also studied polypharmacy and concluded that it was not an adequate form of treatment. More specifically, a number of authors (6–9) have investigated the use of antiparkinson with antipsychotic agents and have presented data indicating that 70% to 90% of all patients so treated were given unnecessary prophylactic antiparkinson agents to reduce or eliminate extrapyramidal-like side effects of the primary antipsychotic medication.

In the course of a regular review of prescriptions written by staff psychiatrists in a large urban community mental health center (CMHC), one of us (H.D.) observed the perpetuation of practices that were increasingly being questioned by leading clinical psychopharmacologists. Monitoring of the prescribing patterns of our dispersed staff of psychiatrists not only met some needs of managerial accountability but also aimed at an optimum and consistent use of psychotropic medications throughout the center's service delivery system.

This was an ambitious undertaking since the West Philadelphia Community Mental Health Consortium, Inc., is a complex, decentralized CMHC that has developed a comprehensive system of mental health/mental retardation (MH/MR) services now consisting of 18 programs in 29 facilities. The Consortium represents its community by coordinating the activities of a local planning agency and the major health and educational institutions in the catchment area. It has brought together 11 institutions and their communities in a collaborative MH/MR venture in southwest Philadelphia. Four general physicians and 16 psychiatrists have responsibility for chemotherapy in the outpatient network of services that has been developed around a group of 7 neighborhood clinics, a partial hospitalization program, and components of an aftercare service.

The goal of developing prescription practices throughout the system that were consistent with the state of the art led to the development of an intramural review of prescriptions that had been issued free of charge by the Consortium's central pharmacy in one member hospital. Prescriptions for patients on public assistance are filled in Pennsylvania by local pharmacists, who are then reimbursed by the Department of Welfare. However, because this information had not yet been computerized, these medical assistance prescriptions were not available to the project.

Two years ago, the monthly prescription summaries began to be reviewed regularly in a conference setting by the medical director and psychiatric staff for rele-

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Dr. Diamond is Medical Director, Dr. Tislow was Director of Emergency Services, Dr. Snyder is Staff Psychiatrist, and Dr. Rickels is Consultant in Psychopharmacology, West Philadelphia Community Mental Health Consortium, Inc., P.O. Box 8076, Philadelphia, Pa. 19101. Dr. Rickels is also Director of Psychopharmacology, Philadelphia General Hospital. The authors are also with the University of Pennsylvania School of Medicine, where Dr. Diamond is Associate Professor of Psychiatry and Associate Director of the Division of Community Psychiatry, Drs. Tislow and Snyder are Associates in Psychiatry, and Dr. Rickels is Professor of Psychiatry and Pharmacology.

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vance to diagnosis, treatment of target symptoms, and the current literature. The conference became an ongoing seminar in psychopharmacology.

Since monitoring the professional performance of psychiatric colleagues presented a delicate although necessary responsibility, great care was taken to explain the purposes of the review, i.e., to improve prescribing skills, upgrade care, reduce costs where possible, and enhance continuing education. The last goal was particularly emphasized and consistently reinforced, and the psychiatric group coalesced around this important educational experience. Many in the group promptly applied their new knowledge to their part-time private practices with good results; this in turn reinforced enthusiastic participation in the review process in the CMHC. Although we had anticipated considerable resistance to this unfamiliar and possibly threatening professional exposure, we found that initial reservations readily gave way to a spirited acceptance of the opportunity for continuing education.

Since biweekly meetings could not possibly cover 450–650 monthly prescriptions, we decided to establish a medication review committee. By group consensus, a committee of 4 senior clinicians was mandated to review all prescriptions on a continuing basis and to question the individual practitioners about their rationale for polypharmacy, duplication of antipsychotic or antidepressant agents, or prescription of long-term antiparkinson medication. A monthly feedback mechanism was established in the form of a nonthreatening statement to each physician identifying the questionable medication regimen and requesting a response to the issues raised. Subsequently, these staff responses were returned to the committee, which then periodically reviewed them with the entire group of physicians in the regular seminar led by our consultant psychopharmacologist (K.R.).

METHOD

In order to study the impact of our peer review system on psychotropic medication prescription patterns, comparisons were made of two 2-month periods, one in 1973 and one in 1974. We compared the daily medication regimens prescribed in April and May of 1973 with those in October and November of 1974; these two periods, 18 months apart, adequately covered the implementation of the review and seminar process through its initial phase. We simply counted how many medications were prescribed once, twice, three times, or four times a day, omitting all prescriptions for antiepileptics and for minor tranquilizers (such as diazepam) that could have been given appropriately several times a day and therefore would not have been expected to show a decreased daily use. We also excluded all hypnotics since they are usually given once daily at bedtime, and their use was therefore not expected to change.

In a second comparison study, we counted the number of different drugs prescribed per patient and compared the results in April and May of 1973 with both April and May of 1974 and October and November of 1974. Because of staff turnover a number of physicians on the staff in 1973 were only available for the April and May comparison in 1974; we therefore chose to study only the prescriptions of those physicians who were available in April and May of 1973 and 1974. Chi-square analyses were then applied to the collected data.

RESULTS

Table 1 summarizes daily medication regimens for 1973 and 1974. Since the 1973 daily dosage schedules were not routinely recorded on the early prescription summaries or readily available, many original clinical records had to be reviewed. The differences in sample size between 1973 and 1974 can be attributed primarily to an increased patient caseload in 1974 and also to some 1973 data that were not readily available. As can be seen in the table, there was a decrease in the daily frequency of drug administration, with significantly more patients prescribed once-a-day medication compared with divided-dose regimens. The shift is highly significant when compared with the former twice, three times, or four times a day dosage patterns ($p<.001$).

Table 2 summarizes the multiple-drug study and presents the number of drugs prescribed to individual patients during two comparable periods. As can be seen, a significant decrease in polypharmacy had occurred—more patients in 1974 than in 1973 were given only 1 or at the most 2 medications.

When we examined the use of antiparkinson drugs during the same two periods, we found that of 205 patients in 1973, 138 (67%) were treated with a combination of antipsychotic and antiparkinson agents, while in 1974 only 103 of 195 patients (53%) were so treated. These data indicate a significant decrease in the use of antiparkinson agents as a result of the peer review ($\chi^2=8.17$, df=1, $p<.01$).

In a separate recently reported study from the seminar group (10), 39 of 49 outpatients (79%) who had received a phenothiazine plus benztropine mesylate (Co-

TABLE 1
Comparison of Two 2-Month Samples of Daily Medication Schedules 18 Months Apart

		Medication Schedule							
		Once a Day		Twice a Day		Three Times a Day		Four Times a Day	
Year	Total	N	%	N	%	N	%	N	%
1973	650	178	27	199	31	184	28	89	14
1974	1078	593	55	242	22	180	17	63	6

TABLE 2
Comparison of Number of Psychotropic Drugs Prescribed During Two Periods 12 Months Apart*

Number of Drugs Prescribed per Patient**	April–May 1973		April–May 1974	
	Number	Percent	Number	Percent
a. 5–3	102	19	66	11
b. 2	150	28	158	26
c. 1	289	53	381	63

* Includes only psychiatrists providing data from both periods.
** Chi square values: a versus c=15.98, p<.001; a versus b and c=13.78, p<.001; a and b versus c=10.35, p<.005.

gentin) for at least 3 months were able to give up benztropine mesylate with minimum or no sequelae, a result consistent with other reports in the literature.

Finally, we compared multiple versus single antipsychotic drugs prescribed in the same treatment periods and found less polypharmacy with antipsychotic drugs in 1974 than in 1973. In 1973, 134 of 205 patients (65%) received only one antipsychotic agent, while in 1974, 152 of 195 patients (78%) received only one antipsychotic agent (χ^2=7.16, df=1, p<.01).

DISCUSSION

This intramural peer review at the West Philadelphia Consortium has had two major spin-offs. First, our psychiatrists have been trained to be more effective chemotherapists. Each staff psychiatrist is now familiar with the state of the art and is more responsive to its guidelines. As a result, there has been a concurrent improvement in this dimension of the quality of psychiatric care in the agency. However, our data also clearly indicate that the optimum use of psychotropic medications throughout the center's outpatient service delivery system still remains a goal that will require active peer review and continuing education.

Unfortunately, the chemotherapy of our hospitalized patients still appears to be rooted in polypharmacy; this carries over and introduces problems into outpatient medication maintenance. In addition, newly recruited clinicians, even though they may be experienced, seem to practice polypharmacy and to lack an adequate appreciation of the risks of nondiagnosis, reduced phenothiazine efficacy, toxic psychosis, and tardive dyskinesia.

A second major spin-off has been the enhanced development of additional components of a more comprehensive peer review mechanism in the decentralized outpatient programs of the center. These new activities now include review committees on continuity and quality of care. This experience in monitoring prescribing patterns has thoroughly sensitized us to the review requirements of recent legislation authorizing the development of Professional Standards Review Organizations (PSROs) (11) and Health Maintenance Organizations (HMOs) (12). There is no doubt that the new standards for CMHCs currently being developed by the Joint Commission on Accreditation of Hospitals as well as the standards set for a future national health insurance program will demand efficient peer review systems to monitor staff utilization, cost-effectiveness, and quality of care. These currents in clinical and community psychiatry clearly signal increasing third-party review of our service delivery systems, substantiation of the effectiveness of our interventions, and justification of our fees. Medication review is an essential beam in a basic framework for such new accountability.

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Use of the Problem-Oriented Medical Record in Psychiatry: A Survey of University-Based Residency Training Programs

BY EDWARD H. LISTON, M.D.

A survey of university-based psychiatric residency training programs revealed that nearly one-half of them use the problem-oriented medical record (POMR) and that conversion to this system appears to be growing rapidly. Experiences with the method have in general been positive. The author identifies factors associated with successful operation of the POMR and suggests that an integrated, hospital-wide adoption of the system may be crucial.

IT HAS BEEN several years since Weed (1–3) first advocated the abandonment of the traditional source-oriented medical record system and the adoption of a problem-oriented medical record (POMR) system. While most of the literature on the POMR has addressed the medical-surgical fields, there has been a recent growth in reports of the application of this method to psychiatry (4–16). However, the extent to which the system is actually used, the ease or difficulty of implementing it, and its success or failure in psychiatry are unknown on a nationwide basis. Moreover, there does not seem to be any indication in the literature of the degree to which residents in psychiatry are being taught or exposed to the POMR system. This paper is a report of an attempt to answer these questions and to provide an overview of the POMR in American psychiatric training programs.

METHOD

In November 1974 a questionnaire designed to obtain data on the use of the POMR was sent to the 95 U.S. university medical centers and schools of medicine that have psychiatric residency programs. The choice of this restricted sample was dictated by the large number of training programs in psychiatry throughout the country and by the advantages of uniformity for purposes of comparison. In addition, the sample selection was predicated on the assumption that experiences in university centers may influence subsequent general application of the POMR to psychiatric education and practice. Respondents from programs that used affiliated hospitals for training were requested to answer the questionnaire as it applied to that facility that was considered to be their primary teaching hospital. The referent POMR system model was one which included the interaction of a data-base, problem-list, plan for each problem, and progress notes organized by problem.

Dr. Liston is Assistant Professor, Department of Psychiatry, and Chief, Adult Inpatient Services, Neuropsychiatric Institute, School of Medicine, University of California, Los Angeles 90024.

RESULTS

Of the 95 institutions surveyed, 59 (62%) responded within four months. Two respondents did not provide sufficient information for analysis and were excluded from tabulation. Of the remaining 57 programs, 24 (42%) used the POMR system and 33 (58%) did not. Unless otherwise noted, statistical comparisons are based on these two proportions. (A telephone survey of 34 of the nonrespondents revealed that 9 used the POMR and 25 did not. The experiences and opinions of both of these groups in general paralleled those noted in the following sections.)

PROGRAMS USING THE POMR

The earliest introduction of the POMR among the 24 training centers using the system was in 1970, but nearly three-fourths of the centers did not adopt the system until 1973 or 1974. The POMR was used throughout the range of clinical services, most frequently on adult inpatient units; and it was used by all professional disciplines in more than 75% of the programs. Similarly, most of the training centers reported wide application of the system to components of the medical record, especially progress notes, and three-quarters of them employed a master problem list. Twenty (83%) of the psychiatric programs reported concurrent use of the POMR on affiliated medical-surgical services.

In addition to on-the-job training, implementation of the system was assisted in almost all programs by a variety of staff education techniques including seminars, lectures, workshops, films, and instruction manuals. While there was general uniformity in the use of these devices, none was demonstrably more effective than any other. However, there is a suggestion that lectures and seminars were more conducive to successful implementation than other techniques.

Factors Associated with Success of the System

Twenty-one (88%) of the residency programs reported successful operation of the POMR system. Only 1 institution reported that the method failed to operate at all effectively, while 2 others stated that it had been somewhat unsuccessful. Several factors were associated with the operational success of the system (see table 1).

None of the differences between the 2 groups with good and poor success reached statistical significance for any of the associated factors, due in part to the technical problem of very small numbers in the poor success group. However, the marked difference noted in the use of the POMR by affiliated medical-surgical specialties approached statistical significance (p=.06, Fisher exact probability test), suggesting that an integrated, hospital-wide adoption of the system may be necessary for successful operation in psychiatric programs. This observation is supported by a report (17) that the absence of uniform use of the POMR among clinical departments constitutes a major problem in teaching this system to medical students. The data indicate that successful operation of the POMR is positively correlated with both comprehensive application of the system and the requirement that it be used.

Length of operation of the POMR system in a program was also associated with successful experience. All 3 of the relatively unsuccessful programs were less than a year old, compared with only 4 of the 21 successful ones. Comparison of these two proportions by the Fisher exact probability test revealed that their difference was significant (p=.02). This finding may be an indication of the growing pains that are to be expected whenever such changes in established procedures are undertaken. In addition, it supports comments by respondents to the effect that as their experience with the POMR grew, critical evaluation and resistance progressively lessened.

TABLE 1
Factors Associated with Operational Success of the POMR System in 24 Programs

Factor	Percent of Programs Reporting Operational Success	
	Good (N=21)	Poor (N=3)
Use of POMR by affiliated medical-surgical specialties	90	33
Use of a master problem list	81	33
Use of POMR in three or more sections of the medical record	76	33
Use of POMR required on one or more clinical services	71	33
Use of POMR by one or more clinical disciplines required	71	33

TABLE 2
Relationship Between Level of Resistance to the POMR System and Its Required Versus Optional Use in Three Clinical Areas

Clinical Areas of POMR Use	Number of Programs Reporting Resistance		Significance*
	Low	High	
Services			
Required	3	0	p=.05
Optional	0	4	
Disciplines			
Required	18	0	p=.001
Optional	0	3	
Medical record			
Required	9	0	p=.01
Optional	0	3	

* Fisher exact probability test.

Resistance

Predictably, resistance (i.e., noncompliance and opposition) to the implementation and operation of the POMR system was higher in institutions reporting poor success than in those centers whose experiences were more positive. Two training centers reported negligible resistance and 2 indicated marked resistance to implementation of the new system. Examination of their questionnaires revealed that low resistance was associated with required use of the method and, conversely, high resistance with the programs that permitted or encouraged but did not require its use. This finding was true for the use of the POMR in all clinical areas, especially in the professional disciplines (see table 2). The importance of mandatory use of the method is further underscored by the assertion of one respondent that resistance to the system was effectively avoided through "implementation by dictum" combined with instructional discussion groups. In this school, implementation of the POMR was very smooth and operation of the system has been very successful.

Resistance to the POMR method by nonmedical disciplines generally tended to be low, although moderate resistance was reported by 40% of the social workers and marked resistance by 15% of the clinical psychologists. However, the greatest opposition was found among the medical faculties of the institutions; resistance lessened with each successively lower step in the level of medical training (see table 3). Chi-square analysis of this pattern shows it to be highly significant (p=.01).

It is, of course, most likely that this finding reflects the greater general conservatism and rigidity that tend to accompany advancing experience and the fact that license for dissent and opposition tends to be restricted at the lower levels of the medical hierarchy. This observation about the POMR has been noted by others (16–19), and it is apparently a critical consideration in the successful implementation and use of the method. That is, several of the respondents emphasized that commitment to the new system throughout all clin-

TABLE 3
Level of Medical Training and Resistance to the POMR System

Training Level	N	Programs Reporting Resistance*			
		Moderate–Marked		None–Mild	
		N	Percent	N	Percent
Medical staff	22	13	59	9	41
Fellows	8	3	38	5	62
Residents	22	7	32	15	68
Medical students	16	1	6	15	94

* There was a pattern of significantly more resistance as the level of medical training increased ($\chi^2 = 11.34$, df=3, p=.01).

ical disciplines is necessary for its optimal operation and that leadership and direction must derive from the highest levels of institutional authority if smooth transition from the source-oriented to the problem-oriented system is to occur.

Comment

Approximately 90% of the training programs that were using the POMR believed that the system is adaptable to and useful in psychiatry and that its advantages outweigh its drawbacks. In addition, while 32% of the respondents reasoned that the POMR has somewhat better applicability to other specialties, 82% advocated widespread use of the system in psychiatry.

PROGRAMS NOT USING THE POMR

Of the 33 training programs that were not using the POMR system, approximately 80% reported that their faculty members generally were familiar with the problem-oriented concept. Plans to implement the POMR in these schools were mixed; 7 intended to adopt the system and 7 did not, while the remaining 19 were uncertain. (One institution reported using the POMR for a three-month period and then discontinuing it because of its rejection by the hospital's medical records committee.) Many of these programs expressed uncertainty about the usefulness of the POMR in psychiatry, and nearly one-fourth of the respondents were not sure whether the system has any value in medicine in general. However, only 3 schools asserted that the method is of no value whatever in psychiatry.

The respondents' remarks ranged from enthusiastic support for the POMR in psychiatry to denial of its usefulness at all; some respondents took a "wait-and-see" position. A common criticism of the system was that it is less relevant or applicable to psychodynamic and analytically oriented approaches than to behavioral and psychobiological frameworks. One respondent felt that the system is inherently excellent but that the disdainful attitude by physicians toward medical record-keeping must be counteracted in order for the POMR to work.

DISCUSSION

More than half of the psychiatric training programs that responded to this survey were using or had plans to adopt the POMR system and, from the reported dates of implementation, there apears to be a rapidly growing conversion to this method of documenting patient care. Furthermore, experience with the POMR in these psychiatric residency programs has been, on the whole, quite successful, notwithstanding its recent invention and its dissimilarity to the more familiar and traditional source-oriented medical record system.

In addition, the survey results substantiate the observations of others (16–19) that resistance to conversion to the POMR is found principally at the upper levels of the medical hierarchy and that opposition to the system can be avoided or reduced by appropriate leadership and planning and by implementation through means of official policy, preferably on a multidepartmental basis. These observations clearly do not lend support to the notion that the POMR system has little or no applicability to psychiatry; indeed, it is apparent that quite the opposite is true.

The survey methodology was not comprehensive, and many additional important questions remain unanswered. For example, it would be instructive to have learned whether training centers of diverse theoretical persuasions differ in their opinions of and experiences with the POMR system. Furthermore, as one respondent observed, there was no provision for assessing the methods and extent of audit and correctional feedback, which have been seen as crucial to the survival of the problem-oriented system (10). Nonetheless, this survey indicates that use of the problem-oriented medical record system in psychiatry is not merely a transitory and fashionable phenomenon. Moreover, the fact that this new method is now being taught in approximately 80% of all U.S. medical schools (17) forecasts a progressive extension of the POMR system throughout all levels of American medicine, which will continue to embrace and influence psychiatric education and practice.

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Low Urinary Dopamine and Prediction of Phenothiazine-Induced Parkinsonism: A Preliminary Report

BY THOMAS J. CROWLEY, M.D., CHARLES O. RUTLEDGE, PH.D., MARGARET M. HOEHN, M.D., MARY ANN STALLINGS, R.N., M.S., AND SEYMOUR SUNDELL, M.D.

Psychiatric patients who excreted larger amounts of urinary free dopamine before treatment were significantly more likely than patients excreting smaller amounts to develop parkinsonian side effects during moderate-dose trifluoperazine therapy. If this finding is replicated, urinary free dopamine determinations could prove valuable in indicating which patients should receive those antipsychotic drugs least likely to produce extrapyramidal side effects.

MOST ANTIPSYCHOTIC (neuroleptic) drugs, including the phenothiazines, produce extrapyramidal side effects, the most common of which is the parkinsonian syndrome of hypokinesia, rigidity, and tremor. The incidence of these parkinsonian reactions varies with the dose and type of drug and perhaps with the patient's age and duration of treatment (1, 2).

In idiopathic Parkinson's disease the nigrostriatal pathway degenerates, and dopamine, the proposed neurotransmitter in this pathway, is then present in amounts insufficient to inhibit neurons originating in the striatum (3–5). The severity of akinesia in idiopathic Parkinson's disease directly correlates with the deficiency of brain dopamine (5). Compared with normal controls, parkinsonian patients also excrete reduced levels of dopamine in the urine (6–8). Urinary levels of free (unconjugated) dopamine appear to correlate negatively with the presence of parkinsonian akinesia (8) and, according to our observations, with its severity. Thus considerable evidence suggests that in Parkinson's disease the levels of free dopamine in the urine may at least reflect differences in brain dopamine metabolism, even though much of this free dopamine may originate peripherally.

Parkinsonism induced by antipsychotic drugs is thought to result from drug blockade of dopamine receptors, rendering striatal neurons less responsive to dopamine inhibition (9, 10). These drugs may also reduce the release of dopamine within the basal ganglia (11). We hypothesized that those psychiatric patients with low urinary dopamine levels before treatment would be at greatest risk for developing parkinsonian side effects from antipsychotic drugs; this article reports a preliminary test of that hypothesis.

When this work was done, the authors were with the University of Colorado Medical Center, 4200 East Ninth Ave., Denver, Colo. 80220, where Dr. Crowley is Associate Professor of Psychiatry, Dr. Rutledge was Associate Professor of Pharmacology, Dr. Hoehn is Assistant Professor of Neurology, Ms. Stallings is Research Nurse in Psychiatry, and Dr. Sundell is Resident in Psychiatry. Dr. Rutledge is now Professor and Chairman, Department of Pharmacology and Toxicology, School of Pharmacy, University of Kansas, Lawrence, Kans.

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METHOD

Criteria for admitting patients to the study were 1) voluntary admission for acute psychiatric treatment to the Colorado Psychiatric Hospital or the Denver Veterans Administration Hospital, 2) a clinician's decision that an antipsychotic drug was needed but that the patient's degree of agitation permitted deferral of treatment for 2 days during initial evaluation, 3) no use of antipsychotic or antidepressant medication by the patient for at least 11 days before entering the study, 4) no recent heavy use of alcohol or other drugs of abuse, and 5) the patient's informed consent to the study procedures.

The subjects were 8 men and 8 women ranging in age from 20 to 57 years (mean=31). Diagnoses according to *DSM-II* (12) criteria were schizophrenia, N=14; psychotic organic brain syndrome, N=1; and obsessive-compulsive personality disorder, N=1. This was the first psychiatric admission for 7 patients and the second or third for 6; 3 patients had had four or more admissions.

The severity of parkinsonian signs was scored from 0 to 100 points on a standardized neurological examination (13) before antipsychotic drug administration and then about 3 times a week for 2 weeks or until unmistakable parkinsonian signs developed.

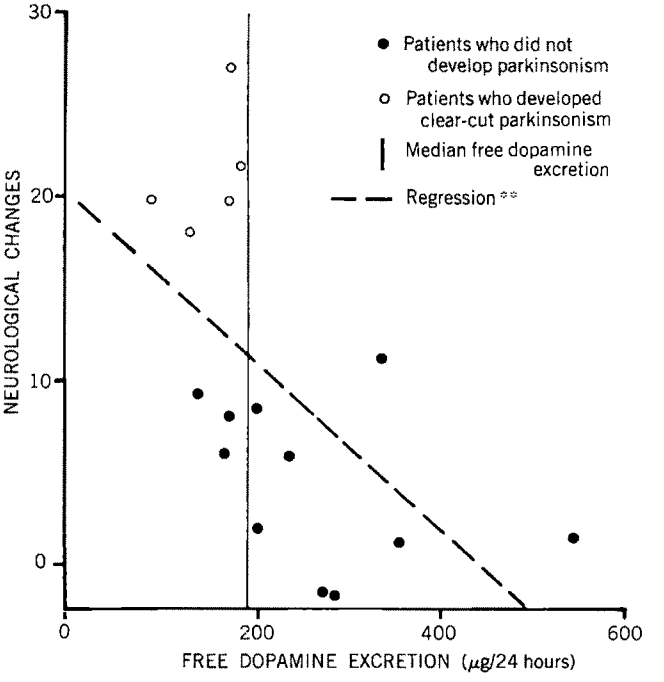
Pretreatment 24-hour urine samples were collected once (1 patient), twice (14 patients), or three times (1 patient) and were analyzed in duplicate for free and conjugated dopamine by techniques described previously (14). The means of the daily values were used to calculate each patient's dopamine excretion rate. The biochemical technicians and neurological examiners were unaware of one another's data.

Patients received oral trifluoperazine HCl (Stelazine) in a low-dose schedule increasing from 0 at a rate of 4 mg/day (15 patients) or in a high-dose schedule increasing by 8 mg/day (1 patient); the high-dose schedule was used when the treating clinician decided that the patient's condition warranted a rapid increase in dose. Daily increases stopped when the dose reached 40 mg/day or when clinical improvement or side effects occurred. The treating physicians could modify the schedules when this was clinically warranted. No antiparkinsonian drugs were administered during the study. Correlations were analyzed by the Pearson product-moment method.

RESULTS

In figure 1 the difference between the predrug neurological score and the highest (most pathological) drug-period score for each patient is plotted against the predrug excretion of free dopamine. All 5 of the patients who developed obvious parkinsonism changed by 18 points or more. One patient who changed by 27 points had severe parkinsonian signs. The other 11 patients changed by less than 11 points and did not develop parkinsonism; their extrapyramidal signs were transient, less severe, and less definite.

FIGURE 1
Pretreatment Free Dopamine Excretion and Neurological Change with Trifluoperazine Therapy*



*Higher scores indicate the development of more marked extrapyramidal signs.
**$Y=a+b_{xy}X$.

The mean 24-hour free dopamine excretion was 225.4 μg (SE_M=28) and the median was 190. The 5 patients who developed parkinsonism were among the 8 patients who excreted less than 190 μg/24 hours of free dopamine. None of the 8 patients who excreted more than that amount developed the syndrome. Parkinsonism was significantly more common among the patients excreting less than 190 μg/24 hours ($p<.025$, Fisher's exact test). There was also a significant negative correlation between the change in neurological score and the pretreatment free dopamine excretion ($r=-.56$, $p<.025$).

Changes in neurological scores did not correlate significantly with total (free plus conjugated) dopamine excretion, age, sex, or the patient's trifluoperazine dosage on the day of his/her maximum neurological score during the drug period (mean=.39 mg/kg, SE_M=.04). Neurological change was also not significantly correlated with the total dose received by each patient over the 2 weeks of the study (mean=4.43 mg/kg, SE_M=.26) or with the maximum daily dose achieved by each patient (mean=.47 mg/kg, SE_M=.03). It was not possible to determine accurately the previous phenothiazine exposure of these patients; we used the number of previous hospitalizations as a rough indicator of previous treatment and found no significant correlation with either neurological change or predrug free dopa-

mine excretion. Furthermore, free dopamine excretion did not correlate significantly with the number of days the patient had received the hospital diet before the first urine collection.

An analysis of variance (15) comparing the dopamine excretion rates among patients, the rates between successive days for each patient, and duplicate assays on each daily sample revealed substantial interpatient differences ($p<.005$). Differences between days approached significance ($.05<p<.10$). Differences between duplicate assays were not significant. Moreover, there was no significant systematic change in the measured dopamine levels across the months in which the determinations were performed (16).

DISCUSSION

Urinary dopamine probably originates from dietary sources (7, 17) and from peripheral organs (8), as well as from the brain. In fact, the central nervous system (CNS) contribution to urinary dopamine may be relatively small. In the rat an 80% decrement in brain dopamine results in a nonsignificant decline (less than 15%) in urinary dopamine (18). Whatever its source, the well-established reduction in urinary dopamine excretion among parkinsonian patients (6, 8), which is observed under either special or ad lib diets (7), suggests that dopamine excretion in some way mirrors CNS dopamine metabolism. Barbeau (8) argued that the pathology of parkinsonism is not limited to the nigrostriatal pathway and that this is a "systemic" disease with widespread abnormalities of dopamine metabolism. This view is supported by recent evidence that patients with Parkinson's disease show impaired uptake of dopamine in blood platelets (19). The concept may be further supported by our observation that psychiatric patients excreting less than 190 μg/24 hours of free dopamine were significantly more likely than other patients to develop extrapyramidal disorders during subsequent treatment with trifluoperazine.

Reported levels of dopamine excretion vary among different subjects and different laboratories (6–8). Our range was well within limits reported elsewhere, and we showed statistically reliable interpatient differences in dopamine excretion. These differences correlated with, and thus predicted, the subsequent development of parkinsonian signs.

Since dopamine excretion may vary with diet, it is possible that by chance alone those patients with the greatest tendency to develop phenothiazine-induced parkinsonism also happened to be those on diets relatively poor in catecholamines, producing a spurious relationship between dopamine excretion and extrapyramidal signs. Similarly, phenothiazine absorption after oral ingestion could vary and, again by chance alone, those patients who excreted less dopamine also could have been those who happened to absorb more trifluoperazine, resulting in higher blood levels and an increased risk of extrapyramidal symptoms. We cannot rule out such chance occurrences, but their probability is less than 3 in 100 according to our statistical analyses.

Might diagnostic or treatment differences have produced the differences in neurological change? Two patients were not schizophrenic. The patient diagnosed as an obsessive-compulsive personality excreted low amounts of dopamine and became parkinsonian; the patient with an organic brain syndrome excreted over 300 μg/24 hours and did not become parkinsonian. Thus, in terms of dopamine and neurological change, these two nonschizophrenic patients responded similarly to the rest of the group. The treating clinicians decided to increase the dose of 1 patient more rapidly than the others, using the "high-dose" schedule. This patient had the lowest dopamine excretion and was among the 5 patients who became clearly parkinsonian. Thus it is possible that dose differences contributed to the final result in this case. However, for the group of patients, the neurological change did not correlate significantly with the total dose received during the study, the highest daily dose received by each patient, the dose received on the day of each patient's highest neurological score, or with a rough estimate of past drug exposure. Furthermore, neither age nor sex was correlated with drug-induced parkinsonism in this sample, although these factors appear to be important in older, more chronic inpatient populations (1, 2). Only pretreatment free dopamine excretion correlated with parkinsonian changes.

If replicated, the correlation we have reported may be of value in indicating which patients should receive those antipsychotic drugs least likely to induce parkinsonism. Its eventual application, however, would depend on the establishment of standard excretion values of dopamine and its metabolites for populations differing in age, prior and recent drug exposure, and planned therapeutic dose. Higher doses, for example, may increase the incidence of extrapyramidal disorders (20); the critical dopamine level for developing parkinsonism might have been other than 190 μg/24 hours if somewhat higher doses or a more potent drug such as fluphenazine (Prolixin) had been administered.

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Sex Bias in Psychotherapy: A Failure to Confirm

BY STEPHEN I. ABRAMOWITZ, PH.D., HOWARD B. ROBACK, PH.D., JOSEPH M. SCHWARTZ, M.A., AMY YASUNA, CHRISTINE V. ABRAMOWITZ, PH.D., AND BEVERLY GOMES, M.A.

The authors examined the questions of sex-related countertransference and bias in psychotherapy by asking 65 male and 57 female experienced group therapists for clinical reactions to case materials of a bogus outpatient who was designated either male or female. They found that varying the patient's gender produced only small differences in the therapists' responses, and conclude that sex-related countertransference problems may not be as prevalent as had been previously thought.

THE REEMERGENCE of the women's rights movement has intensified professional concern about the potential for covert acts of sex discrimination perpetrated under the banner of mental health care (1). The cumulative weight of experimental and archival research with diverse practitioner and clinical populations has not succeeded in allaying this concern (2, 3).

A simple but clever study by Masling and Harris (4) yielded evidence that the clinical situation can be a source of voyeuristic need gratification for the professional. Male clinical psychology graduate students were found to administer more sexual-romantic cards from the Thematic Apperception Test (TAT) to female than to male examinees, and to extend testing sessions with women by presenting more cards to them. Female trainees were apparently unaffected by the examinee's sex. In a recent clinical analogue, clinicians rated psychological adjustment using the case material of college students identified as male or female and as po-

Drs. Stephen and Christine Abramowitz are Assistant Professors of Psychology, Department of Psychiatry, Sacramento Medical Center, University of California, Davis, Sacramento, Calif. Dr. Roback is Associate Professor of Psychiatry (Psychology), Vanderbilt University School of Medicine, Nashville, Tenn., where Mr. Schwartz, Ms. Yasuna, and Ms. Gomes are doctoral candidates, Department of Psychology. Address reprint requests to Dr. S. Abramowitz, East Sacramento Community Mental Health Center, 9555 Kiefer Blvd., Sacramento, Calif. 95826.

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litically liberal or conservative. The liberal woman received the most censorious evaluations, especially when the clinicians were relatively conservative (5).

Results from two investigations (6, 7) tentatively implicate the practitioner's sex-role attitudes as a source of countertransference in case selection and termination. In the first study, 11 male counselors at a university counseling center carried case loads having an approximately equal sex distribution, whereas 4 female counselors' case loads included a disproportionate number of women. Cultural bias against placement of women in dominant social roles over men was offered as an explanation (6). Similar results, as well as more subtle manifestations of sex-role-related countertransference, were subsequently obtained at both a psychiatric and a psychological training facility (7). Women were seen for more sessions than men when the psychotherapy trainee was a man. In addition, female therapists at the psychiatric clinic saw proportionately fewer single or divorced (sexually unattached) patients than did male therapists, and the 2 single female therapists saw a smaller percentage of sexually unattached men than the 1 married female therapist. We surmised (7) that conventional sex-role expectations may prime male therapists to prolong, and their female colleagues to avoid, treatment situations likely to arouse sexual curiosity.

Evidence that sex-role-linked countertransference influences the treatment careers of group therapy patients is limited to a single, tangential finding (8). One female and 8 male psychiatrically trained clinicians performed intake interviews and sometimes conducted the treatment of patients at a university-affiliated health center. The interviewers referred proportionately more female than male applicants to individual than to group psychotherapy, reinforcing the notion of voyeuristic need gratification.

The present study was undertaken to determine if the notion of sex-role countertransference could be generalized to conventionally trained, experienced group therapists. To ensure greater sample representativeness than is usual in studies of political bias, a clinical analogue method adapted from Strupp (9) was used. Male and female practitioners were presented with written case material of a bogus group therapy outpatient designated as male or female. They were then asked to indicate their clinical reactions to this person and to complete a traditional moralism questionnaire.

METHOD

Subjects

Prospective subjects comprised a sex-stratified random sample of 600 members of the American Group Psychotherapy Association (AGPA). The AGPA is a multidisciplinary professional organization whose membership requirements include extensive supervised group therapy experience. Usable data were returned by 65 male and 57 female therapists (slightly over 20% of the target population). Demographic information was obtained from the AGPA directory (10) and from the respondents.

Of the research participants, 53% described themselves as psychodynamically oriented, 21% expressed a preference for a client-centered or Gestalt philosophy, and only 2% favored a behavioral model. An eclectic approach was espoused by 24% of the sample. An overwhelming proportion of the respondents had undergone either analysis or other uncovering therapy, and most had been group therapists for more than 10 years. Comparison of 40 respondents (20 men and 20 women, randomly selected) with the same number of similarly selected nonrespondents revealed no significant differences with regard to highest scholastic degree (a possible index of professional commitment) and membership in one of the affiliate societies of the AGPA. These sampling data suggest that the research participants were roughly representative of those originally surveyed.

Instruments

Six items tapped the therapist's clinical impressions and degree of liking of the stimulus patient. Questions were counterbalanced, and all response continua were 4-point Likert scales. Subjects were also presented with a list of 19 therapist reactions adapted from the Wile Group Therapy Questionnaire (11). They rank-ordered 3 responses that they would have been most likely to use in the therapy incident involving the bogus patient. The 19 response alternatives represented a broad range of individual- and group-directed categories, from supportive and reassuring to confrontative and interpretive (see appendix 1). First-, second-, and third-ranked choices were scored 3, 2, and 1, respectively. The 12-item, fully counterbalanced, 7-point Likert traditional moralism scale (12) was used to measure traditionalism of social values. Although reliability (coefficient alpha=.58) and discriminability (SD=8.1) were somewhat less robust than reported by the scale's developers (12), the levels obtained compared favorably with those generated by the same and similar instruments used in other surveys of professional samples. Respondents whose raw traditional moralism scores fell above the median were designated as relatively traditional, and those whose scores fell below it as relatively nontraditional.

Procedure

A detailed clinical profile of a group therapy outpatient, including information about family background, employment history, marital adjustment, presenting problems, and psychological test results, was prepared. Sexual performance conflicts and hostile-dependent dynamics were prominent. The case protocol concluded with a description of a representative therapy incident, in which the patient was unusually silent for the first part of a meeting and then began to cry.

The clinical profile was designated as belonging to a man or a woman by systematic reversal of pronoun

gender. The male- or female-identified case materials were mailed with the related response sheets to prospective subjects, who were led to believe that the object of the study was to compile normative data on group therapists' clinical opinions. A sealed debriefing letter that was not to be opened until completion of the simulated clinical tasks was also included.

RESULTS

Data were examined and analyses of variance were performed according to the two-level factors of therapist sex, therapist values, and patient sex. Approximately one-half of the therapist judgment and response dimensions did not produce any differences beyond the .10 level: determinations of social adjustment, mental disturbance, and attraction, and response preferences for silence, group-directed, reassurance-approval, structure, attack, member feeling, leader experience, personal life, past and parents, and nonverbal.

Differences according to therapist sex were found with regard to expressed empathy for the patient ($p<.05$) and the therapeutic strategies of group dynamics question ($p<.05$), psychodynamic interpretation ($p<.05$), behavior change ($p<.05$), clarification-confrontation ($p<.05$), and subtle guidance ($p<.10$). In every instance except clarification-confrontation, female therapists were associated with a higher level of the particular attribute.

Four clinical decisions were related to therapist values. Less traditional respondents were more lenient judges of emotional maturity ($p<.05$) and tended toward more deployment of clarification-confrontation ($p<.10$); more traditional respondents were more partial to behavior change ($p<.05$) and slightly more partial to subtle guidance ($p<.10$). Reliance on subtle guidance tended to be strongest among relatively traditional female clinicians ($p<.10$).

The impact of varying the patient's gender was surprisingly slight. The patient received a better prognosis ($p<.05$) and elicited slightly more empathy ($p<.10$) when identified as a woman. In addition, the group atmosphere option was preferred more when the stimulus person was identified as a man ($p<.05$). The degree to which the patient's sex influenced the reactions of therapists depended on the latter's own sex in three instances. Female therapists tended to empathize more strongly with the female-designated than with the male-designated patient; they showed more empathy than male therapists did ($p<.10$) regardless of how the case materials were labeled. Women used behavior change more often with the female-identified than with the male-identified stimulus person and more often in general ($p<.10$) than men did irrespective of the patient's sex. Interestingly, role playing was slightly more popular in the case of opposite-sex than same-sex therapist-patient dyads ($p<.10$).

Relatively traditional clinicians preferred leader feeling slightly more than nontraditional clinicians when the patient was identified as a man, whereas the reverse held when the patient was identified as a woman ($p<.10$). Relatively traditional therapists also were more partial to behavior change with the female than with the male patient, and than nontraditional therapists tended to be ($p<.10$) irrespective of the patient's sex. The female patient was more likely than her male counterpart to elicit a behaviorally focused reaction from traditional female therapists and than either stimulus patient was from any other therapist subgroup ($p<.05$).

DISCUSSION

Previous research (2) supported the assertion that the clinical enterprise is susceptible to the proclivities of the mainstream culture that sanctions it (13). Our data suggest that the amount of sociopolitical bias among experienced group therapy practitioners is comparatively small. The impact of the patient's sex on the therapists' reactions was, for example, quite mild. This outcome may be a function of the respondents' collective clinical wisdom, their predominantly analytically oriented training that presumably included awareness of countertransference phenomena, their sensitization to the recent outpouring of professional concern over possible sex bias in psychotherapy, or limitations of the experimental procedures. Generalization is always a hazard when an analogue task requires professionals to participate vicariously in a clinical encounter. Manipulation of the sex variable within the context of a written clinical profile may have been a subtle cue, and the professional sample may have been too homogeneous in social values to permit a meaningful differentiation. Likewise, a substantial number of subjects may have suspected the true goal of the research despite precautions taken to the contrary, and monitored their responses accordingly. However, positive findings from similarly conceived studies tend to reduce the cogency of such reservations.

Certain isolated patterns in the data offer some support for the notion of sex-role-related countertransference (7). Female rather than male therapists were less disposed to use an overtly directive response (clarification-confrontation) but more disposed to use certain responses whose directiveness is institutionalized by theory (psychodynamic interpretation, behavior change) and certain covertly controlling responses (subtle guidance, group dynamics question). This configuration parallels conventional sex-role prescriptions for assertion and suggests that professional training and status may not guarantee insulation of therapeutic reactions from incorporated sex-role ideals.

Enhanced interpersonal sensitivity, an often cited outcome of feminine socialization, may differentially equip women with a valuable professional commodity. Both in this study and in an earlier study of psychother-

apy trainees (14), men could not marshal as much empathy for a stimulus patient as could women. This questionnaire differential could, however, also be accounted for in terms of sex-role pressure for women to present themselves as nurturant.

Predilection toward use of role playing with an opposite-sex patient is intriguing, all the more so because an identical finding for another Gestalt-type response (nonverbal) nearly achieved statistical respectability ($p<.12$). Research tends to confirm that encounter group participants are aware of an element of sexual play in such exercises (15). Whether or not therapists' preferences for these procedures are partly determined by their voyeuristic needs is an issue that should be tested among sensitivity group leaders.

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APPENDIX 1
Group Therapist Response Alternatives from Wile Group Therapy Questionnaire (11)

Category	Response
1. Silence	Do nothing.
2. Group directed	Ask why no one has said anything about the fact that someone is crying.
3. Reassurance-approval	Express concern and reassurance.
4. Subtle guidance	Continue as if nothing out of the ordinary were happening.
5. Structure	Suggest that it might be more useful for him/her to talk than just to cry.
6. Attack	Accuse him/her of putting on a show.
7. Member feelings	Ask about feelings (e.g., encourage him/her to give words to his/her feelings; ask the members how they feel about his/her crying).
8. Leader feelings	Say how you are feeling (e.g., moved, embarrassed).
9. Leader experience	Share a similar experience in your own life.
10. Clarification-confrontation	Ask him/her why he/she is crying.
11. Group dynamics question	Ask them to describe what is happening at that meeting.
12. Group atmosphere	Say that someone in the group is crying.
13. Group dynamics interpretation	Describe his/her crying as an act of involvement in the group and a willingness to share his/her more private feelings with them.
14. Psychodynamic interpretation	Suggest that he/she may feel that the only time people are willing to listen and pay attention to him/her is when he/she is crying.
15. Personal life	Encourage him/her to talk about the events in his/her life that may be upsetting him/her.
16. Past and parents	Encourage him/her to relate what he/she is feeling to his/her family relationships and past experiences.
17. Behavior change	Encourage him/her to talk about his/her difficulties in behavioral terms.
18. Nonverbal	Use a nonverbal procedure to explore the rich emotional experience of crying.
19. Role playing	Ask him/her to role play the situation that he/she is crying about.

Anticipating the Impact of Public Law 93-641 on Mental Health Services

BY ARNOLD MILSTEIN, M.D., M.P.H.

Implementation of the new federal law setting up Health Systems Agencies (HSAs) on a regional basis offers many opportunities for participation by psychiatry. On the basis of interviews with mental health service providers, planners, and citizen representatives, the author formulated an inventory of the law's potential impacts, grouped around six general themes. He discusses the positive aspects of each, along with possible problems and hazards.

THE NATIONAL HEALTH PLANNING and Resources Development Act of 1974 (PL 93-641) is expected to have a major effect on the delivery of mental health services. Regarded as the federal government's most significant health initiative since the passage of the Medicare law in 1965, PL 93-641 establishes a coordinated health planning network at the regional, state, and national levels. The network replaces three major federal health programs: Comprehensive Health Planning (CHP), Regional Medical Programs, and the Hill-Burton Act authorizing the construction of hospitals. In addition to planning, this new network is charged with responsibilities for regulation and development of resources.

The cornerstone of the network is the Health Systems Agency (HSA), a regional health planning unit run by a governing body with a professional staff of at least five and a consumer majority. Each HSA will assess the existing health services system in its area, prescribe short- and long-range plans for the system's improvement, and use its limited authority, development funds, and technical assistance to bring its plans to fruition. Included in the HSA authority is the power to approve or disapprove federal grants under the Community Mental Health Centers Act on the basis of their harmony with HSA plans.

An HSA will be designated for each of the 202 health services areas approved by the Department of Health, Education, and Welfare in August 1975. Generally, HSAs will be either private nonprofit community agencies or public agencies under the auspices of single or legally joined unit(s) of local government whose jurisdiction coincides with the boundaries of the health service area.

Implementation of the law, now in the formative stages, offers many opportunities for participation by the psychiatric community. An awareness of the law's potential impact on mental health services will provide not only an antidote to "future shock" but also a basis for increasing the effectiveness of such participation. The following inventory of the law's potential consequences is intended to serve these purposes.

I constructed this report from interviews with 40 individuals representing the major groups at the interface between mental health services and HSAs, including citizens representing mental health interests, public and private mental health service providers, and mental and general health planners.

The types of impact that were predicted may be grouped around six general themes: 1) joint planning of mental and physical health services, 2) the relationship between HSAs and existing mental health service planning mechanisms, 3) emphasis on evaluation of mental health services, 4) more consumer input into mental health services planning, 5) increased availability of technical expertise for planning mental health services, and 6) emphasis on regional planning.

JOINT PLANNING OF MENTAL AND PHYSICAL HEALTH SERVICES

A combined planning approach promises three principal benefits. First, the clearly established clinical relationship between physical and mental health services (1) can be better exploited. Second, the wider political support base enjoyed by physical health services represents a new constituency for mental health services. Third, joint planning may hasten the development of a partnership between physical and mental health services necessary for creating generic primary care systems to treat "human problems" whose causes and solutions defy the prevailing boundaries of our health care system.

However, some broad dangers related to combined planning are anticipated. A narrow, traditional view of health and health care (2, p. 1217) may dominate, leading to several secondary threats. Inappropriately nar-

Dr. Milstein is Mental Health Career Development Fellow, National Institute of Mental Health, and Special Assistant to the Director, Division of Resource Development, Public Health Service, Region IX, Department of Health, Education, and Welfare, 50 Fulton St., San Francisco, Calif. 94102.

The views expressed herein are those of the author and do not represent the official positions of the National Institute of Mental Health or the Public Health Service.

row outcome criteria and a constricted range of interventions could be foisted on mental health services. These would not only fail to reflect the complex systems in which mental health and illness are empirically defined (3), but would also ignore much that has been found desirable about the community mental health movement (4, p. 48). Another secondary threat lies in the tendency of the traditional viewpoint to underrate psychological problems and shortchange their treatment. This threat would be accentuated if funds for physical and mental health services were pooled. Finally, health care may be based in institutions, thus endangering the gains made by the community mental health movement and saddling mental health services with an inappropriate share of medical center overhead costs, which tend to be high. A second threat emerges if sufficient funds to enable adequate planning for mental and physical health are not appropriated. If a funding famine materializes, short-rationing of funds for mental health planning may be less visible and therefore more politically feasible than it would be if mental health planning funds were identifiable as a discrete item.

HSAs AND EXISTING PLANNING MECHANISMS

Important benefits are expected to arise from the relationships between HSAs and state and county health and mental health departments and community mental health centers (CMHCs). Duplication of common planning tasks can be avoided, and a common planning format can be employed. Reason, rather than tradition or territoriality, can dictate the allocation of problem analysis and intervention functions to various planning levels and sectors.

Three principal dangers may be foreseen. First, the fact that nonfederal mental health monies are beyond the jurisdiction of the HSA may undermine coordinated planning among highly interdependent systems. Indeed, it is a meaningless exercise to examine CMHC functioning in isolation from state mental health policy in most regions. Second, state and local mental health planners, anxious or angry over their loss of autonomy to the HSAs, may engage in wasteful competitive planning. Finally, the mental health planning mechanisms already (or potentially) functioning well in such states as California and Arizona may be subject to wholesale replacement by the HSAs, or there may be indifference to their further refinement.

EVALUATION

To those who believe that the technology for meaningful mental health service evaluation exists and that this process is financially feasible, PL 93-641 represents an opportunity to better recognize effective components of mental health services. Those with a more pessimistic view of the technology of evaluation fear the creation of incentives for meaningless evaluative criteria. A second hazard derives from the fact that the outcomes of mental health services are inherently less tangible than those of physical health services. As evaluative efforts increase, mental health services may begin to appear less effective than physical health services. This appearance may be based largely on differences in ability to measure rather than on the actual value of the service. With a shared funding pool, the position of mental health programs in competing for funds may be compromised and the danger of their being shortchanged may increase. Another danger is that the informational demands of increased evaluation efforts may threaten the privacy of psychotherapeutic relationships.

CONSUMER INPUT

Since HSA governing board members may be better able to empathize with consumers of mental health services (and in some cases will themselves be consumers), the acceptability and utility of the services should increase. The range of intervention options is also likely to increase with consumer involvement. This prediction is based on two arguments. First, the professional's intervention horizon is inevitably narrowed by his own professional perspective as well as by the need to protect his turf, maintain referrals, and build interagency allegiances, whereas the consumer's is not. Second, while treatment options that are highly visible in the community (e.g., halfway houses) may often be volunteered by citizens themselves, the same proposals may be quashed by the community when they appear as an initiative taken by professionals or agencies (5). A final benefit is that well-informed citizen majorities on governing boards can provide a relatively neutral and democratic mechanism for resource allocation since they tend to have less intense specialty, agency, or program loyalties.

Several dangers are anticipated in this context. The consumer viewpoint may be inadequately represented—as it has been on some CHP and CMHC governing and advisory boards—because of intimidation of consumer representatives by health professionals. The heralded intent of PL 93-641 to solve this problem may lull the general public into trust (which may prove to be unfounded) that these threats to democratic planning are behind us and thus may deter efforts to strengthen consumer participation. Ironically, strong consumer participation can itself pose a threat. Consumer board members unfamiliar with social planning may fail to make appropriate distinctions between overall setting of priorities and internal management functions, at a significant cost to both processes.

TECHNICAL EXPERTISE FOR PLANNING

There are many opportunities for coordinated and effective social planning in the HSA-mental health serv-

ices interface. In relation to mental health services, the benefits could range from more rapid progress toward societal goals that are contingent on effective mental health programs to more prosaic results like better coordination and cooperation among diverse mental health service programs. Other benefits include a better data base for decision-making and improved tools for internal management at program and agency levels.

The greater availability of technical planning resources is not without potential problems. Citizens' lack of education and experience in social planning may limit their ability to use the sometimes complex and technical outputs of planning staffs meaningfully (6). Problems of this type also arose in regard to CHP. The abstract, intangible nature of mental health services compounds the problem of noncomprehension by members of HSA governing boards.

EMPHASIS ON REGIONAL PLANNING

The health service areas mandated by PL 93-641 will be an appropriate scale (2, pp. 294-296) for the understanding and solution of important problems in mental health service delivery (e.g., treatment of individuals requiring long-term psychiatric hospitalization). Also, given a fixed amount of dollars available for health planning, the regional scale increases the financial feasibility of having specialty area planners on HSA staffs. In many health services areas it will be possible to employ a mental health planner. In addition, the larger the area for which plans are developed, the larger the potential political constituency for the plan and the more difficult it will be for policy makers at state and local levels to ignore mental health needs.

However, several liabilities appear to be associated with regional planning. Problems or solutions whose boundaries are those of a county, city, or catchment area may not be appropriately delegated by an HSA. This could occur for several reasons, including a misperception of the most appropriate scale on which to approach an issue or an HSA's overzealous guarding of its legal jurisdiction. Demoralization and apathy could plague existing governing or advisory boards of CMHCs, which might see HSAs as a threat to their sense of autonomy. An important source of input from experienced people working at the subregional level could thus be lost to the larger process of regional planning. A third anticipated danger is that in politically conservative regions, small, poor communities could be deprived of CMHC funding by a regional governing board that is averse to committing local tax revenues to support a CMHC as the levels of federal grants decline. Since the federal government is often the only available source of fiscal support to initiate these services, this "tyranny of the majority" could bring about serious inequities in the mental health service system. A fourth danger may be subsumed under the label "complex system problem." Mental health services are already highly related to many other human service components at several levels (e.g., county probation departments, city jails, state Medicaid programs, and state rehabilitation programs). Adding a new level of aggregation and a new entity with which to relate will definitely increase the complexity of the human service system while offering only a possibility of improving its functioning.

CONCLUSIONS

An awareness of the major forces that shape the conduct of an individual's life is perhaps the psychiatrist's most common "prescription." Public policies are exerting an increasing impact on the practice of medicine. Psychiatrists would thus do well to recommend the same elixir for themselves in relation to future external forces as they do for patients in relation to their past and current internal forces. The anticipated impacts I have outlined provide an initial assessment of major opportunities for, as well as dangers to, the successful delivery of mental health services.

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LETTERS TO THE EDITOR

A Modest Proposal

SIR: I wish the assistance of the *Journal* in conducting a market survey among potential buyers of a new product that promises to be of great use to all practicing psychiatrists, especially those in California.

While reflecting recently upon the plight of our West Coast brethren, a colleague and I came up with a method for notifying potential victims of psychiatric patients who make homicidal threats within earshot of a psychiatrist. Before attempting to sell our idea to Hallmark Cards, we would like to test consumer reaction. Here is our idea.

A tasteful, attractive card bearing the legend "Someone Is Thinking Of You" on the front would, on the reverse side, inform the reader that "A Certain Person has told Dr. _____[space to be imprinted with the psychiatrist's name, office number and provider code] that he wishes to __intends to __or has tried, failed, but is about to try again to __kill you." The sender would check the appropriate space.

As your readers can see at once, this method not only delivers the warning required by the new state law in California but discharges this obligation in a tactful and uniform manner. As an additional service to customers, a detachable card would be retained by the psychiatrist for his records in case he was challenged on compliance with the law.

We hope that if our idea catches on greeting card displays in drugstores and the like will soon have a section marked "Death Threats" (perhaps somewhere between "In Memoriam" and "To My Son the Doctor") displaying a selection of styles.

We would appreciate letters from *Journal* readers telling us how they like this idea. Also, we would like to mail out a test batch to members of the California legislature, since, at a recent medical meeting, one of us distinctly overheard a homicidal threat uttered by a physician who gave every evidence of mental instability, a threat that seemed to be aimed at any or all members of the body that passed the law in question.

DOUGLAS A. SARGENT, M.D.
Thanatos Greeting Card Project

Insuring Psychiatrists

SIR: Several years ago, many of us wondered why the Association decided to change insurance carriers for the group malpractice insurance with a resulting tripling of premiums. Despite the thousands of words that have been written and spoken on the subject, the reasons for this change have not been made clear. Shortly thereafter, it became apparent that some of the increased premium was going to pay for the massive advertising campaign by the Joseph A. Britton Agency, which included sending representatives to the APA annual meetings in Hawaii and Anaheim and placing full-page ads in APA publications.

The final blow has now been struck with the publication of "Professional Liability Insurance and the American Psychiatrist" by Chester L. Trent, M.D., and William P. Muhl (December 1975 issue). Although the *Journal* is supposedly a scientific publication, this article is nothing more than further advertising for the insurance carrier and for the APA-sponsored group policy.

I object to the premium dollars being spent on advertising, and I object even more strenuously to free advertising in the pages of the *Journal* under the disguise of a scientific or informative article.

GEORGE BAJOR, M.D.
Encino, Calif.

SIR: I think it curious indeed when a representative of the American Psychiatric Association teams up with a representative of the insurance industry to explain and justify APA's position on professional liability coverage for American psychiatrists. I am pleased that such coverage is available to APA members like myself, but in no way am I convinced that Dr. Trent and the Committee on Member Insurance and Retirement Plans represented their constituency well by approving outrageous premium hikes in 1975.

This article has increased my doubts. It reveals the very low incidence of malpractice suits against psychiatrists and also the relatively low dollar awards in successful suits. I feel it is specious to determine premiums on anything other than relevant actuarial data, but the article did not address this issue. Instead, it speaks of the claims consciousness and legal sophistication in this country, of a glut of hungry lawyers, and of even more nebulous matters such as civil rights and public advocacy.

Rather than an apologia coauthored by Dr. Trent and Mr. Muhl, a spokesman for the insurance business, let Dr. Trent tell APA members how the new premium rates were negotiated and why he believes that premiums should be based more on prophesy than on facts concerning claims experience for psychiatrists.

BRIAN LIPTON, M.D.
New York, N.Y.

Dr. Trent Replies

SIR: In response to Dr. Bajor, I would like to point out that the change from the previous program was effected to develop a nationwide professional liability program with a financially strong company that would be duly licensed in all states and would afford direct organizational participation.

To achieve this goal, brokers from the Joseph A. Britton Agency are available at the annual meeting to answer members' questions about the program (a service to members), just as other members of APA staff, as well as representa-

tives from other APA programs, drug houses, book companies, etc., attend the meetings. All of this activity is an important aspect of any annual meeting.

From a business point of view, the larger the participation in our program, the more actuarially sound it will become. Any promotional effort that furthers this goal is truly a member service.

This program operates on a 21% expense factor, which means 79% of each premium dollar is available for loss payments. This ratio is superior to that of similar programs. Advertising space in *Psychiatric News* has been provided to this program without charge in order to obtain a part of the highly favorable loss ratio. Expenses would otherwise have been greater.

The committee appreciates the plight of physicians—especially in the states of California, Florida, New York, and Ohio, where premium increases have been large—and is mindful of the financial burden. In the current malpractice climate, however, APA is fortunate to have a national program (except for those states now eliminated by law) to offer at any price.

The pertinent questions raised by Dr. Lipton have also been voiced by many others. Mr. Muhl is with the Joseph A. Britton Agency, the official broker for the APA-sponsored program. He reviews all the official data about APA claims and losses on a daily basis. It is only natural to coordinate a description of our program with its chief operating officer.

Current premium rates for the APA program are based on a 1972 formula adopted by the APA Board of Trustees and Chubb and Sons agreeing that rates in each state on an annual basis would be 60% of Class I (general practitioners) Insurance Service Organization rates, or 25% of the leading carrier, such as a medical society program (in New York, California, and Florida, this rate is 10% below such leading carrier rates because of special problems in these states). Thus the initial rates were set to reflect losses lower than those of other Class I practitioners. In addition, it was agreed that the program would operate on a 75% loss ratio, which was recently revised to 79%. Thus, 21% of each premium dollar goes for expenses and 79% would be utilized for losses. Self-rating is expected when adequate, credible data on the program claims become available.

Current premium increases systematically reflect the above formula. If APA psychiatric losses prove to be less than anticipated—and current experience appears favorable—there is hope for a future premium reduction, provided psychiatric claims and inflation do not continue to escalate. Thus, the APA program was designed from the outset to be based on the most solid actuarial bases possible and has been heading in that direction since September 1972 at the fastest possible rate. Unfortunately, because of the long tail of malpractice, it takes several years to accumulate credible data. If current premiums ultimately prove to be overestimated, 79% of APA members' premium dollars are earmarked for actual APA losses and will be utilized in future premium adjustments based on actual data when accumulated.

Psychiatrists might also be interested to know that before 1975 it was not necessary to report psychiatric claims separately from Class I (general practitioners) claims. This explains why there were no previous systematic data on psychiatric losses that could be used for setting current premiums more accurately.

CHESTER L. TRENT, M.D.
Chairman, Committee on Member Insurance and Retirement Plans

Editor's Note: Like all articles submitted to this journal, the article by Dr. Trent and Mr. Muhl was subjected to our review process. The reviewer, who has a law degree as well as a medical degree and is a nationally recognized forensic psychiatry expert, made the following comment about the paper: "The subject is important, particularly since it involves APA activity in an area essential to medical practice." He recommended publication and assigned the paper a high priority.

Is Countertransference an Appropriate Issue in Clinical Supervision?

SIR: In "Countertransference: A Neglected Subject in Clinical Supervision" (January 1976 issue), Marcia Kraft Goin, M.D., and Frank Kline, M.D., discussed a very important and perhaps not well utilized area in supervision.

However, the article did not seem to assess the reasons why the supervisors did not use countertransference interpretations. Fear of being the trainee's therapist was mentioned, but a very natural reason for avoiding countertransference interpretations was not. The study has documented and the positive responses of the residents whose supervisors did deal with these issues—but what about those residents who do not respond so positively? Are there no such cases? What are the casualties, the risks of such negative responses, and their effect on the further learning abilities of such residents? I am surprised that the trainee's own negative response or resistance was not given as a possible (and perhaps the most appropriate) reason to avoid delving into countertransferences.

U.P. BHATT, M.D.
Riverside, R.I.

Dr. Goin Replies

SIR: Do supervisors avoid making countertransference interpretations because they fear the residents will respond negatively? Dr. Bhatt's letter raises a very important issue that I am glad to have the opportunity to clarify.

Residents are often offended when supervisors make any kind of interpretation about their attitudes, feelings, or behavior. They experience the interpretation as an attack, or they feel they are being put into the patient role, with all the attendant fears and anxieties this role arouses but none of the usual reassurance that the therapist will work with them until the conflict is fully resolved. We were not suggesting that supervisors make countertransference interpretations but that they provide an atmosphere in which the resident can feel free to talk about his or her feelings and not avoid the subject when it is brought up by the resident. We also believe that a supervisor should tell a resident when he thinks a countertransference reaction is distorting the therapeutic interaction.

The videotapes that we looked at showed several instances in which residents wished to talk about countertransference and their supervisors avoided the subject. The tapes also showed one session in which the way the supervisor approached the subject was certainly counterproductive, particularly because it did not allow for open discussion.

Those supervisors who were successful in their discussion of countertransference had a good working relationship with their residents and an attitude of colleagues learning togeth-

er. In this situation, if a trainee did not feel comfortable about pursuing the subject, the discussion of countertransference was dropped and this did not disrupt the teaching-learning relationship.

MARCIA KRAFT GOIN, M.D.
Los Angeles, Calif.

Reporting Side Effects of Imipramine in Children

SIR: In "Blood Pressure and Pulse Changes in Hyperactive Children Treated with Imipramine" (December 1975 issue), Lawrence M. Greenberg, M.D., and Absalom M. Yellin, Ph.D., reported increases in these measures. In the May 1975 issue of the *Journal*, Winsberg and associates (1) reported very clearly that cardiac effects of imipramine are related to the milligram per kilogram dosage given.

Drs. Greenberg and Yellin did not give any information on the weights of their patients, and in their experimental design they used a dosage of 100 mg/day, which is unusually high for the average child. In our description of the use of imipramine for childhood behavior disorder (2), the average dose used was 50 mg/day. Also, in the May 1975 issue, Hayes and associates (3) stated that imipramine dosage in children should be reported in milligrams per kilogram. Their study indicated that a dosage of 3.3 mg/kg appeared to be safe. Our experience with the use of imipramine for both behavior disorders and enuresis indicates that in a large percentage of cases the therapeutic dosage is well below this limit.

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HANS R. HUESSY, M.D.
Burlington, Vt.

Drs. Greenberg and Yellin Reply

SIR: The mean daily dosage of imipramine in our study was 3.6 mg/kg, with a range of 2.2–5.9 mg/kg. Methodological considerations guided us in our decision to use the same maximum dosage for all the children, who were very closely monitored throughout the study (1). Analysis failed to reveal any significant dosage/weight interactions. However, Dr. Huessy's concerns are most appropriate. Our concern was to point out that the treatment of hyperkinesis with imipramine deserves additional study in terms of both side effects and behavioral effects. Our data on side effects (reported in the article Dr. Huessy is commenting on), performance (2), and behavior rating (3) indicate that imipramine may not be the medication of choice with respect to the more important target symptoms of hyperkinesis, such as attention span and distractibility, and that the seeming efficacy of imipramine with respect to overactivity may be related to sedation effects. Others have also voiced similar concerns (4).

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LAWRENCE M. GREENBERG, M.D.
ABSALOM M. YELLIN, PH.D.
Minneapolis, Minn.

Should Intensive Psychotherapy Be Insured?

SIR: In "Insuring Intensive Psychotherapy" (December 1975 issue), Steven S. Sharfstein, M.D., and Howard L. Magnas offered some useful data. However, I question some of their conclusions.

They estimated that only 0.9%–1.1% of all psychiatric outpatients covered by a large health insurance plan (the Blue Cross/Blue Shield plan for federal employees) required intensive psychotherapy and that this activity consumed only 8.7%–10.3% of the total cost for physicians' treatment of mental disorders. Intensive psychotherapy was defined as expenditure of more than $6,000 a year for outpatient psychotherapy (roughly 150 visits a year at $40 a visit). From these data, the authors conclude that it is feasible to finance intensive psychotherapy under a comprehensive health insurance program.

I must acknowledge that my interpretation of their data is biased by my experience in a prepaid mental health system wherein more than 95% of patients with emotional problems can be effectively treated within 20 outpatient visits a year. Interestingly, the authors' data also suggest that practitioners in the indemnity sector find that the majority of their patients respond to brief psychotherapy—approximately 79% of their patients were treated at an annual expenditure of less than $999 or within fewer than 25 visits. Unfortunately, the remaining 21% of patients consumed more than two-thirds of the total cost for outpatient treatment.

Could we not apply the description "long-term" to the patients who used more than 25 visits a year? By using this label only for those who consumed more than 150 visits a year, the authors have made an arbitrary decision that shapes all of their subsequent arguments.

Given the gradual inflation of health care costs, it is difficult to be sanguine about the cost-effectiveness of mental health delivery, at least as it is revealed by the authors. The percentage of psychologically impaired people in a given population ranges (depending on the degree of impairment) from approximately 20% to 80% (1, 2). We also know that the initiation of help-seeking behavior, the form of that behavior, and the response of care providers is controlled by many factors other than the intensity of the patient's need for help (3). When the authors imply that utilization of mental health services has stabilized at a cost less than 7.3% of the total benefits, we must be aware that the actual number of patients involved is probably only a small fraction of those patients who could potentially use such services. Whatever stability has developed in the past 3 years may change

with changes in provider attitudes and patient receptivity to mental health services. Thus I question the authors' confidence that "It is clear that in this system, which offers a comprehensive benefit . . . the numbers of people electing high cost psychotherapies as the treatment of choice remain consistently low—a self-stabilizing factor mitigating against threats of exorbitant overutilization of the mental health benefit."

In addition Dr. Sharfstein and Mr. Magnas do not seem to appreciate the fact that by sanctioning an open-ended system of insuring psychotherapy (controlled only by peer review), they are sanctioning longer term treatment at the expense of the development of shorter term modalities. Several factors operate to bias therapists toward long-term treatment. First, therapists tend to view long-term treatment as being more comfortable and convenient because patient turnover and the effort involved in assessing patient problems and treatment goals are less when one sees fewer patients. Second, patient dependency needs are more readily indulged in long-term treatment. Third, permissiveness toward long-term treatment biases the process of psychotherapy because therapists are not challenged to develop short-term treatment skills. Finally, when therapists' calendars are filled with long-term treatment cases they are accessible to fewer patients who need help.

When we consider the absence of adequate outcome studies justifying the choice of long-term over short-term psychotherapy, sanctioning of long-term treatment without financial constraints becomes specious.

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THOMAS E. BITTKER, M.D.
Phoenix, Ariz.

SIR: Dr. Sharfstein and Mr. Magnas note that prior experience with insurance for intensive psychotherapy demonstrates that few patients will utilize this therapy. They also make an analogy between such intensive psychotherapy, at a cost of $6,000 a year, and catastrophic medical illness. This analogy is overly broad. "Catastrophic" medical illness means just that—a medical and financial catastrophe. Public policy supports the great cost of care in such catastrophes as renal failure, long-term dialysis, and renal transplant. In contrast, psychoanalysis deals with minor problems in psychoneurotic individuals who do not have major "ego defects." These patients are predominantly bright, young, verbal, successful, and have incomes considerably above the national median.

The authors also pointed out the need for better diagnosis and definition of therapeutic goals for such treatment. In a free society, an individual who is suffering from anxiety is free to undergo any treatment or educative process he wishes. It is an entirely different matter to ask the insurance industry and, ultimately, the vast majority of policy holders who would never choose psychoanalytic therapy to bear this financial burden.

According to Dr. Sharfstein and Mr. Magnas, "Health care is a right and, as such, includes the right to improved well-being. Limiting mental health care coverage under health insurance limits that right and diminishes the opportunities for total human growth and development." This rhetoric is garbage. Obviously, human growth and development are limited by myriad factors, including the economic cost of many growth-promoting experiences.

Health care is much too serious a concern to be jeopardized by such dubious claims as those the authors have made. There are crying needs for financial aid in the treatment of schizophrenia, mental retardation, and child abuse—to name just a few areas. Physicians are needed to aid the poor in the urban ghetto and in isolated rural areas. In the absence of scientific proof of the efficacy of psychoanalysis, there is no policy value in subsidizing an expensive, intensive psychotherapy for minor conditions in a privileged few.

WILLIAM L. ALLAN, M.D.
West Chester, Pa.

Dr. Sharfstein and Mr. Magnas Reply

SIR: In our paper we did not take up the issue of the type and degree of illness that analytic patients bring to treatment. There is now some evidence that these patients are indeed sick and in need of treatment rather than suffering from minor problems, as Dr. Allan suggests. In a Joint Information Service publication, it was estimated that 83% of analytic patients were moderately to severely impaired (1). To the extent that psychoanalysis is a growth and educative experience, its coverage under insurance should be limited or eliminated. However, to the extent that it is a treatment for very impaired, suffering individuals, it deserves no less coverage than any other medical treatment. One might ask Dr. Allan why it is that psychoanalysis, invented 60 years ago, continues to be practiced primarily by physicians rather than educators or "self-actualizers."

We did emphasize in our paper the need for outcome studies to prove the efficacy of such expensive treatment. This is no less true for any other expensive medical treatment.

Dr. Bittker's letter deserves perhaps another study comparing psychotherapy in prepaid medical health systems and psychotherapy in fee-for-service insurance mental health systems. Clearly, when one has a fixed budget and personnel contraints, practice changes and there is little opportunity for the "luxury" of long-term psychotherapy. Perhaps "intensive psychotherapy," which implies a specific treatment that attempts major personality change in a severely emotionally ill individual, is a better description than "long-term psychotherapy." Dr. Bittker's point about the prevalence of psychologically impaired people seems to miss the mark. What is more specific is the "treated prevalence" or utilization by a subpopulation such as federal employees, who are at high risk for undergoing psychotherapy because they are well-educated and highly verbal. In spite of being at high risk and more likely to use available benefits, the utilization and costs of long-term psychotherapy in this group have predictability and stability, which should enable insurance actuaries to compute premiums and consumers to decide whether they want to pay the few pennies extra in order to have the opportunity for psychotherapy, both short term and long term.

We fail to see Dr. Bittker's point that sanctioning long-term treatment will be detrimental to short-term modalities.

We are more concerned with Dr. Allan's point that the needs of other very impaired individuals, with schizophrenia, mental retardation, and other conditions that make them poor candidates for psychotherapy, will be neglected because highly skilled physicians would prefer to treat very impaired neurotics by psychotherapy. We must be increasingly concerned with the overall scarcity of resources and the fact that there is no sure way to ensure equity of access to high-quality care. However, the lesson of the Federal Employees Program is clear: In a large insured population, intensive psychotherapy is insurable and will have predictable utilization and stable costs.

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STEVEN S. SHARFSTEIN, M.D.
Rockville, Md.
HOWARD L. MAGNAS
Washington, D.C.

Viral Encephalopathy and Psychosis

SIR: The article "Viral Encephalopathy Mimicking Functional Psychosis" by Lawrence G. Wilson, M.D. (February 1976 issue), strongly suggested an association between herpes encephalitis and catatonia. In our review of four patients admitted to a psychiatric service with an initial diagnosis of acute psychosis, and later diagnosed as probably suffering from acute viral encephalitis, we were struck by several features that bear further emphasis. As in two of Dr. Wilson's cases, the diagnosis of herpes encephalitis could only be strongly suspected in two of our cases on the basis of the clinical evidence and the EEG. In a third case, postmortem examination was confirmatory. Serum antiherpes antibody titers were not obtained.

Of our three cases of suspected herpes encephalitis, none presented with catatonic features. One patient became acutely gradiose and paranoid, and exhibited bizarre and obscene behavior (he masturbated and smeared feces). Another was acutely excited, bizarre, and obscene (he masturbated openly). The third patient had auditory hallucinations and was paranoid. While the first two cases might resemble cases of catatonic excitement, there were never periods of catatonic rigidity, and the paranoid and grandiose features of the mental status of all three patients were impressive. A fourth patient did present catatonic and hebephrenic features, but after an initial suspicion by the psychiatric service of organic encephalopathy, careful medical evaluation including serology for viral antibodies and an eventual postmortem examination led to the diagnosis of subacute sclerosing panencephalitis (1). Based on these experiences, we suggest that herpes encephalitis need not present with catatonic rigidity or stupor, and conversely, that catatonia can be associated with other viral encephalitides.

In reviewing our cases, it was instructive to discover what information was most helpful diagnostically. All cases presented as subacute change in mental status and behavior that evolved within one or two weeks in previously normal young adults or adolescents. The mental symptoms were not typical of functional psychoses: hallucinations included olfactory and auditory illusions; there was remarkable disinhibition of behavior, with acute loss of verbal, social, and sexual continence (and later, urinary and fecal continence); and profound regression more typical of chronic schizophrenia. Many of the symptoms were associated with a feeling of strangeness and impending doom. These features suggest pathological changes in the temporal and frontal lobes. Only later did neurological findings come in to the picture. First, there were clonic, focal, or versive seizures, altered consciousness, increased muscle tone and tendon reflexes, and Babinski sign. Frontal lobe signs (snout, suck, and grasp reflexes), frontotemporal slowing in the EEG, and mild cerebrospinal fluid lymphocytosis and increased protein with a low-grade fever appeared even later; there were never signs of meningismus. Skull films, carotid angiography, computer-augmented tomographic radiography, and brain biopsy were not helpful in any of the cases. None of the patients had prior herpetic skin lesions.

In conclusion, it is not reasonable to assume that an acute psychosis or the syndrome of catatonia is schizophrenia. Since many encephalopathies are treatable and even the chemotherapy of viral encephalitides is making some progress, it is increasingly important to suspect acute viral and other organic encephalopathies in the differential diagnosis of all acute psychoses. It is particularly important to emphasize that psychiatrists have the most powerful diagnostic test to pick up such cases early—the careful mental status examination.

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R. MALCOLM STEWART, M.D.
ROSS J. BALDESSARINI, M.D.
Boston, Mass.

Practicing What We Preach

SIR: The overviews by John M. Davis, M.D., in the December 1975 and January 1976 issues of the *Journal* on maintenance therapy in schizophrenia and affective disorders are timely from more than one perspective. It is reassuring to see that research and therapeutic outcome studies are beginning to result in clear findings, such as the following: "The evidence that lithium prevents recurrence of manic-depressive illness has . . . been established beyond the shadow of a doubt. Given that there . . . were several suicides in the placebo groups, further replications of the above finding would only reincur these risks needlessly." Later in the article, Dr. Davis stated, "*In general it appears that lithium is approximately equally effective in unipolar and bipolar patients.*"

The above statements give clear guidance to the practicing psychiatrist. This letter is related to the nature of this guidance. More specifically, it seems that conducting placebo-lithium research or omitting the possibility of lithium therapy in any type of manic-depressive illness could be tantamount to malpractice. On the other hand, the Food and Drug Administration (FDA) has not approved lithium for use in unipolar illness.

I hope that most physicians do not practice medicine strictly according to the *Physicians' Desk Reference (PDR)* but rather use the *PDR* and the FDA as agents of informa-

tion. However, physicians do seem to view the FDA as a quasi-legal agency that somehow has regulatory power over the practice of medicine (1). Physicians appear to be practicing defensive-conservative medicine these days, and I wonder how many psychiatrists would never prescribe lithium in unipolar illness?

I feel that for these reasons specific questions need to be asked and faced.

1. Is the physician taking a malpractice risk by prescribing for indications other than those in the *PDR*?
2. If a physician prescribes for a reason not indicated in the *PDR*, should the patient always be so informed?
3. Is a physician taking a malpractice risk if he *avoids* prescribing in instances that the current literature would support?

There are other agents (e.g., propranolol, droperidol, and bethanechol) that are useful in psychiatric treatment. The above questions apply to these substances as well as lithium.

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LT. COL. R. HARLAN BRIDENBAUGH, MC, USA
Washington, D.C.

On the Boards Again

SIR: I agree with the recommendations and conclusions in "Experiences of Psychiatry Board Exam Casualties: A Survey" by Martin R. Lipp, M.D. (March 1976 issue).

Years ago, I talked with a number of candidates who passed the Boards and some who failed one or another portion. Those who failed were bitter, railing against the very poor quality of the choices of possible answers to the questions and also against the often obvious and occasionally vindictive biases or prejudices of the examiners.

One man was brilliant in his ability to memorize books of references, etc. During the orals, his sole task was to find out the "school" of the examiner and then to slant his exposition. He passed easily, but as a clinician he was a nothing.

When I worked for the Department of Mental Hygiene some time ago, I took a promotional examination. At that time, my orientation within the field of psychiatry was changing to a more physical and nutrients-disturbance etiologic approach. I suddenly found myself in empathic disarray with the examiners and realized my progressive disaffection with the field of psychiatry and my increasing discomfiture as a traditional psychiatrist. That was a devastating period for me. I eventually left state employ for private practice.

The psychiatric literature and I are increasingly at odds, to the point that I experience "psychiatry" as an alien intrusion. Given this, I could imagine the scenes should I attempt the Boards, which, in my opinion, do not reflect the individual's knowledge, skills, or comfort in working within the field of psychiatry. They reflect the expectations of a group of like-minded persons, the need for conformity, and the societal strength that comes from such conformity in addition to the biases and prejudices of the examiners and individuals who make up the questions.

The Boards should reflect capacity for work, thinking ability and range, creativity within working relationships, individual integrity and openness—the marks of an expert within his chosen field—without regard to someone else's preconceptions. I would consider standing for such a display of my thinking and working. Until then, I consider the Boards an onerous and unfair device for image projection to society, a restrictive device within the psychiatric profession, and an ego display to oneself.

GEORGE P. CONGRAM, M.D.
Dix Hills, N.Y.

Clarification

SIR: The closing sentence of "The Phasing Out of Mental Hospitals in the United States" by Milton Greenblatt, M.D., and Elizabeth Glazier, M.P.H. (November 1975 issue), points to the need for "accurate, standardized information regarding our present systems of care in order to make just and rational decisions regarding future allocations of scarce mental health resources." Consequently, we would like to inform *Journal* readers of a misunderstanding that might arise from appendix 2 of that article regarding Stockton State Hospital.

Although Stockton State Hospital is its legal name, its preferred name and the one by which it is known to the Joint Commission on Accreditation of Hospitals is Stockton Residential Facility. The facility consists of 18 units for the developmentally disabled (mentally retarded) and 2 units for the mentally disordered (mentally ill), not 2 units as suggested in the article.

The 18 units for the developmentally disabled serve all of California, not specifically "one county's program." In fact, San Joaquin County provides only approximately 27% of the residents of the units for the developmentally disabled. The 2 units for the mentally disabled do serve San Joaquin County by a unique leasing program for community mental health services. This program, in conjunction with the University of California, Davis, and the State of California, supplied a three-way contract that provided the residency training in our psychiatric education program.

The facility is academically involved not only with the nursing program at Delta Junior College, as was suggested in the article, but also with the nursing program at Modesto Junior College. It is also associated with the University of the Pacific, the University of California, Davis, and the State University at Sacramento in one or more of their programs in pharmacy, psychology, psychiatric residency, and social work.

We hope that this clarification abides by the spirit and the letter of the important article by Dr. Greenblatt and Ms. Glazier.

MICHAEL F. HEIMAN, M.D.
Stockton, Calif.
MARTIN A. JOHNSON, M.D.
Sacramento, Calif.

Dr. Greenblatt and Ms. Glazier Reply

SIR: We wish to thank Drs. Heiman and Johnson for clarifying and updating information regarding the current training and clinical program at Stockton State Hospital, now more properly known as the Stockton Residential Facility.

Although the standardized information called for in the closing paragraph of our article was intended primarily as a reference to standardization of state and federal statistical reporting systems, we agree that accurate information regarding types of programs is of equal importance. The abbreviated list of programs in appendix 2 of our article should have stated that the Stockton facility is currently academically involved with nursing programs at two junior colleges plus training programs in psychology, pharmacy, and social work at various other state institutions and that, although the program for the mentally ill serves only San Joaquin County, the program for the developmentally disabled serves all of California.

MILTON GREENBLATT, M.D.
ELIZABETH GLAZIER, M.P.H.
Sepulveda, Calif.

Hiccups Following Ingestion of Oral Chlordiazepoxide

SIR: I recently treated a 19-year-old soldier with 10 mg of chlordiazepoxide four times a day for anxiety secondary to situational legal difficulties. At the time of his second interview he gave the following interesting report. Within one hour after taking his first chlordiazepoxide capsule, he noted the onset of hiccups. The hiccups persisted despite his use of the conventional home remedy of holding his breath. He continued the medication for three days, and during that time his only relief from the hiccups occurred during sleep. On the second day he was seen by his dispensary physician, was found to be otherwise in good health, and was told to initiate bag-breathing. This also failed to alleviate his problem.

On the third day the patient decided that perhaps the medication was the source of his problem. Approximately 5 hours after he discontinued the medication his hiccups ceased. Being a somewhat inquisitive sort of person, he decided to test his hypothesis with a drug challenge on the following day. Within an hour, his hiccups returned. They disappeared spontaneously after 2 or 3 hours and had not returned 2 weeks later.

Although Greenblatt and Shader (1) note that hiccups have been reported following intravenous administration of diazepam for induction of anesthesia, they made no note of hiccups as a side effect of oral diazepam or chlordiazepoxide. My own review of the literature has also failed to disclose any such reports. I would be interested to hear whether any other physicians have noted this side effect with oral chlordiazepoxide.

REFERENCE

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MAJ. DANIEL K. WINSTEAD, MC, USA
APO New York 09696

Triple Mosaicism of X Chromosomes in Bipolar Affective Disorder

SIR: I would like to report an instance of triple mosaicism of X chromosomes with severe bipolar affective disorder. I reported on another such case in 1973 (1) and discussed the possible relationship between X chromosome mosaicism and bipolar affective disorder.

The patient is a 30-year-old woman who had been in and out of mental hospitals until July 1974, when she was given lithium carbonate. She has been responding to lithium satisfactorily and is free of psychosis. She was given the diagnosis of manic depressive illness, manic type, during most of her stay in state hospitals.

Her mental status at the time of the manic phase was described as hyperactive and over-talkative, with flight of ideas to the point of incoherence at times. Some degree of expansiveness and grandiose delusions was also noted, but there was no evidence of hallucinations or autistic thought. The depressive phase was described as being agitated, with some degree of psychomotor excitation.

The patient had some physical indications of Turner's syndrome, including webbed neck and low hairline. However, she gave birth to two children, a boy and a girl, and classical Turner's syndrome includes infertility. Karyotyping revealed 45X0/46XX/47XXX mosaicism with 79% XO, 3% XX, 17% XXX, and 1% aneuploidy in 100 cells. Her 17-month-old daughter's karyotype is 46XX normal female.

The patient is one of six siblings, three males and three females. Her paternal grandmother resembled her physically but had no reported history of psychosis. The patient's family history is negative for the presence of affective disorders or other psychoses.

My experiences of two mosaicisms of X chromosomes in individuals with bipolar affective disorder support my speculation that certain subclinical types of bipolar affective disorder may be associated with the presence of X chromosome mosaicism—especially when XO cell lines and XXX cell lines exist in the same individual.

It will be necessary to find more such cases to establish a new disease entity in affective disorders. We are currently in the process of studying karyotypes of patients who reveal severe bipolar affective disorder with physical resemblance to Turner's syndrome (especially low hairline and short stature) and weak or negative family history of affective disorders.

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YOSHI-TARO KUSUMI, M.D.
Raleigh, N.C.

"Waltons Syndrome"

SIR: I would like to share with your readers a new syndrome that has surfaced in the "last frontier" of Alaska and is related to contemporary economic conditions. Since the trans-Alaska pipeline boom began up here approximately two years ago, I have seen as patients a number of single men in their early twenties who have left their jobs in other areas because of the economic slump. These young men came to Alaska in an effort to make a lot of money and return home to help rescue brothers, sisters, and parents who are also experiencing relative poverty due to job losses. These patients are all very family oriented and conscientious and are the oldest children in their families.

The symptoms of these young men have largely been those of situational depression. The very high prices and living costs here have made it impossible for them to save nearly as much money as they had hoped. This is complicated by the sometimes very severe social and environmental aspects of life in Fairbanks with its long, cold winters (temperatures often drop below −50° F). I have generally used psychotherapy, antidepressant medications such as imipramine and amitriptyline, and occasionally, ECT to treat these patients. All of them have responded well to treatment. I see their depression as part of the maturing process rather than the beginning of any deep-seated problem.

These young men struck me as similar in attitude and disposition to John Boy, the hero of the television series "The Waltons," so I have named this type of reaction the "Waltons syndrome."

FRANCIS J. WHELAN, M.D.
Fairbanks, Alaska

In a Semantic Mood

SIR: Before I read "Affect, Mood, Emotion, and Feeling: Semantic Considerations" by Richard Ketai, M.D. (November 1975 issue), I had always thought affect was a display, mood referred to spirit, and emotion meant a feeling in a subjective sense. I stand corrected?

R.H. ISAAC, M.D.
Pasadena, Calif.

Dr. Ketai Replies

SIR: Dr. Isaac may stand corrected if he so desires. The reason for my article was to sort out the confusing and contradictory conceptualizations of these words as they are used in psychiatry. For every person who would agree with Dr. Isaac's definitions, there is someone else with an alternative idea, although neither is necessarily incorrect.

Words may signify whatever we define them to mean, the problem being to achieve consensus. I have proposed one way to define these words in order to understand and communicate about specific important concepts. My proposal was based on a careful review of pertinent literature, and the result reflects what I considered to be the best ideas of others integrated with what made the most sense to me. As far as I know, this is the only proposal that takes into account the multiple connotations of all these words. These include the scientific meaning of the words per se, the temporal features of the experiences reflected in the meanings of the words, the subjective-experienced meanings versus the objective-displayed meanings, and the distinctions between what is displayed and how it is displayed.

Thus when Dr. Isaac says he believes affect is a display I would not disagree—but I would emphasize that it is a transient display (affective display), that it is transiently experienced (affective sensation), and that it may be displayed in a variety of ways (affect quality). Mood may refer to "spirit" as Dr. Isaac suggests, and I would agree that this is true in a subjective, less fluctuating sense, but it should also be able to be described objectively by the observer. I maintain that emotion and feeling are more general and approximate synonyms for either mood or affect and are probably less scientific descriptors. Emotion is believed by some to refer to feeling states that are accompanied by obvious physiological arousal.

RICHARD KETAI, M.D.
Ann Arbor, Mich.

Neuropsychiatric Symptoms in Hypoglycemia

SIR: As one of the oldest living researchers in the field of neuropsychiatric phenomena in hypoglycemia, I take the liberty of making a few supplementary remarks on the excellent paper "A Psychiatric Study of Patients Referred with a Diagnosis of Hypoglycemia," by Charles V. Ford, M.D., and associates (March 1976 issue).

The simplest method to determine if neurological and psychiatric symptoms are due to hypoglycemia is to feed or, better still, inject intravenously a certain quantity of dextrose. If the symptoms do not promptly disappear they are not due to hypoglycemia.

I have pointed out elsewhere that for many hypoglycemic patients hospitalization is a psychic trauma that tends to temporarily raise their blood sugar levels. In such cases I recommend waiting for a few days after admission before starting observations and serum glucose studies.

JOSEPH WILDER, M.D.
West Hartford, Conn.

Correction

In "Decreased Blood Platelet MAO Activity in Unmedicated Chronic Schizophrenic Patients" by Edward F. Domino, M.D., and Sumathy Sampath Khanna, Ph.D. (March 1976 issue), the units of MAO activity were incorrectly described as μmol/mg protein/hour; this should have read nmol/mg protein/hour. This error was pointed out to us and to Dr. Domino by Ross J. Baldessarini, M.D. Dr. Domino has informed us that the problem resulted from typographical errors on the original manuscript and wishes to apologize for any confusion this may have caused *Journal* readers. The staff wishes to thank Drs. Domino and Baldessarini for correcting this error.

BOOK REVIEWS

Psychotherapy of Schizophrenia, *edited by John G. Gunderson, M.D., and Loren R. Mosher, M.D. New York, N.Y., Jason Aronson, 1975, 433 pp., $17.50.*

In a disorder that is so inherently heterogeneous as the schizophrenic syndrome, it is to be expected that multiple therapies compete for primacy and that their supporters tend to suffer at times from an excess of passion rather than perspective. The somatic therapies, i.e., convulsive and surgical, are attacked and defended violently in an atmosphere that is remarkably devoid of data. Partisan positions continue to be held in the face of contrary evidence. Megavitamin therapy, for example, has not disappeared even though carefully controlled studies failing to show its value have been published. The psychotherapy of schizophrenia has endured many of the same vicissitudes.

Intensive individual work with schizophrenic patients dominated the training centers in American psychiatry before the advent of chlorpromazine. During this period psychotherapy was seen as the preferred if not the only treatment. Students were taught that psychotherapy helped the basic or true problems causing the illness rather than merely reducing symptoms. The initial reaction of psychoanalytically oriented centers to the neuroleptics was hostile and depreciating. It was even argued that these drugs interfered with psychotherapy and should therefore not be used. This antiscientific attitude helped to polarize psychopharmacology from psychotherapy. Today there are only a few colleagues who are genuinely skilled in both treatment modalities.

Gradually, chlorpromazine and other phenothiazines displaced psychotherapy as the major method of treatment. The number of people teaching and learning intensive individual psychotherapy diminished. Institutions shifted to short-term care and reliance on drugs. Psychotherapists tended to retreat to a few centers that carried on the older traditions with a selected patient population. The political climate changed dramatically. This can be best seen in statements about the relative efficacy of pharmacotherapy versus psychotherapy. In the 1950s the drug companies suggested that psychopharmacological agents made it more possible to do psychotherapeutic work with schizophrenic patients. More recently it has been suggested that psychotherapy encourages patients to come back to the clinic and take their medication. As with all emotionally charged debates, the first victim in this controversy has been rationality.

The editors of this volume hope to improve the situation. They have brought together a group of colleagues who are primarily identified with the individual psychotherapy of schizophrenia. Many outstanding therapists are represented in this book. The entire first section is devoted to clinical practice and reviews the major controversies concerning the psychotherapy of schizophrenic patients. Every clinician will find something of value here. The second section is more theoretical in nature and touches upon several different formulations of schizophrenia. The final section deals with research on psychotherapy. Here balance and perspective are brought to bear on the question. The issues are joined scientifically rather than affectively. Evidence is reviewed and evaluated rigorously. The authors attempt to identify what further data would be useful in evaluating the effectiveness of psychotherapy.

As is to be expected in a multiauthored volume, the chapters demonstrate not only a range of viewpoints but a range of quality as well. Nevertheless, the editors have done an excellent job of maintaining a coherent volume in the face of this diversity. The book is an outstanding effort to deal with many of the pertinent issues surrounding the psychotherapy of schizophrenia and can be strongly recommended to anyone interested in the subject.

ROBERT CANCRO, M.D.
Farmington, Conn.

The Psychoanalytic Study of the Child, Vol. 30, *edited by Ruth S. Eissler, Anna Freud, Marianne Kris, and Albert J. Solnit. New Haven, Conn., Yale University Press, 1975, 751 pp., $22.50.*

Entering its fourth decade, the *Psychoanalytic Study of the Child* continues to take a seminal role in developing psychoanalytic theory and practice. Heavily influenced by the Hampstead Child Therapy Clinic, this annual contains 28 generally useful papers on child development, theory, clinical problems, preventive psychiatry, and psychohistory.

A feeling for this volume can be gained from skimming the surface of its contents, beginning with Dorothy Burlingham's list of blind infants' special problems and ending with Stephen Weissman's psychological study of Frederick Douglass. In between are discussions of the congenital absence of sensation, language deficits in children, infant observation, the phallic-narcissistic phase, the origins of awe, ambivalence, the "mirror" dream, childhood memories as contents of schizophrenic hallucinations and delusions, preventive intervention in adolescence, and the impact of surgery on boys.

A number of papers deserve special attention because they are significant contributions. First is the description of a central psychic constellation by Silverman, Rees, and Neubauer. This article nicely clarifies the personality organization of the pre-oedipal child and the appearance of the synthetic function of the ego during the fourth year of life. The authors offer tools for recognizing the enduring foundations of a child's personality by the age of three and a half in the form of characteristic ways of responding to drives, using external resources, and expressing self-esteem.

Burness Moore performs a service by reviewing the concept of narcissism, a long-standing source of confusion in the

literature. He defines narcissim as a positive, libidinal feeling toward the self with parallel and intermingled aggressive feelings. He sees narcissism as the energy interchanging with mental representations in psychoanalytic theory just as energy and matter interchange in the physical world. He endorses Mahler's view that narcissistic disorders result when a mother who had serious problems in her own separation-individuation is not gratified by her own child's individuation. As a consequence, her child incorporates her image, and the child's own self remains an alien, unsatisfying "as-if" object. In a similar vein Berger and Kennedy extend our knowledge of the maternal contributions to learning disorders in children. They found that a mother's lack of pride and pleasure in her child can lead the child to unconsciously comply with the mother's imposed image of backwardness, with resulting personality distortion.

Although addressed to child analysis, the discussion of transference by Sandler, Kennedy, and Tyson stands as a classic for transference manifestations in all forms of psychotherapy. Drawing upon Hampstead Index data, they describe four kinds of transference phenomena. First are the patient's habitual ways of generally relating to people, including the therapist; second are reactions from current relationships that "spill over" into the therapeutic relationship; third are past experiences that are relived during the therapeutic process; and last is the transference neurosis, which is a concentration of distortion within the therapeutic process with consequent diminution in their appearance elsewhere. The authors also wonder how much of the presumed transference neurosis with children actually is a result of the child's license to regress in treatment and discharge regressive wishes that then no longer appear elsewhere.

Another helpful therapeutic clarification is offered by Van Dam, Heinicke, and Shane. They see treatment termination as an extended process, evoking case-specific and phase-appropriate defense mechanisms in response to the loss of the therapist. They suggest a multiple-aspect view during the pretermination phase of therapy that takes into account all of the factors that determine the child's overall functioning in addition to treatment material. They also note that children quickly develop an amnesia for treatment, often remembering the analyst only in demeaning terms.

The most innovative paper is Gilbert Kliman's description of the Cornerstone method, an integration of analytic practice with a nursery school for emotionally disturbed children. Treatment is performed only in the classroom and augmented by the nursery school staff. Kliman's experience stands as a model for the effective psychoeducational treatment of the young child. In a similar way a model for the humanitarian care of surgical patients is offered by Solnit and Priel in their sensitive description of the team treatment of Israeli soldiers with severe burns.

In the social arena, Zinberg draws on current clinical experience and demonstrates that the physical and social settings must be taken into account in the genesis of drug addiction. Motivation and unconscious conflicts leading to drug dependency are important variables but not the direct causes of addicts' deterioration.

The foster care system is indicted by Goldstein, who points out that foster care frequently deprives the child of a family. Among other things he recommends the statutory creation of a "foster child with tenure" by a cutoff period of 12 to 18 months after which the foster relationship is defined as no longer temporary to facilitate adoption of the child by the foster parents.

An evening with this volume will acquaint the reader with a rich resource for further reference and with the value of the *Psychoanalytic Study of the Child* as an annual addition to the psychotherapist's library.

JACK C. WESTMAN, M.D.
Madison, Wis.

Europe's Inner Demons: An Enquiry Inspired by the Great Witch-Hunt, *by Norman Cohn. New York, N.Y., Basic Books, 1975, 292 pp., $12.50.*

The great medieval witch-hunt has long interested modern psychiatrists, partly because the delusional notions underlying it so closely resemble the psychotic ideas we encounter daily. This resemblance has led some psychiatrists, e.g., Zilboorg, to see the witch-hunt as arising primarily from individual psychopathology, an understanding of which, they suggest, can best help us understand the witch craze.

Medievalists, on the other hand, tend to seek explanations of the witch-hunt primarily in political, religious, and ideological history. Therefore, they view the delusions themselves, both individual and group, as secondary. Professor Cohn, the distinguished Chairman of Sussex University's Center of Research in Collective Psychopathology (now the Columbus Center) in England, demonstrates this approach most convincingly in this book. In presenting the definitive account of the various ideological currents—political, social, religious, psychopathological, and even fraudulent—which, coming together, produced the witch-hunt, he also corrects many of the accidental and intentional errors that have so gravely distorted many earlier accounts and appraisals.

The key belief underlying both the medieval witch-hunt and those of Hitler's Germany and McCarthyite America was the belief that, because of their alleged adherence to Evil, certain individuals were so terrible a threat to society that they had to be denied the ordinary protections and procedures of the law. When this occurred and, particularly, when torture became a major method through which such individuals were forced to "confess," the witch-hunt reached its peak. The fear of these "inhuman humans," plus their own "confessions" to the most outlandish and impossible "crimes," led to an abdication of reality testing at the highest social and political levels.

Each of the separate elements leading to the view that some humans are or have become inhuman has a long and complicated history, usually religious in origin, and Cohn delineates them carefully. Accusations of group infanticide, cannibalism, and incest, for example, were brought against second-century Christians by religious adversaries; later, the same accusations were leveled against "heretics" and other opponents by the Christian Church.

When legal protections were removed, the accused became vulnerable to the accusations of both sincere fanatics and mendacious careerists, who enhanced their own power by using torture to obtain the "confessions." These were carefully recorded, creating a corpus of concepts for later inquisitors to "elicit" from their own tortured victims. The repeated reassertion of such unbelievable fantasies can cause them to be believed, particularly in times of stress and especially when backed by the highest authority. This was Hitler's and the witch-hunt's "big lie."
As Cohn points out,

> The origin of the new [sixteenth and seventeenth century] stereotype of the witch lay . . . in the evidence ex-

tracted during the trials themselves. The grain of truth in the evidence lay in the fact that some people really believed themselves to fly at night, and that some women believed themselves to copulate at night with incubi. The rest came from the imagination of certain inquisitors, bishops and magistrates, who used and abused the inquisitorial procedure to obtain all the confirmation they needed. (p. 238)

Really massive witch-hunts occurred only where the concept of witchcraft included the sabbat ["the sacreligious and orgiastic gatherings" of the witches] and where judicial procedure included torture. (p. 254)

Cohn also delineates some of the erroneous notions that have arisen concerning the witch-hunt, such as the false belief that there were indeed conspiratorial political groups against which the hunt was directed. His meticulous research of the sources of this notion, some erroneous and some even fraudulent, is a pleasure to read.

This volume is a fascinating, scholarly, yet easily readable analysis of the many ideological sources of the witch-hunt, among which individual psychopathology is relatively unimportant. It is highly recommended to all interested in the origins and development of the collective myths and fantasies underlying one of Europe's most shameful episodes, one that has reverberated loudly even into our own times.

NATHANIEL S. LEHRMAN, M.D.
Roslyn, N.Y.

Hallucinations: Behavior, Experience, and Theory, *edited by R.K. Siegel and L.J. West. New York, N.Y., John Wiley & Sons, 1975, 311 pp., $25.00.*

Hallucinations are strange. Some artists have them, some inventors have them, some musicians have them, and even some schizophrenic persons have them. Now science is about to explain their significance; psychiatry cannot. I am not sure that philosophy can either, although philosophers have tried—Aristotle, Plato, Descartes, Hobbes, Russell, Wittgenstein, and James.

This book is filled with gems, but it is not easy to read. There are many differences in writing styles, models, theories, approaches, content, and context. Personally, I would prefer to experience hallucinations than to read about them.

The subtitle of this book, "Behavior, Experience, and Theory," reflects some of its boundaries. Siegel and West are to be congratulated on assembling such a dedicated group of researchers who know the literature and who are devoted to expanding man's knowledge of his dreams and perceptions, experiences and sensations—the whole gamut of varieties of levels of consciousness.

If I were to relive my professional life and training, I would insist on at least a full year in philosophy at some place like the Institute of Philosophy in Louvain, Belgium. This book reinforces such dreams.

Some of the authors, in particular Fischer, Sarbin, Juhasz, and West, deeply dissect our APA-approved definitions of perception, hallucination, pseudo-hallucination, hypnagogic and hypnopompic hallucination, reality, and sensation. In certain ways the book is like an encyclopedia of physiology or pharmacology. It is attractively printed, and the art by Sheridan and Yando is excellent.

The book is scientific in the best sense, i.e., it is not pompous, admits the limitation of knowledge about hallucinations, and yet offers a myriad of information, theories, facts, and hopes for the future. One of the authors predicts that it will take 100 years to really understand hallucinations. Other authors advise caution to us clinicians. Hallucinations may be transpersonal; it may take the psychiatrist as well as the patient to complete the act of diagnosing someone as hallucinating. Therein lies a warning:

Hallucinations are too important to be left entirely to clinical psychiatrists. . . . The average psychiatrist is confronted all too often by uncreative hallucinating patients who are stuck with conflict and unable to follow that "vast and painful initiation rite" (Bateson, 1972) [(1)], the path from agony to ecstasy: the process of disintegration and rebirth through symbolic experiences. (p. 233)

At least in the popular culture, hallucinations now can have a positive valuation, e.g., Laing, Castaneda, Fischer, Scheff, Goffman, Mutwa, and even Jung!

Intermediate cases between dreams and hallucinations can be found among the so-called hypnagogic [sleep-inducing] and hypnopompic [sleep-terminating] hallucinations. (p. 263)

The distinction between sensations and perceptions is one of the most popular and yet one of the most obscure distinctions in psychology. (p. 265)

This book will be avidly read by certain researchers and for Lent by others. I particularly recommend it to clinicians for bedtime reading.

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1. Bateson G: Steps to an Ecology of Mind. New York, Ballantine Books, 1972

ROSS V. SPECK, M.D.
Philadelphia, Pa.

The Experience of Introversion: An Integration of Phenomenological, Empirical, and Jungian Approaches, *by Kenneth Joel Shapiro and Irving E. Alexander. Durham, N.C., Duke University Press, 1975, 177 pp., $8.75.*

The authors have set out to "confirm a debt" to Jung as "one of the most sensitive observers of the subtle but pervasive individual differences that constitute human personality." The focus is on Jung's seminal, intuitive insight into the typology of personality and the concept of the introvertive personality. Shapiro and Alexander examine a proposed integrated empirical methodology and an experiential phenomenological formulation.

From their Thematic Apperception Test research with a group of undergraduate students, the authors conclude that the concept of psychological distance is probably central to all considerations of introversion and extroversion. The sense of distance between people leads to their discussion of the "introvert's people problem." The way in which the introvert lives and reflects experience results in a reversal of what is usually public and what is usually private. The introvert's life experience is a "peculiarly private affair since it is felt to be unshared in the moment." In compensation, the

introvert attempts to share his private thoughts and feelings, but, because this is asserted as a product intimately related to his own self-development, it has a disengaging effect. The feeling of exclusive possession, density, and opacity of the introvert's transformed world and the mediated nature of his experience, according to the authors, make the introvert's world a cumbersome burden in an interpersonal setting. The sense of interpersonal distance remains omnipresent.

(Jung said he used his typological work to understand the differences between himself and Freud: he discovered that he was an introverted, intuitive, thinking type; Freud was apparently an extroverted, sensation, thinking type. They therefore saw psychological and reality phenomena differently, as different truths.)

Shapiro and Alexander say that Jung was not only rescuing the introvert but perhaps recasting and elevating him above the extrovert. The evidence for this is inconclusive, but it seems clear that Jung created a new awareness of introversion as a normal human attitude reflected in particular behavior. Although he did not lead an introversion liberation movement, Jung did partially rescue ordinary introverts from the early Freudian interpretation as "a turning of libido from the object to the self-yielding fantasy, a kind of narcissistic cathexis of one's wish-fulfilling instincts . . . a pathological adjustment."

Citing Jung's Tavistock lectures of 1935, Shapiro and Alexander state that Jung reverted to a notion that introversion was pathological: "What is left of introversion, if anything, is indistinguishable from Jung's notion of neuroticism." This is not consistent, however, with Jung's concept of the normality of attitudes and functions except when they are extremely dominant in consciousness or disturbed and projected from the unconscious. Because Jung was using a different nomenclature in the Tavistock lectures, the authors have identified one of the many paradoxes and contradictions in Jung's writings.

This intellectual book is somewhat difficult to follow because of the writing style, which moves from formulations of great precision to rhetorical ambiguity. An example follows:

> In a way, with his I/E [introvert/extrovert] distinction, what Jung has done with respect to some fundamental problems in philosophy and psychology is facile; and, in a way, it is elegant. From one point of view, he has begged the philosophical question or dilemma of what is the proper and orginal grounding of reality, the knower or the known, the world-creator or world. He has also begged the psychological question as to the sorting out of the respective roles of the subjective and the objective in an individual's consciousness. His "answer" is the clear equivocation: There are two classes of individuals, the one oriented to the subject in consciousness, the other to the object. But from another point of view, what Jung has perpetrated is a dazzling coup in the history of ideas.

The book is recommended to research psychologists and students of typology. However, the rich humanness of the introverted experience can be found in Jung's *Psychological Types* (1), which the authors state is "one of the most compelling descriptions of people in the personality literature, or any other literature. One wonders why it is not read more."

The authors conclude, "We recognize immediately, without reflection, introvertedness. We know its appearance, but we want to know how to understand it, how to say what it is, and we want to know what it is to live it." Maybe this can be found only by being an introvert or by living with one—in analysis or, better still, in the works of artists and writers, especially poets, dramatists, and novelists.

REFERENCE

1. Jung CG: Psychological Types. The Collected Works, vol 6. Translated by Barnes HG, revised by Hull RFC, edited by Read H, Fordham M, Adler M. Princeton, NJ, Princeton University Press, 1971

HARRY A. WILMER, M.D., PH.D.
San Antonio, Tex.

Predictability in Psychopharmacology: Preclinical and Clinical Correlations, *edited by Abraham Sudilovsky, M.D., Samuel Gershon, M.D., and Bernard Beer, Ph.D. New York, N.Y., Raven Press, 1975, 283 pp., $22.50.*

Development of most prototypic psychotherapeutic drugs is characterized by a strong element of empiricism. The goal of science is to be able to predict. The contrast between the way new psychotherapeutic drugs are found and the way they should be discovered according to scientific thought has put most psychopharmacologists in a bind. This collection of papers relates to the subject of improving predictability. As is usually the case with such collections, topics are covered scantily, and many of the papers have appeared elsewhere.

One of the major problems in the clinical study of drugs is the accuracy of diagnosis. One long chapter in this book recounts the development and present state of research diagnostic criteria. Another chapter tries to make some sense out of depressive disorders. One supposes that it would be a step forward if everyone would at least clearly define the diagnostic terms they use. Two chapters deal with prediction of drug effects by using the EEG profile. After a decade of work on this area, only a couple of laboratories actively pursue this approach; independent vertification of the procedures is therefore lacking.

Two chapters on antianxiety drugs trot out the old notions that these drugs can be detected by the various impairments they produce, by subjective reports of relief of anxiety, and by changes in galvanic skin response. The animal separation model of depression is given a plug, but this analogue of so-called anaclitic depression in humans has not been proven to be universally predictable for other kinds of antidepressant treatment. The idea of rating animal behavior in the same way we rate human behavior and characterizing drugs by their behavioral profile is hardly new, but techniques have been further refined. Some techniques may be like the relationship between the barometer and the weather: the barometer tells you what kind of weather is present.

Long-acting neuroleptics can be tested by using the conditioned avoidance paradigm, perhaps the first of the major pharmacological screening tests for antipsychotic drugs. A more exciting and novel but highly laborious technique for screening antipsychotic drugs is to measure single-cell dopaminergic neuron firing. Antipsychotic drugs reverse amphetamine-induced depression of firing.

One of the chapters in this book speculates on the possible "endogenous psychotogen" basis for schizophrenia and suggests that a selective antagonist of dimethyltryptamine might be a valuable antipsychotic drug. This speculation is interesting but a bit farfetched.

One may be able to learn something about the nature of new compounds by the way they interact with receptors.

Benzodiazepines displace strychnine from glycine receptors, but such binding is probably less related to antianxiety than to muscle relaxant effects. Phenothiazines and tricyclics bind to muscarinic receptors, but these are hardly related to their antipsychotic effects. Still, like the use of single-cell firing rates, receptor interactions are potentially more specific screening tests that any we have had before.

This volume would be of most use to those who have the job of trying to screen new compounds for potential therapeutic usefulness, primarily at the preclinical level. It is doubtful that the clinical evaluation of such compounds will be much changed by any ideas put forth in this book.

LEO E. HOLLISTER, M.D.
Palo Alto, Calif.

The Inability To Mourn: Principles of Collective Behavior, *by Alexander and Margarete Mitscherlich; translated by Beverley R. Placzek. New York, N.Y., Grove Press (Random House, distributor), 1975, 308 pp., $12.50.*

This husband-wife team of German psychoanalysts examines "certain psychological processes which, taking place within large groups, result in the spread of either freedom or unfreedom of thought and insight." Their sociopsychological analysis in the first chapter attempts to deal with the illusions in German thought and behavior before, during, and after the Nazi experience. Having luxuriated in the world-redeeming dreams of greatness and in the idealization of Hitler as the Führer, Germans reacted to their defeat in World War II with effective but psychologically and politically expensive defenses that obviated mass melancholia by derealization of the past, identification with the victors, and manic efforts at physical and economic reconstruction. Three brief case vignettes of individuals with various political attitudes illustrate these psychological processes.

The remaining chapters discuss the broader issues of the subtitle, "Principles of Collective Behavior," from the psychoanalytic point of view. Man's adaptability to civilization is critically challenged by the increasingly complex conditions of our historical evolution. Our drives and needs, the ever present readiness for aggression, are inadequately controlled by the conservative values of traditional moralities, with their taboos and prejudices, which are made obsolete by the transformation of our environment through progressive industrialization, scientific permeation, and all-encompassing administrative machinery. Recent history has altered and weakened the roles of parental models and thereby created identification difficulties for adolescents, resulting in prolongation of adolescence. Clinical studies have suggested rather precisely age-related effects on growing youngsters of the absence of fathers during World War II and the delayed postwar recognition of "how deeply Germany had been compromised."

Several chapters consider tolerance (belatedly proclaimed and hesitatingly practiced), the social and personal ego, and the changing patterns of authority. Finally, the authors discuss the consequences of our historic evolution: affluence and the problem of renunciation; "traceless work," which erodes self-esteem and creates aggressive-depressive tensions; and increasing administration by specialists, leading to fraternal controls superseding the paternal and aggravating the problems of envy.

The book is a collection of separate essays. It is noted in the German edition (1) that, although the first and last chapters were written specifically for this book, the others are compositions prepared for the radio or for diverse publications. Thus the focus of the book—whether the authors address themselves to the expert, the student, or the lay reader—is not clear. The attempt to relate individual psychology of the unconscious to the sociology of collective behavior complicates the issues of readership and level of discourse. From the point of view of the authors' psychoanalytic orientation, the volume also suffers from a dichotomy of emphasis: the first chapter reflects a classical orientation heavily based on Freud's drive theory, but the later discussions reflect and emphasize the more recent developments and orientation of ego psychology. The Mitscherliches appear very well acquainted with the psychoanalytic literature in English.

The translation is faithful and competent. The index, however, is not improved by adding "Dresden bombing" and omitting "depression" in this work on mourning.

The original German publication was on the German best-seller list for about a year in 1967 and sold more than 100,000 hardcover copies, but it had little impact in the United States. Nevertheless, as Robert Jay Lifton points out in his preface to this edition, the Mitscherliches' discussion is most timely for post-Viet Nam and post-Nixon America. I particularly recommend the first chapter to all psychiatrists.

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1. Mitscherlich A, Mitscherlich M: Die Unfähigkeit zu Trauern. Munich, R Piper & Co Verlag, 1967

CHRISTOPHER T. BEVER, M.D.
Washington, D.C.

Medical Aspects of Human Sexuality: 750 Questions Answered by 500 Authorities, *compiled by Harold I. Lief, M.D. Baltimore, Md., Williams & Wilkins Co., 1975, 314 pp., $14.00.*

In 1967 Dr. Harold Lief helped establish the new journal *Medical Aspects of Human Sexuality*. Since then this periodical has fulfilled its promise of providing information and critical discussion on sexual topics for medical practitioners. This book consists of questions and answers reprinted from the journal.

The strengths and weaknesses of this volume are a function of its format. It is meant to be practical and oriented toward the practitioner's office rather than the classroom or the scientific meeting. To their credit, the respondents have obviously taken pains to write simple, lucid prose. Because the individuals answering the questions have had different types of training, the reader is provided with a perspective on the similarities and differences among medical practitioners, Ph.D.s in many fields, researchers, and clinicians. Of particular value is the fact that the personalities of the authorities are often revealed as they would be in an informal seminar situation. Although some of the replies are marvelously compressed minilectures, some of necessity suffer because the topic defies the structural requirement of brevity. One frequently but not invariably successful method used by Dr. Lief to avoid oversimplification was to include more than one answer to the same question by authorities of differing views.

Reflecting the interests of medical practitioners who wrote for the journal between 1967 and 1975, the book omits areas

that will perhaps receive more attention in the future. Cancer, facial injuries, renal dialysis, and loss of body parts other than sexual organs could have been profitably discussed. The section on pharmacology is interesting but brief; future dialogue between practitioners and their consultants will no doubt emphasize in greater detail the numerous drugs commonly used that may influence erotic behavior.

Some questions in this book would not have been asked by psychiatrists, although the entire volume should prove useful reading for medical students and general practitioners. However, only psychiatrists who specialize in sexual behavior will know the answers to the majority of the questions. Large parts of the volume will therefore be of interest. In addition, the reader will enjoy meeting experts he might not have been privileged to know. On the whole, this is an interesting compilation and one hopes it is the first in a series.

RICHARD C. FRIEDMAN, M.D.
New York, N.Y.

Psychiatric Illness in Adolescence: Its Psychotherapy and Prognosis, *by M.T. Haslam, M.A., M.D., D.P.M. London, England, Butterworths, 1975, 118 pp., £5.00.*

This very short book convincingly validates theories about the origins of adolescent emotional illness that have been talked and written about for decades. After a tedious but thorough review of the literature relating to early childhood deprivation, the author describes his own research study and minutely examines its statistical findings.

Haslam matched 50 consecutive admissions to a psychiatric unit against the same number of adolescents of similar age and sex who were seen at a casualty unit and had no psychiatric symptoms. Comprehensive questionnaires were administered to both groups. The important historical facts in the questionnaire centered around separation, traumatic experiences other than separation, total adequate relationships (TAR), and mutual understanding response (MUR), an unusual category that rated the degree of difference between the parents' and the adolescents' views of the nature of their relationship early in the adolescent's life.

As might be expected, the psychiatric patient group had a more deprived or traumatic history than the control group, but there were some surprises in the correlations between diagnosis and age at separation and length of separation. For example, the author found that early separation (birth to 18 months) was correlated positively with the development of anxiety symptoms in adolescence, but that separation later in childhood (18 months to 4 years) occurred most frequently in adolescents who developed psychotic symptoms. Relationships between TAR and diagnosis and the prevalence of a low MUR in the psychiatric patient group were also of particular interest.

In addition to the original study, Dr. Haslam conducted 2–5-year follow-up studies on 20 of the 50 psychiatric patients and came to some interesting conclusions in regard to prognosis. For instance, he states, "Bad mothering and separation at a critical age may . . . lower I.Q. and this in turn leads to a poor prognosis."

It was disappointing to learn that what the author terms "relationship therapy" proved to be more effective than no treatment only for patients diagnosed as having adolescent personality disorder—not for patients in other diagnostic categories, such as schizophrenia, depression, and anxiety.

This book makes valuable reading because of its confirmation of the important influence of childhood experiences and relationships on adolescent adjustment. The research material is presented in such detail that the reader must be well versed in statistical analysis to understand it all, but the author's summaries are clear and concise, so the conclusions of the studies are easily grasped. Everyone interested in the dynamics of emotional illness will profit from reading this report.

GRAHAM B. BLAINE, JR., M.D.
Cambridge, Mass.

Safeguarding Psychiatric Privacy: Computer Systems and Their Uses, *edited by Eugene M. Laska, Ph.D., and Rheta Bank, M.A. New York, N.Y., Wiley Biomedical (John Wiley & Sons), 1976, 445 pp., $22.00.*

From every side, the privacy of citizens is abridged as the nation becomes more centralized and more authoritarian. With our present emphasis on community mental health centers and third-party payment for health services, our need to protect the privacy of the consulting room increases. Conflicts between the patient's right to privacy and society's alleged need to protect its citizens and monitor its expenditures become more common. Central to this controversy is the issue of medical records—their content, control, and availability, not only for the immediate needs of the physician-patient interaction, but for Professional Standards Review Organization (PSRO) and utilization reviews, accountability, and funding.

The issue has surfaced in two ways in psychiatric practice: in individual legal challenges to the confidentiality of psychiatric records and in the growing use of digital computers to record, maintain, and analyze psychiatric records. *Safeguarding Psychiatric Privacy*, a collection of reports by a diverse group of writers, is an excellent introduction to two aspects of the problem: the technical issues of data collection using the multistate information system (MSIS) and the legal and political issues concerning access to records.

The major thrust of the volume is a description of the development and present status of this widely used record system, as developed at Rockland State Hospital in Orangeburg, N.Y. Case forms and sample outputs are described and portrayed and their many uses suggested. The text is designed for the medical reader rather than for the computer or engineering specialist. The flexibility of the MSIS is seen in its interesting applications: to administrative decision making and program evaluation at a Connecticut mental health center; to diagnosis and patient monitoring; to the development of the problem-oriented record for psychiatrists; and to setting up new mental health delivery systems in Israel and in Indonesia. The system described in these chapters is substantially more mature and sophisticated than the system described in 1968 (1) and even the one described in 1974 (2).

This volume is particularly stimulating in its second, less technical section, which is devoted to the issue of confidentiality. If records are computerized and data banks are accessible across the nation (and indeed the world) by computer cross-talk, how can we protect the confidentiality of psychiatric records? The authors of these chapters exclude the likelihood of an extension of the medical ethical code to computer technicians as well as a voluntary code of confidentiality. They suggest legal protection of access to data banks by appropriate federal laws. They also appeal to the

World Health Organization to develop a model international law.

They see the issue of confidentiality as transcending national boundaries. Their plea is idealistic in these times of global power struggles and the reawakening of the cold war. They also suggest more local regulation because they hesitate to depend on federal regulation alone. Passage of the Federal Privacy Act of 1975 (which went into effect on September 1, 1975) is seen as a first step in this needed development. Articles by Senator Barry Goldwater of Arizona and Representative E.I. Koch of New York provide an interesting background to this legislation.

The volume is an introduction to the uses of MSIS and to the problem of privacy of medical records. As psychiatrists participate in the progressive nationalization of medical practice, the issues in this volume will become more compelling. Laska and Bank have done psychiatry a service by alerting us to these important issues and by providing thoughtful postures for clinicians, politicians, and our legal colleagues—to whom this volume is highly recommended.

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MAX FINK, M.D.
Stony Brook, N.Y.

Forensic Psychiatry: A Practical Guide for Lawyers and Psychiatrists, *by Robert L. Sadoff, M.D. Springfield, Ill., Charles C Thomas, 1975, 245 pp., $14.50.*

The word "practical" in the subtitle of this book is most apropos. This is a book for working professionals. Dr. Sadoff addresses both psychiatrists and lawyers on the subject of what each should expect from the other in the many areas in which their professions converge. The attorney will learn, for example, the benefits of the psychiatric expert's knowing as much as possible rather than his or her being embarrassed under cross-examination, the medical rights of the patient who is his or her client, and the medical responsibilities of the psychiatrist as a consultant in the courtroom. It seems to me that much of the practical material in this work is directed toward the psychiatrist who is just getting into legal matters by one who has had experience. There is a great deal of useful advice on how to make a forensic report, how to prepare expert psychiatric testimony, how to use the results and interpretations of laboratory and psychological tests in court, and even how to dress for a hearing or trial.

Dr. Sadoff recognizes basic differences between the orientation of the professions of law and psychiatry. The aim of law is to control behavior; the goal of psychiatry is to understand it. The main intent of the book seems to be to enhance service to the court, the individuals, and the community through interdisciplinary cooperation and compromise.

One problem raised by Dr. Sadoff is that in both criminal and civil cases a psychiatrist is retained by the prosecution or the defense; there may well be bias in the psychiatrist's not being more neutrally appointed by the court. The function of the psychiatrist, he emphasizes, is to be a consultant and educator to the court; it remains with the judge and/or jury to assess guilt.

Chapters such as those on the juvenile and criminal law, sexual offenders, alcohol and drugs, mental competency, domestic relations, and commitment show the wide range of issues presented in this book. Sample cases highlight tendencies in each kind of case as well as unique aspects that dramatize the points being made.

University training is available in the field of forensic psychiatry. The author points out that only a naïve psychiatrist could plan to avoid court proceedings. The psychiatrist may well become involved with the law in two areas of malpractice: he or she may be an expert witness in a case involving another doctor or he or she may be a defendant in proceedings. In either instance, the psychiatrist must be prepared with the knowledge of the legal factors leading to malpractice, the relevant particulars of the case, and appropriate court presentation.

The book seems more directly addressed to psychiatrists than to attorneys. It smooths the way for more effective communication between two disciplines that increasingly must understand each other.

MYRON H. MARSHALL, M.D.
Buffalo, N.Y.

Childhood in China, *edited by William Kessen. New Haven, Conn., Yale University Press, 1975, 234 pp., $12.50; $3.95 (paper).*

Anyone concerned with child development will find this report of a visit of 13 American specialists in childhood to the People's Republic of China a necessary book. The various authors offer a thoughtful, ranging review of child rearing, family relationships, the status of schools, and the development of child health services. The combination of well-written text and arresting photographs offers a particularly detailed, vivid look into what is going on in the fascinating country of mainland China.

The group of distinguished visitors included psychologists, sociologists, a nursery school teacher, and a pediatrician who were invited by the Chinese government to observe a considerable number of nursery schools, kindergartens, primary schools, middle schools, and boarding schools. The group visited hospitals, health clinics, and the homes of Chinese families. Their many and varied observations have been collated into this detailed, evocative report on the status of childhood in China. Photographs of children in natural settings—playing, dancing, learning, interacting with their families—greatly enhance the value of this indispensable guide to the status of both children and families in the People's Republic of China.

Differences between American child-rearing practices and those in China are contrasted through anecdotes culled from many settings. The visitors observed that Chinese children were

> far less restless, less intense in their motor actions, and displayed less crying and whining than American children in similar situations. We were constantly struck by the quiet, gentle and controlled manner of Chinese children. All of us agreed that the gentleness and control of the children bore a resemblance to the gentleness and control of the teachers.

Repeatedly, the observers note with considerable awe that Chinese children were well mannered yet spontaneous, interested in the observers yet quite comfortable and able to continue with their activities. Children accepted observers as natural, pleasant additions rather than stimuli for excitement, disorganization, or regression.

The authors note that typical school lessons in China include moral lessons, lessons of cooperation rather than competition. In their descriptions the observers note that Chinese children are taught through books, stories, and recitations to share with others, to give another child a place in the line, and to cooperate with other children. Such descriptions call to mind the contrary observations made of the American competition-independence-disengagement ethic described by Philip Slater (1).

The observers note that "nowhere is there any evidence of inclusion of an activity in the program simply for its own sake." Rather, each activity is made to fit in with the moral development of the child and the needs of the community and the society. The observers emphasize the seriousness of the lessons Chinese children are given in their classrooms and compare them with the American educational system's concern with "nonsense like the 'Eency-Beency Spider.' " Standards were uniformly high, and children were taught by positive examples and gentle repetition. The teachers were said "to consider individual criticism and punishment to be undesirable methods of improving performance."

The observers comment on the prodigious ability of the children for memorization and accurate reproduction of dances and pictures as well as their effectiveness in acting out long dramatic scenes from plays. They state, "Everything we saw was on cue, and no one except the children of foreign diplomats missed a cue."

The observers remark repeatedly on the absence of fighting, pushing, and other disruptive behavior on the part of children. Similarly, they note the uniformly supportive and positive attitude of teachers and other staff toward their children and conclude that "teachers and staff members established an atmosphere of warmth, approval and perhaps most important, a confident expectation that each child would behave himself appropriately and succeed at the task at hand." This general atmosphere of approval is said to derive from the fact that Chinese teachers believe in what they are doing and in the future of the children whom they teach. The authors note that before the advent of Maoist China "teachers were said to have been content to care for the children and to play with them. Now they teach because they are involved in developing children who will work hard and be useful to themselves and their society."

These acute observations and comments by a group of observant, sophisticated American child development specialists merit our serious attention. They cannot be dismissed with such statements as that of a colleague with whom I discussed the book, who asked, "But are those children happy?" Perhaps such a comment reveals assumptions about our own society that a reading of this report will challenge. It is difficult to frame serious criticisms—other than political ones—of a child development system that seems to be working so well. It may be more realistic to ask what we can learn from the child development and educational practices of a country involved in a gigantic and seemingly humane experiment in child rearing.

This is a landmark book that should be studied by everyone involved in child development. The lessons in it are clear to anyone who works with children and deserve to be integrated with our own philosophy, or lack of philosophy, in regard to the best ways to educate children to be participants in a healthy, democratic society.

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SIDNEY WERKMAN, M.D.
Denver, Colo.

Mental Health: New York Law and Practice, Vols. 1 and 2, *by Jack Zusman, M.D., and William A. Carnahan. New York, N.Y., Matthew Bender, 1975, unnumbered pages, no price listed.*

These two volumes provide the basis for an exploration, analysis, and evaluation of mental health jurisprudence from both theoretical and practical perspectives. The collected experiences of forensic psychiatrists support the assumption that neither lawyers nor judges are familiar with the mental health system. Mental health professionals have therefore been relied on to circumvent the difficulties of administering the jurisprudence of the nonrational, the irrational, and, on occasion, the absurd. As the authors point out, "As a first step in dispelling interdisciplinary ignorance, misunderstandings, and animosities, this book was written."

These volumes attempt an exhaustive exposition of New York's mental health jurisprudence; an authoritative analysis of mental health and legal issues in administrative, civil, and criminal law areas; and a balanced critique of the mental health system from the vantage point of participants in this system. These volumes do not represent a theoretical critique of the mental health system or a polemical assault on the role of institutional psychiatry in legal settings. Although ostensibly written for attorneys and jurists who practice within the mental health system, they represent a fine addition to the forensic psychiatry literature and a model for similar treatises in other jurisdictions.

STANLEY L. PORTNOW, M.D.
New York, N.Y.

Legal Issues in Psychiatric Care, *by Laurence R. Tancredi, M.D., J.D., Julian Lieb, M.B., B.Ch., and Andrew E. Slaby, M.D., M.P.H. Hagerstown, Md., Harper & Row Medical Department, 1975, 160 pp., $10.75 (paper).*

According to the authors,

> [This book] was written for use as a textbook for students in all areas of mental health—psychiatrists, nurses, psychiatric aides and social workers, and psychologists, as well as general practitioners and family specialists. Law students and lawyers with a special interest in these issues should find the information and discussions presented here to be of particular value. When psychiatric medicolegal issues arise in medical offices or mental health clinics, a clinician may reach for this book and find ready guidelines without a morass of legal jargon.

Neither the content nor the presentation of this book can be commended. Perhaps the language became awkward and the substance dilute in the attempt to meet everyone's

needs, but I prefer standard legal jargon to such statements as, "Law has been constructed to prevent and account for injuries that are incurred through direct intentional acts or through negligent conduct . . . [whereas psychiatry deals with] the way behavior manifests itself."

The book does not seem to have been checked for internal consistency. For example, we are told on page 14 that "epileptics also are often subject to involuntary commitment" and then advised on page 47 that "with the advent of drugs that effectively control the seizures of epilepsy, the epileptic is rarely hospitalized through involuntary commitment."

The authors' negative claims for psychiatry would qualify them to write for the Scientologists, but their one positive claim is equally dubious, i.e., that there is some valid illustration of "psychiatry's unique position in helping women, blacks and Puerto Ricans." In a chapter titled "Definitional Problems in Psychiatry" they graciously indicate that they will "in all instances take the rather traditional approach that there is indeed something called mental illness."

In summary, this ambitious attempt lacks the substance for a text and the balance and scope for a guide.

DOROTHY A. STARR, M.D.
Washington, D.C.

Psychoactive Drugs and Social Judgment: Theory and Research, *edited by Kenneth R. Hammond and C.R.B. Joyce. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1975, 266 pp., no price listed.*

That psychoactive drugs may produce behavioral toxicity in addition to their therapeutic effects was pointed out by Jonathan Cole and others years ago, but our grasp of this area remains shaky.

The editors of this book present a body of research that attempts to bring the evaluation of the toxic effects of psychoactive drugs on social judgment into the experimental laboratory. The basic question for all such studies is whether the experimental model used is a convincing analogue of social judgment and has been validated against criterion ratings of social judgment in appropriate reference groups. Unfortunately, the primary task used in these investigations is neither convincing nor validated. It consists of a procedure through which the subject is exposed to three numbers and then asked to guess a fourth number. After each trial the subject is informed of the correct answer. The subject has to induce the proper rule.

In one procedure, only one of the cues is relevant to the target quantity. In a second, all three cues are relevant to the target quantity and must be combined mentally by the subject.

This does not strike me as a valid experimental model of real-life judgmental processes. The measure concerns accuracy in partly informed guessing of the size of a number. Most social judgmental processes do not involve making a guess concerning quantity but making a guess concerning the truth of a propositional statement or argument.

We may have to estimate a man's IQ by the number of books in his library in combination with the correctness of his English. It is far more likely, however, that we will try to decide whether to move across town, marry, or smile politely. Presumably these decisions can be placed in a propositional framework, e.g., "true or false: entering this new business is likely to increase my income eventually, after an initial dip."

A further drawback to the studies is that no systematic attempt is made to dissect out the variables that might affect social judgment adversely, e.g., is the patient sleepy, inattentive, motivated, "parkinsonized," etc.? Also, the studies suffer from very small samples and a considerable use of retrospective data analysis rather than hypothesis testing.

Some of the studies are naturalistic. Patients are assigned to different drugs by clinical process rather than randomly. These analyses are interpreted in terms of drug effect rather than the possible confounding effects of natural selection. This sort of elementary confusion does not belong in a book of model analyses.

Finally, with regard to the suitability of patient populations, there are studies on acute and chronic schizophrenic patients. One would think that if you are looking for psychotoxic effects on social judgment, the key patient group would be schizophrenic outpatients in remission who are being maintained on medication.

To sum up, this is a disappointing book about an important topic.

DONALD F. KLEIN, M.D.
Glen Oaks, N.Y.

Treatment Settings in Psychiatry: A Comparative Study, *by John B. Copas, B.Sc., Ph.D., Michael Fryer, B.Sc., and Ashley Robin, M.D., D.P.M. London, England, Henry Kimpton (New York, N.Y., British Book Centre, distributor), 1976, 103 pp., $9.50 (paper).*

This careful study examines treatment outcomes for 98 matched patient pairs seen in two inpatient units in Great Britain—a large mental hospital and the psychiatric unit of a district general hospital. The reader is treated to that scarce phenomenon in our field, a well-conceived, methodologically tight, statistically detailed prospective study of demographic and outcome variables that attempts to discover differences and relative superiorities between the general hospital and the large psychiatric hospital as treatment settings. The two hospitals compared, each of which is in the county borough of Southend, are the Rochford General Hospital and the Runwell (Psychiatric) Hospital.

The questions examined by this British study are relevant to treatment planning in the United States, given the large number of psychiatric units opening each year in community general hospitals. Although there is no federal plan for the United States to close public mental hospitals (as the 1971 policy of the British Department of Health and Social Security apparently proposes), the thrust toward "deinstitutionalization" in the United States makes timely an attempt to evaluate the relative therapeutic effectiveness of a psychiatric unit in a community general hospital.

The aim of the authors' project was to compare two inpatient settings with regard to treatment outcome by studying matched patient pairs on a large number of outcome variables using the Wing Present State Examination. Before elaborating their study, the authors describe the community and services of Southend and the two hospitals evaluated. They then present a careful analysis of demographic and diagnostic data for the 98 patient pairs; they call these data "input factors." They then attempt to demonstrate that there was no significant difference between patients referred to each of the two hospitals. Results of treatment are examined in terms of "output factors," i.e., patients were eval-

uated at admission, one month after admission or at discharge, and six months after discharge. Social workers obtained data at similar time frames from the patients' families.

In view of this detailed build-up and the elegant statistical methods and justifications used, the reader is disappointed to learn that there were no major differences between the two groups of patients on any of the treatment variables, although, happily, patients in each setting showed rather marked improvement in symptoms after one month. The authors describe only minor differences in treatment approaches, e.g., Runwell patients tended to remain inpatients longer, and Rochford patients were treated in day programs for equal lengths of time.

The authors acknowledge that they cannot generalize from their findings about the relative effectiveness of the two types of settings per se, but they conclude that there is suggestive evidence that many patients treated in psychiatric hospitals could be treated equally well in psychiatric units of general hospitals. This conclusion seems reasonable, but it is based on an assumption of no initial difference between the patients at admission to each of the units. I believe that this crucial assumption is not supported by their data, even though they interpret them in this way; this one flaw in their study is, unfortunately, a major one. They found that the diagnoses for patients assigned to each hospital were the same for 7 of 11 categories. However, Rochford patients showed greater anxiety and depersonalization and less disturbance of motor behavior. In addition, relationships were less satisfactory for Runwell patients: 13% of them lived alone before admission, compared with none of the Rochford patients. Runwell patients had been unemployed for at least six months more often than Rochford patients, and they had a higher incidence of suicidal risk and previous self-injury.

According to these input factors, it seems to me that one of the groups was indeed more disturbed than the other and, therefore, that inferences based on assumptions of similarity cannot be supported. In an example of gross carelessness uncharacteristic of the rest of their study, the authors ascribe more social isolation to the Rochford patients, but their data demonstrate that the opposite is true (p. 75). Thus, in terms of prognostic criteria, the Runwell patients seem to have been more severely impaired on admission than Rochford patients, findings quite consistent with the impression that patients admitted to psychiatric wards of state hospitals are more profoundly disturbed than those admitted to psychiatric wards of general hospitals. The authors would do well to deal directly with these differences between their two experimental groups rather than blurring them through a wish to find the samples prognostically identical.

ANDREW P. MORRISON, M.D.
Belmont, Mass.

Changing Patterns of Psychiatric Care, *by Bernard L. Bloom. New York, N.Y., Human Sciences Press (Behavioral Publications), 1975, 354 pp., $15.95.*

Critical assessment of mental health care delivery systems continues to be a timely and relevant pursuit. This text describes a social analysis and epidemiological study of psychiatric care in a single catchment area of 120,000 people during the decade 1960–1970. Census tract and inpatient questionnaire data were scrutinized for findings that have implications for mental health programming and preventive psychiatry approaches. The methodology used requires acceptance of the basic assumption that measures of treated incidence in a population bear some positive relationship to the true incidence of all treated and untreated disorders in the same population.

This book, organized into four parts, compares the data gathered in 1960 with those for 1970 from various perspectives. The text has many pages of figures and tables, and although the style is sometimes dull and repetitious, a careful reading reveals startling findings.

In both 1960 and 1970 more than two-thirds of all psychiatric hospitalizations were in private facilities, in which lengths of stay were shorter than in public facilities for every diagnostic group regardless of patient age, sex, or ethnic group. Thirty-three percent of all psychiatric admissions in 1960 were for psychoneuroses; in 1970 this figure was 52%. The number of admissions also increased during the decade (2.73 per 1,000 in 1960 versus 4.81 per 1,000 in 1970), but the percent of diagnoses of functional psychosis was stable, and the number of admissions for organic brain syndrome markedly decreased. Much of the increased admission rate was due to the hospitalization of more people under 21 years old for nonorganic, nonpsychotic disorders.

There was an increase in the number of inpatient beds and a dramatic proliferation of outpatient services during the decade. However, this study indicates that the expansion took place in an uncoordinated fashion. The increase in number and diversity of outpatient programs did not lead to a decrease in inpatient use. In fact, the majority of first inpatient admissions in 1970 were neither screened nor treated first as outpatients.

Cluster analysis of 35 variables used in this study separated out three specific cluster groups: socioeconomic affluence, social disequilibrium, and young marrieds. The three cluster configurations included more than 90% of the communality identified for both 1960 and 1970 census data.

Social class per se was not found to be convincingly associated with treatment rates. Marital disruption in the social disequilibrium cluster was the most striking individual and census tract correlate of patienthood. High frequency of low-level occupation and a greater percent of minority ethnic group individuals distinguished the patient group from the general population, but educational level did not.

The author scrutinizes his findings closely for identifiable stresses to lend support to the social causation hypothesis. Marital disruption, social isolation, and minority status are identified as highly significant stresses. Could preventive psychiatry approaches targeted specifically for these situations lead to a decrease in psychopathology?

Mental health care delivery programs in this community changed so significantly during the decade of study that they are reported to be qualitatively different. However, there is little evidence of cooperation in program planning and implementation between private and public sectors. Although high-risk neighborhoods and individual social situations are identifiable, too little preventive psychiatry has taken place. The author concludes that there is much that is not comprehensive about mental health care in this catchment area. During this decade of increasing funding and personnel effort, there has been little evidence of impact on incidence, prevalence, or severity of psychiatric disability in the community served.

This volume is not a polemic. It is a carefully detailed and thorough study. The author does not extend his comments beyond the limits of his data. Repetition of projects such as this should help us to identify more accurately the results of

community psychiatry programs and will require us to focus on the kinds of changes needed to improve outcome.

WILLIAM D. WEITZEL, M.D.
Lexington, Ky.

The Best Years of Your Life: A Guide to the Art and Science of Aging, *by Leopold Bellak, M.D. New York, N.Y., Atheneum, 1975, 288 pp., $10.00.*

The nineteenth century found the United States in the midst of promise and progress. The standard of living had increased, but epidemics, poverty, and war continued. At the beginning of the twentieth century these evils continued, but inroads were made against the problems, fostering the hope for better things to come. Advances were bound to occur, and they did: education became more universal, there was a greater abundance of commodities, there was more time for rest and leisure, and there was an even better promise for the future.

In 1976, however, a more pessimistic attitude prevails. The war to end all wars did not; other wars followed, and we still face the possibility of war. We also face the possibility of another depression, one not unlike that of the 1930s, which lasted for more than 6 years. Millions of American citizens still suffer from a lack of even the simplest commodities. The promise we felt at the beginning of the twentieth century has failed to be realized.

One of the most striking developments of modern times is the increase in the population of people over 65 years old. The reasons for this are obvious: preventive immunization, a better food supply, better hygiene, and better medical treatment in general. The span of life has widened, and a new and different problem results from man's longevity. It is in this context that Dr. Bellak writes his guide to the art and science of aging.

The Best Years of Your Life is a timely and necessary volume, although many writers have tackled the problems of aging, some more than 30 years ago (1, 2). Dr. Bellak's book is refreshingly free of post hoc propter hoc principles. He approaches the problem of aging with knowledge of the behavior of the senior citizen and of the interplay of factors affecting the individual and his environment. He avoids resorting to subliminal, subconscious, and assumed explanations.

Bellak dispels to some extent the myths surrounding aging and concludes, correctly, that the feeling of getting old is a mental state. His first 12 chapters are excellent and, in my opinion, factual. Chapter 13 and the following ones might prove to be somewhat distressing to the elderly. As a senior citizen myself, I was left with the feeling that the text is excellent for my children and relatives but not for myself. For the aged I would prefer a text with more discussion of mental hygiene. I would also like to have seen a little more emphasis on religion and the role of the religious counselor, plus more on the economic problems of the elderly. These subjects are certainly mentioned in the book, but the discussion is limited.

In my opinion, the author's most important effort in this book is the attempt to dispel the equation of age with organic brain syndrome. He also includes an unfortunately short paragraph on ECT, which has proved to be a safe and important therapeutic procedure for the old and for the young.

Research on aging has a long way to go; comparatively little knowledge is yet available. Is it possible to increase man's usefulness as well as his life span? This book represents a contribution to our knowledge. It is well constructed and delightful to read. Students, social workers, and anyone else who comes in contact with the elderly should read it.

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ISADORE GREEN, M.D.
Boston, Mass.

Theory of Motivation, 2nd ed., *by Robert C. Bolles. New York, N.Y., Harper & Row, 1975, 556 pp., no price listed.*

This is the second edition of a book initially published in 1966 and well received at that time. In the current edition Bolles attempts to update his earlier work to encompass developments that have occurred during the past decade, which are reflected in his approach to behavioral research and motivational theory.

The basic outline of the book remains unchanged: the subject of motivation is viewed from a historical viewpoint. The author begins by contrasting the mechanistic and rationalistic schools of motivational thinking and then traces the evolutionary development of what he terms "empirical determinism." In many ways he sees motivational concepts as having served their purpose and therefore no longer of much intrinsic value.

Bolles devotes slightly less space to discussion of drive theory in this edition. He discusses antecedent aspects of drive, their energizing effects, associative aspects of drive, and the concept of acquired drive.

The final chapters are devoted to the concepts of primary and secondary reinforcement, which Bolles clearly feels offer a thrust toward a more meaningful explanation of behavior. In many ways motivation was a concept that grew out of a desire to explain behavior in a broad sense; until recently, however, it was largely theoretical and untestable in a laboratory setting. Bolles is not particularly dogmatic in his approach; rather, he turns toward the idea that reinforcement concepts are more valid and reproducible, serving to replace earlier theoretical models.

In many ways this book is a rather exhaustive literary review of the field, but with a limited scope. The 77-page bibliography is impressive and attests to the strength of the author's scholarship. Most of the research efforts on behavior are animal studies, primarily with rats. The aspects of emotion and conflict are not dealt with because they are not within the purview that the author has defined for himself.

The primary appeal of this book would appear to be to those concerned with experimental psychology and, to some degree, to those interested in behavioral modification. It is an admirable achievement, but it has a rather limited appeal.

THOMAS C. BOND, M.D.
Belmont, Mass.

This Month's Reviewers

ROBERT CANCRO, M.D., is Professor, Department of Psychiatry, University of Connecticut Health Center, Farmington, Conn.

JACK C. WESTMAN, M.D., is Professor of Psychiatry, University of Wisconsin Medical School, Madison, Wis.

NATHANIEL S. LEHRMAN, M.D., is Deputy Director, Clinical, Kingsboro Psychiatric Center, Brooklyn, N.Y. He is also in private practice in Roslyn, N.Y.

ROSS V. SPECK, M.D., is in private practice in Philadelphia, Pa.

HARRY A. WILMER, M.D., PH.D., is Professor of Psychiatry, University of Texas Health Science Center, San Antonio, Tex.

LEO E. HOLLISTER, M.D., is Medical Investigator, Veterans Administration Hospital, Palo Alto, Calif.

CHRISTOPHER T. BEVER, M.D., is Clinical Professor of Psychiatry and Behavioral Sciences, George Washington University Medical School, Washington, D.C.

RICHARD C. FRIEDMAN, M.D., is Director, Behavioral Science Unit, International Institute for the Study of Human Reproduction, Columbia University College of Physicians and Surgeons, New York, N.Y.

GRAHAM B. BLAINE, JR., M.D., is Consultant in Psychiatry, Harvard University Health Service, Cambridge, Mass.

MAX FINK, M.D., is Professor of Psychiatry, Health Science Center, State University of New York at Stony Brook, N.Y.

MYRON H. MARSHALL, M.D., is Medical Director, Psychiatric Institute, Buffalo, N.Y.

SIDNEY WERKMAN, M.D., is Professor of Psychiatry, University of Colorado School of Medicine, Denver, Colo.

STANLEY L. PORTNOW, M.D., is Clinical Associate Professor of Psychiatry, New York University School of Medicine, New York, N.Y.

DOROTHY A. STARR, M.D., is in private practice in Washington, D.C.

DONALD F. KLEIN, M.D., is Director of Research and Evaluation, Department of Psychiatry, Long Island Jewish-Hillside Medical Center, Glen Oaks, N.Y.

ANDREW P. MORRISON, M.D., is Assistant Professor of Psychiatry, Tufts University Medical School, Boston, Mass.

WILLIAM D. WEITZEL, M.D., is Assistant Professor, Department of Psychiatry, University of Kentucky College of Medicine, and Director, Inpatient Psychiatry Service, University Hospital, Lexington, Ky.

ISADORE GREEN, M.D., is Lecturer in Neurology, Tufts University School of Medicine, and Chief of Neuropsychiatry, Soldiers Home Hospital, Chelsea, Mass.

THOMAS C. BOND, M.D., is Assistant Psychiatrist, McLean Hospital, Belmont, Mass., and Instructor in Psychiatry, Harvard Medical School, Boston, Mass.

Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Second Chance To Live: The Suicide Syndrome, *photographs by George Alpert, text by Ernest Leogrande. New York, N.Y., Da Capo Press, 1975, unnumbered pages, $6.95 (paper).*

The Social Animal, 2nd ed., *by Elliot Aronson. San Francisco, Calif., W.H. Freeman and Co., 1976, 326 pp., $4.95 (paper).*

Mental Health Services in Developing Countries: Papers Presented at a WHO Seminar on the Organization of Mental Health Services, Addis Ababa, 27 November–4 December 1973, *edited by T.A. Baasher, G.M. Carstairs, R. Giel, and F.R. Hassler. Geneva, Switzerland, World Health Organization (New York, N.Y., Q Corp., distributor), 1975, 132 pp., 18 Swiss francs (paper).*

The Structure of Evil: An Essay on the Unification of the Science of Man, *by Ernest Becker. New York, N.Y., Free Press (Macmillan Publishing Co.), 1976, 419 pp., $2.95 (paper).*

Control of Communicable Diseases in Man, 12th ed., 1975, *edited by Abram S. Benenson. Washington, D.C., American Public Health Association, 1975, 388 pp., $4.00 (paper).*

The Application of Social Psychology to Clinical Practice, *by Sharon S. Brehm. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1976, 265 pp., $17.50.*

Progress in Behaviour Therapy, *edited by J.C. Brengelmann, J.T. Quinn, P.J. Graham, J.J.M. Harbison, and H. McAllister. New York, N.Y., Springer-Verlag, 1975, 307 pp., $31.00.*

Behavior and Brain Electrical Activity, *edited by Neil Burch and H.L. Altshuler. New York, N.Y., Plenum Publishing Corp., 1975, 555 pp., $32.50.*

The Great Universe of Kota: Stress, Change and Mental Disorder in an Indian Village, *by G.M. Carstairs and R.L. Kapur. Berkeley, Calif., University of California Press, 1976, 174 pp., $12.50.*

The Annual of Psychoanalysis, Vol. III, *edited by the Chicago Institute for Psychoanalysis. New York, N.Y., International Universities Press, 1975, 436 pp., $17.50.*

Assertion Training: A Humanistic-Behavioral Guide to Self-Dignity, *by Sherwin B. Cotler and Julio J. Guerra. Champaign, Ill., Research Press, 1976, 229 pp., $5.95 (paper).*

Understanding Piaget, *by R. Droz and M. Rahmy; translated by Joyce Diamanti. New York, N.Y., International Universities Press, 1976, 212 pp., $10.00.*

Auditory Competence in Early Life: The Roots of Communicative Behavior, *by Rita B. Eisenberg, Sc.D. Baltimore, Md., University Park Press, 1976, 310 pp., $18.50.*

Biology of Brain Dysfunction, Vol. 3, *edited by Gerald E. Gaull. New York, N.Y., Plenum Press, 1975, 475 pp., $45.00.*

Sensory Saltation: Metastability in the Perceptual World, *by Frank A. Geldard. Hillsdale, N.J., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1975, 125 pp., no price listed.*

Is Alcoholism Hereditary? *by Donald Goodwin, M.D. New York, N.Y., Oxford University Press, 1976, 167 pp., $7.95.*

Awareness Games: Personal Growth Through Group Interaction, *by Claus-Jürgen Höper, Ulrike Kutzleb, Alke Stobbe, and Bertram Weber; translated by Hilary Davies. New York, N.Y., St. Martin's Press, 1975, 156 pp., $3.50 (paper).*

Sex Therapy at Home, *by David J. Kass, M.D., and Fred F. Stauss. New York, N.Y., Simon and Schuster, 1975, 240 pp., $8.95.*

The Marriage Savers, *by Joanne and Lew Koch. New York, N.Y., Coward, McCann & Geoghegan, 1976, 277 pp., $8.95.*

The Mentally Retarded Child and His Family: A Multidisciplinary Handbook, revised ed., *edited by Richard Koch, M.D., and James C. Dobson, Ph.D. New York, N.Y., Brunner/Mazel, 1976, 532 pp., $15.00.*

The Facts of Life: An Essay in Feelings, Facts, and Fantasy, *by R.D. Laing. New York, N.Y., Pantheon Books (Random House), 1976, 153 pp., $7.95.*

Boundaries: Psychological Man in Revolution, *by Robert Jay*

Lifton. New York, N.Y., Simon and Schuster, 1976, 113 pp., $2.95 (paper).

Creativity Mobilization Technique, *by Wolfgang Luthe, M.D. New York, N.Y., Grune & Stratton, 1976, 284 pp., $28.50.*

Psychiatry and the Criminal: A Guide to Psychiatric Examinations for the Criminal Courts, 3rd ed., *by John M. Macdonald, M.D. Springfield, Ill., Charles C Thomas, 1976, 494 pp., $28.50.*

A Prince of Our Disorder: The Life of T.E. Lawrence, *by John E. Mack. Boston, Mass., Little, Brown and Co., 1976, 537 pp., $15.00.*

A Marital Therapy Manual, *by Peter A. Martin, M.D. New York, N.Y., Brunner/Mazel, 1976, 197 pp., $12.50.*

Psiquiatría Social, *by José Luis Martí-Tusquets. Barcelona, Spain, Editorial Herder, 1976, 429 pp., 680 pesetas (paper).*

Insight: A Workbook for Self-Understanding and Personality Exploration, *by Chris Meriam and Lance Harris, Ph.D. Tacoma, Wash., Greater Lakes Mental Health Center, 1976, 185 pp., no price listed (loose-leaf notebook).*

Social Work Practice, 2nd ed., *by Carol H. Meyer. New York, N.Y., Free Press (Macmillan Publishing Co.), 1976, 260 pp., $10.95.*

Are You Driving Your Children to Drink? *by Donald A. Moses, M.D., and Robert E. Burger. New York, N.Y., Van Nostrand Reinhold Co. (Litton Educational Publishing), 1975, 219 pp., $8.95.*

Suicide and Morality: The Theories of Plato, Aquinas and Kant and Their Relevance for Suicidology, *by David Novak. New York, N.Y., Scholars Studies Press, 1975, 134 pp., $7.50.*

Conjuring Up Philip: An Adventure in Psychokinesis, *by Iris M. Owen with Margaret Sparrow. New York, N.Y., Harper & Row, 1976, 217 pp., $8.95.*

Treason, Say I, *by Joe Partyka. Mountain View, Calif., Joe Partyka, 1976, 185 pp., $1.95 (paper).*

Anna, *by David Reed. New York, N.Y., Basic Books, 1976, 243 pp., $10.00.*

The Mentally Retarded Child: A Psychological Approach, 2nd ed., *by Nancy M. Robinson, Ph.D., and Halbert B. Robinson, Ph.D. New York, N.Y., McGraw-Hill Book Co., 1976, 565 pp., $14.95.*

Psychological Apects of Learning Disabilities and Reading Disorders, *by Alan O. Ross. New York, N.Y., McGraw-Hill Book Co., 1976, 178 pp., $10.95.*

Knowledge in Action: Communication, Social Operations, and Management, *by Jurgen Ruesch. New York, N.Y., Jason Aronson, 1975, 342 pp., $15.00.*

Latency, *by Charles Sarnoff, M.D. New York, N.Y., Jason Aronson, 1976, 394 pp., $20.00.*

Drug Treatment of Mental Disorders, *edited by Lance L. Simpson. New York, N.Y., Raven Press, 1976, 309 pp., $13.50.*

Crisis Services for Campus and Community: A Handbook for the Volunteer, *by E. Robert Sinnett, Ph.D. Springfield, Ill., Charles C Thomas, 1976, 236 pp., $12.50.*

Handbook of Clinical Pathology, *by Joseph A. Sisson, M.D. Philadelphia, Pa., J.B. Lippincott Co., 1976, 494 pp., $18.00 (paper).*

Particulars of My Life, *by B.F. Skinner. New York, N.Y., Alfred A. Knopf, 1976, 319 pp., $10.00.*

Walden Two, *by B.F. Skinner. New York, N.Y., Macmillan Publishing Co., 1976, 301 pp., $7.95.*

Powers of Mind, *by Adam Smith. New York, N.Y., Random House, 1975, 418 pp., $10.00.*

The Growing Edge of Gestalt Therapy, *edited by Edward W.L. Smith, Ph.D. New York, N.Y., Brunner/Mazel, 1976, 234 pp., $12.95.*

An Atlas of Clinical Neurology, 2nd ed., *by John D. Spillane, M.D. New York, N.Y., Oxford University Press, 1975, 432 pp., $27.00.*

Evaluation of Psychological Therapies: Psychotherapies, Behavior Therapies, and Their Interactions, *edited by Robert L. Spitzer and Donald F. Klein. Baltimore, Md., Johns Hopkins University Press, 1976, 312 pp., $17.00.*

States of Consciousness, *by Charles T. Tart. New York, N.Y., E.P. Dutton & Co., 1975, 295 pp., $4.95 (paper).*

Chronic Illness in Children: Its Impact on Child and Family, *by Georgia Travis. Stanford, Calif., Stanford University Press, 1976, 538 pp., $19.50.*

The Fascist Ego: A Political Biography of Robert Brasillach, *by William R. Tucker. Berkeley, Calif., University of California Press, 1975, 318 pp., $21.50.*

Sexual Assault: The Victim and the Rapist, *edited by Marcia J. Walker and Stanley L. Brodsky. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1976, 175 pp., $15.00.*

Physical and Sexual Abuse of Children: Causes and Treatment, *by David R. Walters. Bloomington, Ind., Indiana University Press, 1975, 188 pp., $7.95.*

Behaviour Modification for the Treatment of Alcoholism: An Annotated Bibliography. Bibliographic Series 10, *compiled by C.E. Weise with the assistance of S. Busse, N.E. Reed, and S.F. Price. Toronto, Ont., Canada, Addiction Research Foundation, 1975, 275 pp., $8.00 (paper).*

Behavioral Toxicology, *edited by Bernard Weiss and Victor G. Laties. New York, N.Y., Plenum Press, 1975, 445 pp., $39.50.*

Advances in Sleep Research, Vol. 2, *edited by Elliot D. Weitz-*

man, M.D. Holliswood, N.Y., Spectrum Publications (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1976, 231 pp., no price listed.

Behavioral Science in Clinical Medicine, *by Stewart Wolf, M.D., and Helen Goodell. Springfield, Ill., Charles C Thomas, 1976, 218 pp., $14.50.*

The Therapist's Handbook: Treatment Methods of Mental Disorders, *edited by Benjamin B. Wolman, Ph.D. New York, N.Y., Van Nostrand Reinhold Co. (Litton Educational Publishing), 1976, 519 pp., $22.50.*

What Are You Afraid Of? A Guide to Dealing with Your Fears, *by John T. Wood. Englewood Cliffs, N.J., Prentice-Hall, 1976, 173 pp., $7.95.*

Abnormal Psychology: Changing Conceptions, 2nd ed., *by Melvin Zax and Emory L. Cowen. New York, N.Y., Holt, Rinehart and Winston, 1976, 611 pp., no price listed.*

Teaching Social Change: A Group Approach, *by Norman E. Zinberg, Harold N. Boris, and Marylynn Boris. Baltimore, Md., Johns Hopkins University Press, 1976, 243 pp., $12.95.*

Cerebral Localization: An Otfrid Foerster Symposium, *edited by K.J. Zülch, O. Creutzfeldt, and G.C. Galbraith. New York, N.Y., Springer-Verlag, 1975, 331 pp., $37.90.*

OFFICIAL ACTIONS

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in Los Angeles, Calif., March 8–9, 1976.

CHILD PSYCHIATRY

Anders, Thomas F., M.D., Stanford, Calif.
Arensdorf, Alfred M., M.D., Wailuku, Hawaii

Beg, Raihana R., M.D., Melrose Park, Pa.
Benitez, Jose-Luis, M.D., Paramus, N.J.
Berent, Philip J., M.D., Niles, Ill.
Blau, Stephen, M.D., Larchmont, N.Y.
Bramson, Stewart Mark, M.D., Minneapolis, Minn.
Browning, Diane Heiskell, M.D., Portland, Ore.

Campbell, Donald Ross, M.D., Chattanooga, Tenn.
Cantwell, Dennis Patrick, M.D., Woodland Hills, Calif.
Cohen, Donald J., M.D., New Haven, Conn.
Colley, Katharine D., M.D., Columbus, Ohio
Colombel, George, M.D., Cincinnati, Ohio
Combrinck-Graham, Lee, M.D., Philadelphia, Pa.

Davis, Paul H., M.D., Brooklyn, N.Y.

Etemad, Jacqueline, M.D., Tiburon, Calif.
Evans, Richard C., M.D., Irvington, N.Y.

Farley, Arthur J., M.D., Los Angeles, Calif.
Flanagan, Clyde Harvey Jr., M.D., Brookeville, Md.
Franklin, Roy Witsell Jr., M.D., San Bernadino, Calif.
Freeman, Ellen Deborah, M.D., New York, N.Y.

Geller, Barbara Gordon, M.D., New York, N.Y.
Glasser, Martin Evan, M.D., San Francisco, Calif.
Goldfine, Lee M., M.D., Santa Monica, Calif.
Greene, Jacquelyn K., M.D., Pasadena, Calif.
Greenhill, Laurence L., M.D., Mamaroneck, N.Y.
Gurnack, William, M.D., Boston, Mass.

Halperin, Edward Nathaniel, M.D., New York, N.Y.
Hammerman, Jeffery L., M.D., Melville, N.Y.
Haney, Renee, M.D., Tampa, Fla.
Hinshaw, Mark W., M.D., Grand Rapids, Mich.
Hoffman, Bernard, M.D., Dix Hills, N.Y.
Holzman, Bruce Rigler, M.D., Columbia, S.C.
Houston, Alma Faye, M.D., Little Rock, Ark.

Jacks, Brian Paul, M.D., Hollywood, Calif.
Jaso, Hector C., M.D., Providence, R.I.

Karatela, Mohammed Iqbal, M.D., Milledgeville, Ga.

Lansky, Shirley B., M.D., Overland Park, Kans.

Lee, Bernard Jae-Myoung, M.D., Brooklyn, N.Y.
Lowenstein, Robert A., M.D., New York, N.Y.
Lowrance, Bobby R., M.D., Houston, Tex.

Mega, Lesly Tamarin, M.D., New York, N.Y.
Michaels, Alvin Barry, M.D., Southfield, Mich.
Myers, Dan Allen, M.D., Dallas, Tex.

O'Byrne, Natalie K., M.D., San Anselmo, Calif.
Olson, Robert Nolan, M.D., Potomac, Md.

Persky, Murray, M.D., San Francisco, Calif.
Petti, Theodore A., M.D., New York, N.Y.
Pfeffer, Cynthia R., M.D., New York, N.Y.
Piggott, Leonard R., M.D., Detroit, Mich.

Raza, Syed Akhtar, M.D., St. Louis, Mo.
Reinhart, John Michael, M.D., Torrance, Calif.
Rich, Anthony M., M.D., Dearborn Heights, Mich.
Rich, Charles F., Jr., M.D., Middletown, Conn.
Rogeness, Graham Arthur, M.D., San Antonio, Tex.
Rosenblum, James P., M.D., Beverly Hills, Calif.
Rosenfeld, Robert Lee, M.D., Glencoe, Ill.

Sack, William H., M.D., Portland, Ore.
Sarles, Richard M., M.D., Baltimore, Md.
Sarma, P.S. Balasubramania, M.D., Wilmette, Ill.
Scharff, David Edward, M.D., Bethesda, Md.
Schmitt, Raymond F., Jr., M.D., Chapel Hill, N.C.
Schneider, Maria I., M.D., New York, N.Y.
Sholevar, G. Pirooz, M.D., Narberth, Pa.
Silverman, Gilbert, M.D., Richmond, Va.
Simeon, Jovan Georgi, M.D., Ottawa, Ont., Canada
Sours, John Appling, M.D., New York, N.Y.
Steg, John Paul, M.D., McLean, Va.
Stevenson, Karl William, M.D., Durham, N.C.

Teal, Stewart E., M.D., Davis, Calif.

Van Alstine, Susana L. Lopez, M.D., New York, N.Y.

Weiner, Michael Samuel, M.D., Austin, Tex.
White, Allen Collier, M.D., San Jose, Calif.
Williams, Daniel T., M.D., New York, N.Y.

Yates, Alayne, M.D., Blue Jay, Calif.

Zelman, Arthur Bernard, M.D., Rye, N.Y.

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See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

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In the five years since its introduction, Sinequan (doxepin HCl) has objectively demonstrated its value in the treatment of clinical depression/anxiety. Clinical studies have shown Sinequan to be unsurpassed in effectiveness when compared to other tricyclic antidepressants.

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Marked antianxiety activity

In addition to its significant antidepressant activity, which is usually seen two to three weeks after initiation of therapy, Sinequan possesses marked antianxiety activity.

In double-blind comparisons between doxepin and amitriptyline,[1-3] the antidepressant effects of both drugs were comparable; however, more notable antianxiety effects were seen with doxepin. Superior antianxiety activity was reported among depressed patients treated in a crisis intervention clinic.[4]

Prompt sedative activity

Of the tricyclic antidepressants, Sinequan has been found to have one of the most prominent sedative effects.[5] This sedative property can be taken advantage of by administering the major portion of the daily dose *h.s.* Since clinically depressed/anxious patients frequently have accompanying sleep disturbances, this may be a valuable benefit.

Generally does not affect the activity of guanethidine at usual clinical doses[6]

"A new tricyclic antidepressant, doxepin, was evaluated for its ability to block the amine uptake mechanisms of the peripheral adrenergic neuron and blood platelet in man. At low doses, there was no evidence of inhibition of the amine pump." At clinical dosages up to 150 mg. per day, Sinequan can be given to man concomitantly with guanethidine and related compounds without blocking the antihypertensive effect.[6]

Cardiovascular side effects reported infrequently

In a series of double-blind comparative studies, 1706 doxepin-treated patients were evaluated for side effects. Incidence of tachycardia was associated with only 45 (2.6%) clinically depressed/anxious patients.[3]

REFERENCES:
1. Grof, P. et al.: Curr. Ther. Res. 16:470, May 1974.
2. Hackett, E. and Kline, N. S.: Psychosomatics 10:21, May-June (Section II) 1969.
3. Pitts, N. E.: Psychosomatics 10:164, May-June 1969.
4. Kiev, A. and Slavin, J. R.: Scientific Exhibit, Presented at the 126th Annual Meeting of the American Psychiatric Association, Honolulu, Hawaii, May 7-11, 1973.
5. Claghorn, J. L., in Claghorn, J. L. et al.: Patient Care 5:108, Oct. 15, 1971.
6. Fann, W. E. et al.: Psychopharmacologia 22:111, 1971.

See Brief Summary on last page of advertisement for information on adverse reactions, contraindications, warnings and precautions.

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BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, paresthesias, ataxia, and extrapyramidal symptoms.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, facial edema, photosensitization, pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, and anorexia have been reported. (See anticholinergic effects).

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice and alopecia have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg. t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg./day to 150 mg./day.

In more severely ill patients an initial dose of 50 mg. t.i.d. may be required with subsequent gradual increase to 300 mg./day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg./day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg./day.

As an alternative regimen, the total daily dosage, up to 150 mg., may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Supply. Available as capsules containing doxepin HCl equivalent to 10 mg., 25 mg., 50 mg., and 100 mg. of doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN (doxepin HCl) 25 mg. and 50 mg. also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg./ml.) is available in 120 ml. bottles with an accompanying dropper calibrated at 5 mg., 10 mg., 15 mg., 20 mg., and 25 mg. SINEQUAN Oral Concentrate should be diluted with approximately 120 ml. of water or orange juice prior to administration.

More detailed professional information available on request.

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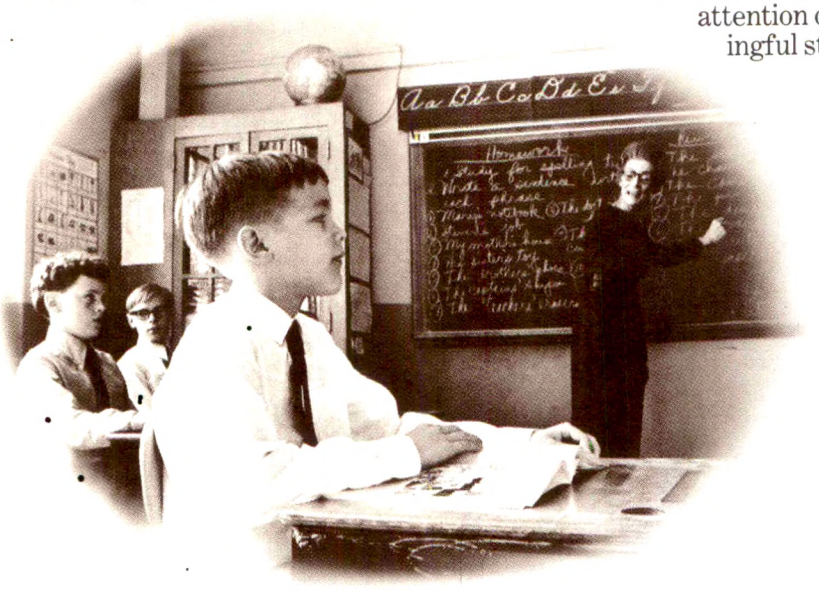
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Correction: In the 2/76 Halsted Press ad, three books—Gallant: Depression, Weitzman: Advances in Sleep Research, vol. 2, and Essman: Current Developments in Psychopharmacology, vol. 2—were incorrectly listed as Hemisphere publications. They are Spectrum publications.

Tested by time and experience in

1962

"...a considerable decrease of hyperactivity...."[1]
Knobel, 1962



Over a decade of controlled studies and clinical experience has shown the effectiveness of Ritalin in reducing the hyperactivity,[1-3] distractibility,[1,4,5] and disorganized behavior[1-8] in the MBD child.

By lessening the effects of motor and attentional disorders, Ritalin can help the MBD child to better focus his attention on meaningful stimuli and thus can often improve cognition and promote learning.[6,9]

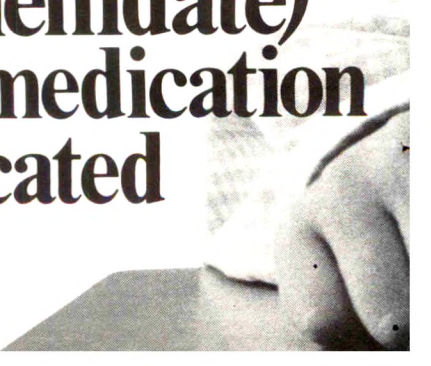
And side effects – insomnia and appetite loss – with Ritalin have occurred less frequently than with dextroamphetamine.[10,11]

Indeed, Ritalin is currently a drug of choice in many MBD situations,[10,12] and can prove to be an important element in many complete remedial programs for MBD.

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Drug treatment is not indicated for all children with MBD.

Dosage should be periodically interrupted. Often, these interruptions reveal some "stabilization" in the child's behavior even without medication, permitting a reduction in dosage and eventual discontinuance of drug therapy.

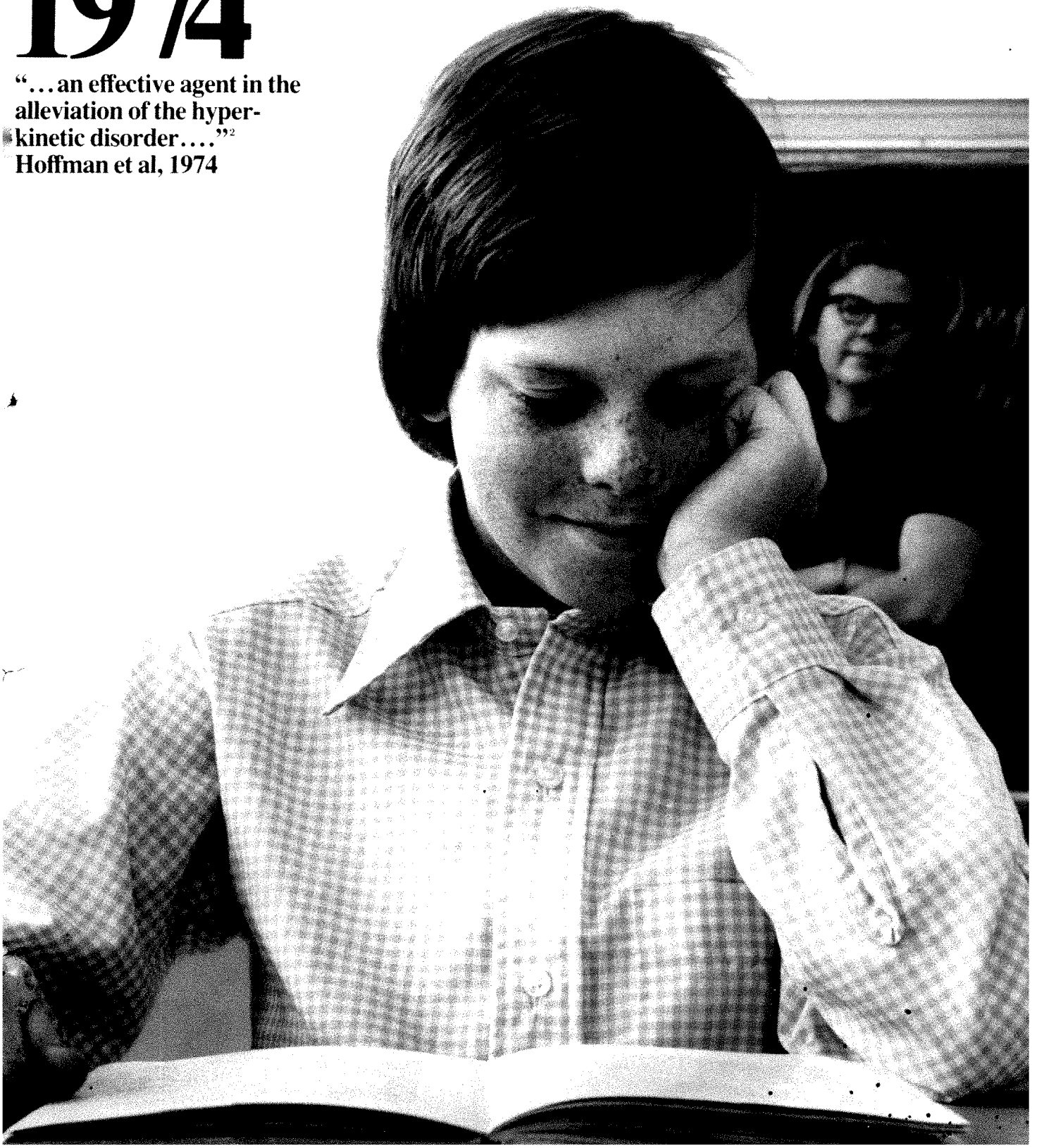
Ritalin®
(methylphenidate)
Only when medication is indicated

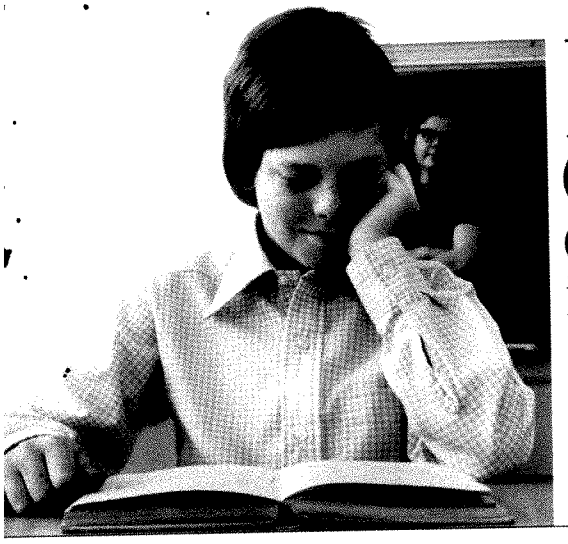


the treatment of MBD

1974

"...an effective agent in the alleviation of the hyperkinetic disorder...."[2]
Hoffman et al, 1974





Ritalin®
(methylphenidate)
Only when medication is indicated

Ritalin® hydrochloride ℂ
(methylphenidate hydrochloride)

TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over)

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

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2/5031 17

CIBA

No default insurance

Tofranil-**PM**®
imipramine **pamoate**

Geigy

In depression

Daily Dosage Chart



1 h.s.

A major reason for drug defaulting is multiple daily dosage schedules.

Ayd* reports that 70 percent of patients default on a q.i.d. regimen; 60 percent on t.i.d.; 30 percent on b.i.d., and only 7 percent on a single daily dose.

A single daily capsule that provides a full day's medication would logically assure better therapeutic results.

Because one capsule lasts from bedtime to bedtime, Tofranil-**PM**® may markedly reduce the probability of missed doses.

After therapy is initiated with the 75-mg. capsule, the 150-mg. capsule is the dose level at which optimum response is usually obtained.

Please review the prescribing information summarized on the back of this page.

*Ayd, F.J., Jr. (Ed.): Int. Drug Ther. Newslett. 7(9/10):35-50 (Nov./Dec.) 1972.

Tofranil-PM®
imipramine pamoate

One capsule
lasts from bedtime
to bedtime.

Initial Dose

For Maintenance Therapy

Starting Dose	Usual Optimum Response Dose	A Full Range to Choose From*			
					
75 mg.	150 mg.	150 mg.	125 mg.	100 mg.	75 mg.

*Each capsule contains imipramine pamoate equivalent to 150, 125, 100 or 75 mg. imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;

—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;

—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;

—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;

—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)

(B) 98-146-840-A(9/75) 667120

For complete details, including dosage and administration, please refer to the full prescribing information.

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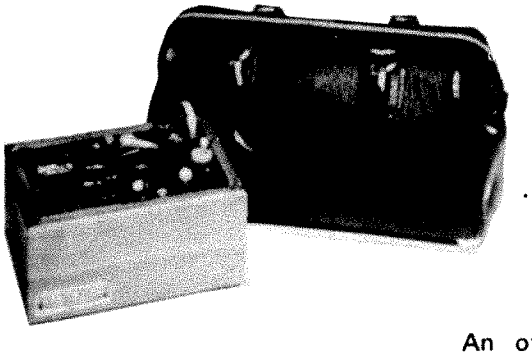
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in clinically significant depression...

penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

In clinically significant depression, the patient often becomes barricaded behind his own symptoms during psychotherapy. Because these symptoms can be so sharply defined and somatic in expression, he may reject the fact that they are psychic in origin. Sleep disturbance, gastrointestinal complaints, anorexia, chronic fatigue, and other psychosomatic symptoms may so preoccupy—or overwhelm—the depressed patient that truly productive therapeutic sessions are difficult to achieve. Instead of being able to concentrate on the core emotional problems of the depression itself, he may for example go on at length about how broken his sleep is or keep reiterating (while the therapeutic hour slips by) how physically miserable he feels.

ELAVIL®
(AMITRIPTYLINE HCl, MSD)
to help penetrate the symptom barrier in depression requiring medication

ELAVIL, a highly effective tricyclic antidepressant, will often alleviate these symptoms. With the help of ELAVIL, sleep disturbance—so common in clinically depressed patients and often the symptom that distresses them most—as



well as other "barrier symptoms" may be lessened to the point where they no longer come between you and the patient.

psychotherapy takes direction

As these preoccupying complaints are lifted, psychotherapy sessions can become more productive. Patients are enabled to respond more fruitfully during therapeutic hours and to resume or pursue more efficiently the necessary activities in their personal lives.

meeting therapeutic goals

And as the antidepressant activity of ELAVIL (Amitriptyline HCl, MSD) takes hold, it helps relieve the depressive condition itself. Sometimes only a minimum of actual psychotherapy is required. The patient may experience a more rapid recovery—while you conserve valuable time.

Prescribe ELAVIL then, to help lighten the patient's burden in depression—and yours in its management.

ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Its safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg
INJECTION: 10 mg per ml

ELAVIL®

(AMITRIPTYLINE HCl | MSD)

an antidepressant with an anxiety-reducing sedative component to its action



MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see following page.

MSD
MERCK SHARP & DOHME

addendum

ELAVIL®

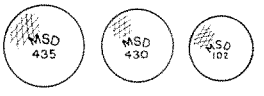
(AMITRIPTYLINE HCl | MSD)

Five tablet strengths for differing patient needs. The variety of dosage strengths available with ELAVIL makes possible great prescribing flexibility.

Once-a-day dosage at bedtime is an appropriate way to start—and maintain—many patients on therapy. The simplicity of this regimen helps improve patient compliance. ELAVIL may also be prescribed in divided daily doses.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

100 mg
75 mg
50 mg



These tablets may be advantageous when initiating therapy with once-daily bedtime dosage in adult outpatients. The usual starting dosage is 75 mg daily. Therapy may be initiated with 50 to 100 mg daily. This may be increased by 25 or 50 mg as necessary in the bedtime dose to a total of 150 mg per day.

These tablets may also be convenient for many hospitalized patients. These patients may need 100 mg a day initially which can be increased gradually to 200 mg a day if necessary. A small number of such patients may need as much as 300 mg a day.

25 mg



This tablet may prove useful when initiating therapy with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime doses.

10 mg



Because lower doses are generally recommended for adolescents and elderly patients, the 10-mg tablets may be most serviceable. Ten mg three times a day with 20 mg at bedtime may be satisfactory in adolescent and elderly patients who do not tolerate higher doses.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor or within at least 14 days following the discontinuance of a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.

When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains during treatment and until significant remission occurs; this type of patient should not have access to large quantities of the drug. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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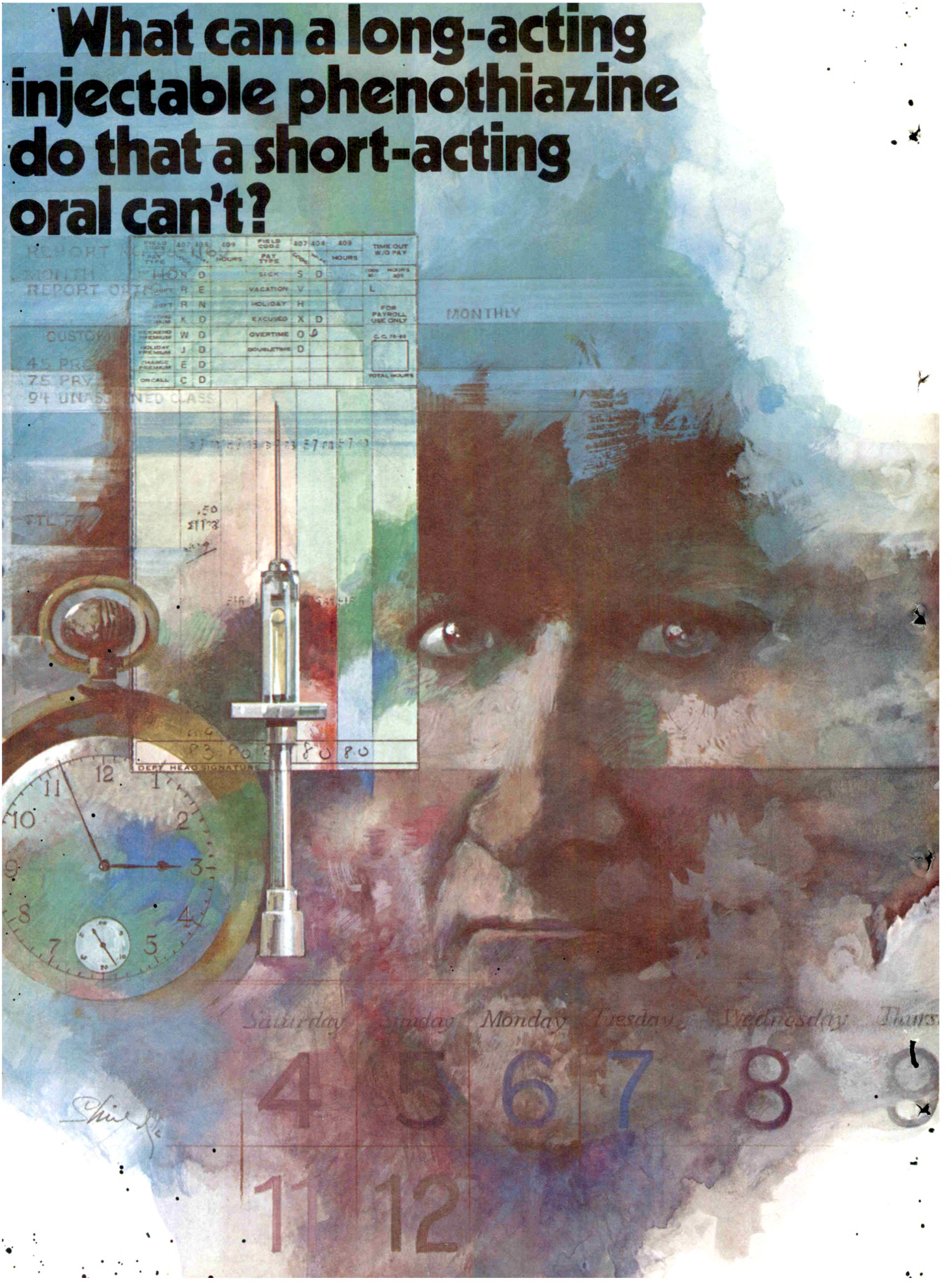
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It can save time, it can save money, and it can even save people – by reducing readmissions.

Time Prolixin Decanoate (Fluphenazine Decanoate Injection), with duration of action that may last up to 4 weeks or longer in patients on maintenance therapy, can effect important savings in nursing time.

Approximate Staff Time Required to Medicate 18 Schizophrenic Patients*

8 a.m. ¾ hr. 1 p.m. ¾ hr. 6 p.m. ¾ hr.	1 injection every 28 days for most patients
= 2¼ hours nursing time	4 minutes required for each injection
2¼ hrs. x 28 days = 63 hours of nursing time every 28 days	= 1 hr., 10 minutes nursing time in 28 days
NURSING TIME SAVED IN 28 DAYS	61 hours and 50 minutes or more than 7½ eight-hour working days

*Adapted from Platt R: Br J Social Psychiatry 2: 187-191, 1968.

Money Martin and Townend[1], who found from their study of Prolixin Decanoate that 15 of 39 patients could be maintained on 25 mg. every 4 weeks, also note that on this basis "a year's maintenance on chlorpromazine represents the order of 110 grams of phenothiazine as opposed to 0.33 gram of fluphenazine." Translating this into dollars shows the following potential hospital savings on the basis of patient population:

Agent and amount/year	Form	Cost/patient/year	Cost/500 patients/year
Chlorpromazine (SKF brand) 110 grams	8 oz. concentrate 100 mg./ml.†	$48.75*	$24,375
Prolixin Decanoate .33 grams	5 ml. vials 25 mg./ml.	23.17**	11,585
SAVINGS		$25.58 patient, year	$12,790 500 pts/yr.

* Calculated from prices published in 1975 Red Book.
** Calculated from Squibb 1975 price catalogue.
† With tablets, the annual cost is even greater.

People The controlled drug delivery system of Prolixin Decanoate helps get schizophrenic patients out of the hospital and helps them stay out. It promotes continuity of therapy—reducing outpatient drug defaulting from approximately 50% with oral medications according to one report[2] to about 16% according to another report[3].

It reduces the number and length of rehospitalizations. In one study of 103 patients maintained on injections of long-acting fluphenazines, total hospital readmissions for a year were cut from 191 to 50 and inpatient time from 8,713 days to 1,335 days[4].

It facilitates return to a productive life. A 12-month followup of 103 discharged patients revealed 77% in full-time employment or household duties and only 23% unemployed[4].

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may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

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References:
1. Martin ICA and Townend RA: Brit J Psychiat 124: 173-6, 1974.
2. Goldberg HL, DiMascio A, Chaudhary B: Psychosomatics 11: 173-177, 1970.
3. Medical World News, February 11, 1972, p. 58H.
4. Denham J and Adamson L: Can Psychiat Assoc J 18: 235-7, 1973.

For product brief summary see following page.

H425-001R

PROLIXIN DECANOATE®

(Fluphenazine Decanoate Injection)

may control schizophrenic symptoms for up to 4 weeks or longer in patients on maintenance therapy.

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia, liver damage or renal insufficiency, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

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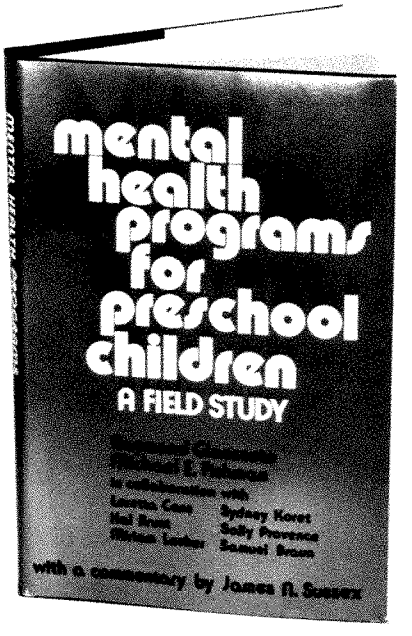
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This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



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President, American Association of Psychiatric Services for Children

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INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F Co.) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only) 10 mg./ml.

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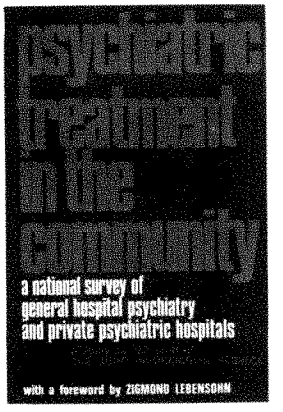
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This study also involves the private psychiatric hospitals, which have not changed much in number but are admitting about 10,000 more patients than in 1964—and they, too, are providing a remarkably comprehensive program.

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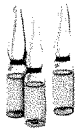
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CONTRAINDICATIONS TRILAFON is contraindicated in drug-associated central nervous system depression (barbiturates, alcohol, narcotics, analgesics, antihistamines). Perphenazine is contraindicated in the presence of existing blood dyscrasias, bone marrow depression and pre-existing liver damage, and in patients who are hypersensitive to perphenazine.

TRILAFON **Injection** should not be given to patients in coma or severely depressed states.

WARNINGS **Dosage in pregnancy:** Perphenazine should only be given to pregnant patients when, in the judgment of the physician, the potential benefits outweigh the possible risks.

Perphenazine can lower the convulsive threshold in susceptible individuals; it should be used with caution in patients with convulsive disorders. If the patient is being treated with an anticonvulsant agent, increased dosage of that agent may be required when perphenazine is used concomitantly.

Perphenazine should be used with caution in patients with psychic depression.

Perphenazine is not recommended for children under 12 years of age.

Perphenazine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks, such as driving a car or operating machinery.

PRECAUTIONS As with any potent medication, patients receiving perphenazine should be under medical supervision, particularly if they are receiving high doses. Patients who have had any severe reaction to phenothiazines or to imipramine should be treated cautiously, under close medical supervision.

Although the following adverse reactions have not been reported in patients treated with perphenazine, the possibility that they might occur with TRILAFON should be considered: blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, eosinophilia); liver damage (biliary stasis); narrowing of the visual fields; pigmentation of the retina, cornea, or lens; cerebral edema; polyphagia; photophobia; hyperpyrexia.

If hypotension develops, levarterenol (norepinephrine) can be used, but not epinephrine, because epinephrine's action is blocked and partly reversed by perphenazine. Severe, acute hypotension has occurred with the use of phenothiazines and is of particular concern in patients with mitral insufficiency or pheochromocytoma.

A significant rise in body temperature may indicate an idiosyncratic reaction to perphenazine; treatment with perphenazine should be stopped if this occurs.

The antiemetic effect of perphenazine can obscure signs of toxicity due to overdosage of other drugs, or mask the symptoms of disease (eg, brain tumor or intestinal obstruction).

Contact dermatitis has been reported with a perphenazine solution; therefore, contact of hands or clothing by those handling perphenazine solutions should be avoided.

POTENTIATION Since phenothiazines can potentiate the central-nervous-system-depressant actions of opiates, antihistamines, barbiturates, and alcohol, less than the usual dosage of these agents is required when they are administered concomitantly with TRILAFON. Patients should be cautioned that their response to alcohol may be increased while they are being treated with TRILAFON.

Phenothiazines also potentiate the effects of atropine, heat, and phosphorus insecticides, and should be used with caution in persons exposed to these agents.

ADVERSE REACTIONS *Extrapyramidal reactions:* dystonia including protrusion, discoloration, aching and rounding of the tongue; tonic spasm of the masticatory muscles, tight feeling in the throat, slurred speech, dysphagia, oculogyric crisis, trismus, torticollis, retrocollis, muscle weakness, and aching and numbness of the limbs; akathisia; motor restlessness; dyskinesia, parkinsonism; hyperreflexia; and ataxia. The incidence and severity of these reactions usually increase with increased dosage, but have occurred in some patients receiving low dosage. Reduction in dosage or treatment with an antispasmodic agent will usually control extrapyramidal reactions. In some instances, however, these reactions may persist after discontinuation of treatment with perphenazine.

Persistent tardive dyskinesia: As with all antipsychotic agents, tardive dyskinesia may appear in some patients on long-term therapy or may appear after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (eg, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities. There is no known effective treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine, vermicular movements of the tongue may be an early sign of the syndrome, and if the medication is stopped at that time the syndrome may not develop.

Allergic reactions: erythema, pruritus, urticaria, eczema, anaphylactoid reactions, and local and generalized edema. In extremely rare instances, individual idiosyncrasy or hypersensitivity to phenothiazines has resulted in cerebral edema, circulatory collapse, and death. Photosensitization, asthma, and exfoliative dermatitis have also occurred in patients treated with phenothiazines.

Autonomic reactions: blurred vision, dry mouth or salivation, nasal congestion, nausea, vomiting, hypertension, tachycardia, hypotension, anorexia, urinary frequency or incontinence, and constipation. Significant autonomic effects have been infrequent in patients receiving less than 24 mg. perphenazine daily.

Other reactions: endocrine disturbances (lactation, gynecomastia, galactorrhea, disturbances in the menstrual cycle), headaches, mild insomnia, altered cerebrospinal fluid proteins, ECG abnormalities, reactivation of psychosis, paradoxical excitement, paranoid-like reactions, catatonia, and systemic lupus erythematosus-like syndrome. Hypnotic effects appear to be minimal, particularly in patients who are permitted to remain active. The following adverse reactions, though rare, have also been reported to be associated with perphenazine treatment: agranulocytosis; jaundice; hyperpigmentation of the skin; grand mal convulsions; failure of ejaculation; hyperglycemia.

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Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

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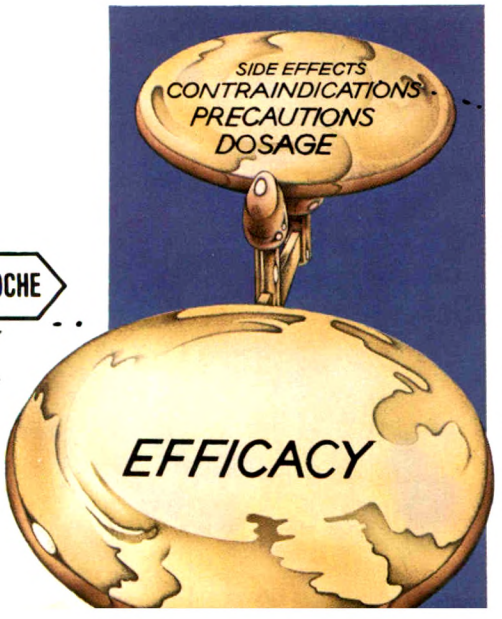
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